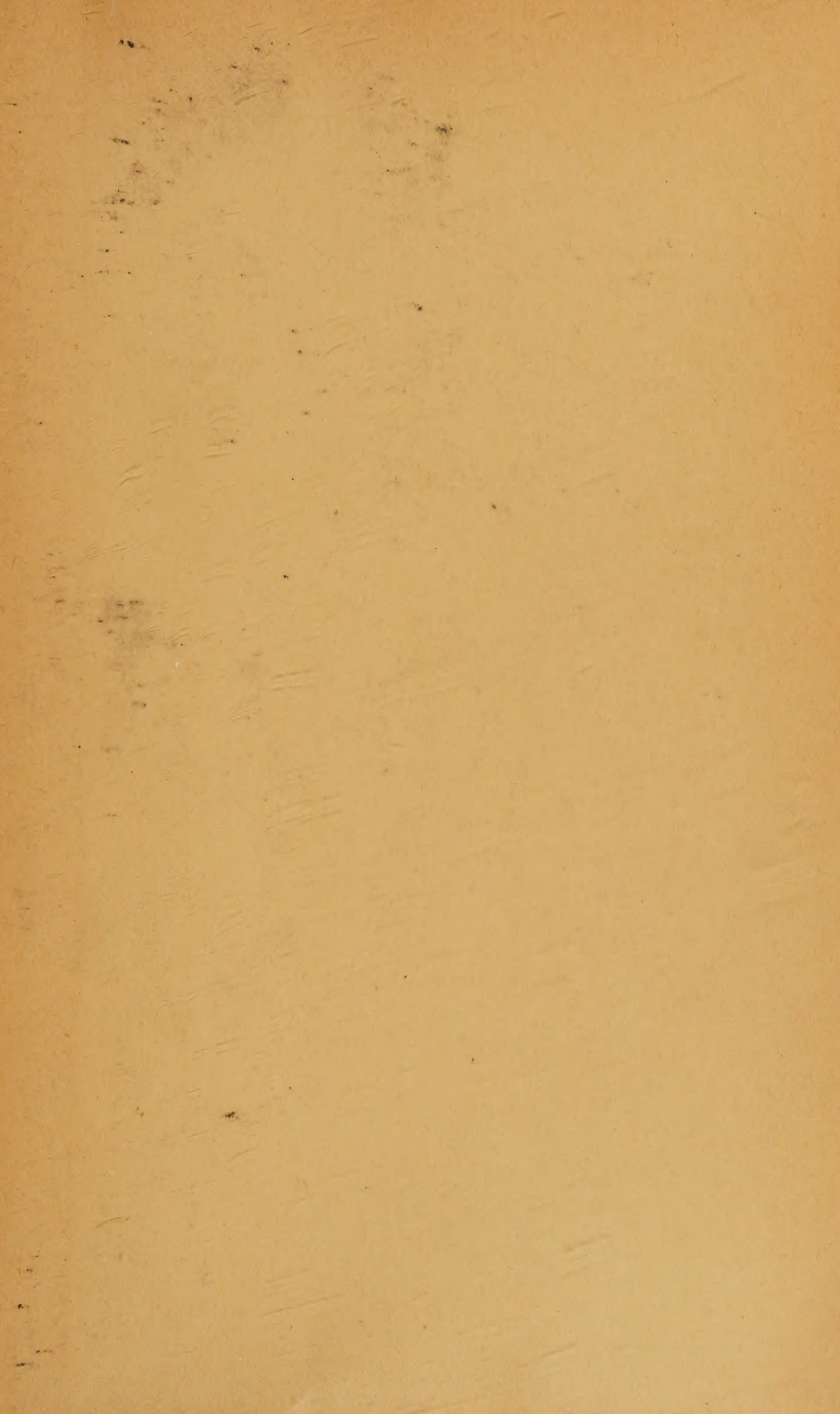




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# THE NOSE•THROAT•AND EAR AND THEIR DISEASES

IN ORIGINAL CONTRIBUTIONS BY AMERICAN AND EUROPEAN AUTHORS

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## PREFACE

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IN a systematic work by many authors one does not expect to find the degree of consistency usual in a book written entirely by one author. Indeed it is one of the advantages of such a work that conflicting opinions by leading authorities may be presented. The very complete index will refer the reader to different articles in which certain subjects are separately considered by two or more authors from radically different points of view. It is hoped that this will make the work more useful than it might have been if the editors had held the respective authors to narrower limits. Each author, being a leading authority on his subject, has been allowed to divide it as seemed to him best; no rigid adherence to the ideal system of classification has been enforced. Historical data have been eliminated. The aim has been to present the opinion of today rather than the developmental stages by which that opinion has been reached. *What to do* and *how to do it* has been considered the kind of information for which most readers will consult this book. Anyone bent on research will find references leading as far afield as he may wish to go.

Thanks are due to the collaborators for their hearty coöperation; to the various publishers and instrument makers who have lent material for illustrations; and to the personnel of the W. B. Saunders Company for their unfailing courtesy and aid.

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# DISEASES OF THE NOSE, THROAT, AND EAR

## PART I—NOSE AND ACCESSORY SINUSES

### THE ANATOMY OF THE NOSE

IN the space allotted only the salient features of the development and the anatomy of the external nose, the nasal cavity, and the paranasal (accessory) sinuses can be presented. Detailed descriptions and mooted points will, therefore, be omitted. The reader is referred to special treatises on the anatomy of the nose and the ancillary and related structures for more extended discussions and additional subject matter.

### EMBRYOLOGY

The nose appears as paired areas of thickened ectoderm, the *ectodermal placodes*, on the outer surface of the wall of the forebrain just above the

*Brain*



Fig. 1.—Photomicrograph of frontal section through the head of an embryo aged thirty-five days, showing the manner of fusion of the maxillary process and the medial nasal process in the caudal closure of the early nasal pit.

primitive mouth cavity. During the fourth week of embryonal life the nasal areas become depressed between the medial and the lateral nasal

processes and form the early *nasal pits*. These are at first continuous with the mouth cavity. Later, by fusion of the maxillary and the lateral nasal processes with the medial nasal process, the nasal pits are separated from the mouth cavity. The nasal pits (forty-five-day embryo) establish secondary connections with the roof of the early mouth cavity by a thinning out and rupture of the *bucconasal membranes*. This leads to the formation of the *primitive choanæ* (primitive posterior nares) and the establishment of the *primitive palate*. Later, the more cranial portion of the mouth cavity becomes part of the nasal cavity by the formation, growth, and fusion in the median plane of the paired palatal processes of the later maxillæ and the palatine bones. This leads to the formation of the *definitive*

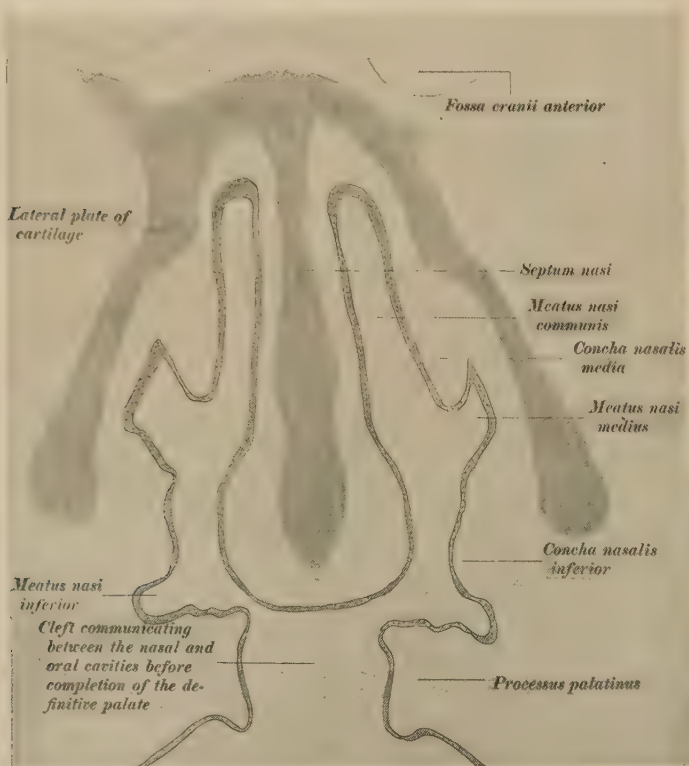


Fig. 2.—A section through the nasal fossæ before the completion of the permanent palate. Human embryo 12 H, aged forty-nine days.

*palate* and to the establishment of communication between the nasal fossæ and the nasal pharynx (Fig. 2). Lack of fusion and the separation of the several nasal and palatine processes lead to cleft-palate and harelip. Divers types and degrees of these malformations occur both unilaterally and bilaterally. The septum between the nasal pits narrows and becomes the *nasal septum* proper and divides the nasal cavity into the paired *nasal fossæ* (Figs. 1, 2).

The lateral walls of the nasal fossæ, while at first smooth and even, at an early time present grooves or furrows, the precursors of the *nasal meatus*. The furrows delimit folds which are the precursors of the *nasal conchæ*. Later, cartilage develops within the epithelially covered conchæ which,

together with the cartilage of the lateral nasal walls and the nasal septum, largely undergoes ossification. The nasal mucous membrane also evaginates into neighboring bones, leading to the formation of the *maxillary*, *sphenoidal* and *frontal sinuses*, and the *ethmoidal cells*. The initial points of outgrowth remain as the ostia of the adult sinuses and cells.

The *paranasal sinuses* are preformed in the nasal meatus and the secondary furrows which mould the lateral wall of the nasal fossæ. This is true of all the paranasal sinuses save the sphenoidal, which arises in connection with the posterior cupola of the cartilaginous nasal capsule, and may be considered a constriction of the nasal mucosa from the region above and behind the highest turbinate present. No paranasal sinus develops from the inferior nasal meatus.

The pre-existing spaces from which paranasal sinuses and cells develop are: (1) The frontal furrows; (2) the frontal recess, both of the ascending rami of the middle nasal meatus; (3) the suprabullar recess; (4) the bullar furrow; (5) the infrabullar furrow; (6) the ethmoidal infundibulum, all of the descending rami of the middle nasal meatus; (7) the anterior extremity and the superior and inferior recesses of the superior nasal meatus; (8) the supreme nasal meatus; (9) the pheno-ethmoidal recess.

#### THE EXTERNAL NOSE

The external nose, shaped like a triangular pyramid, is composed of a bony and cartilaginous framework, covered externally by the integument, a sparse subcutaneous tela and certain muscles, which functionally are

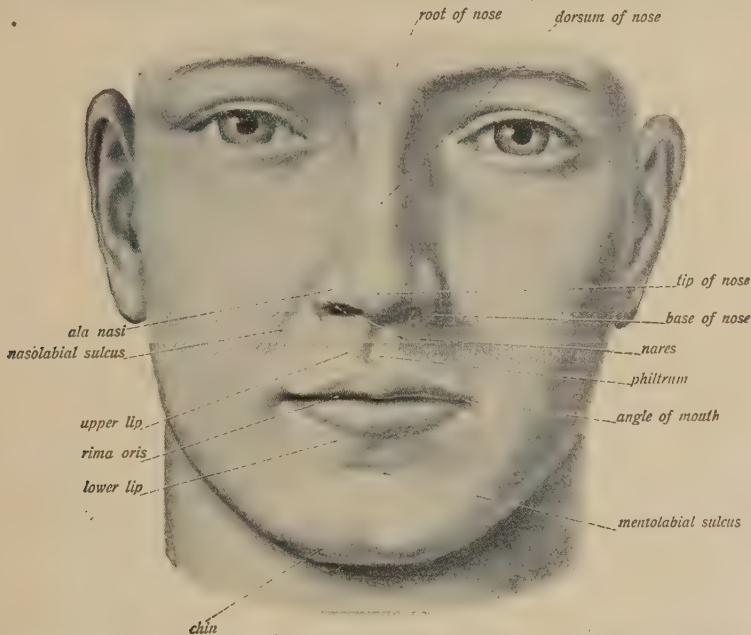


Fig. 3.—The mouth, chin, and nasal region seen from in front. (Sobotta and McMurrich.)

classed as dilators and contractors, and lined internally, and variously so, by periosteum and perichondrium, overspread by mucous membrane.

The *root of the nose* is located at the forehead between the eyes, from which extends the *dorsum of the nose* distally to the *apex of the nose*. The

proximal or superior part of the dorsum is known as the *bridge of the nose*. Distally and inferiorly, overhanging the upper lip, is the *base of the nose*, which presents two orifices, the *nares* or nostrils, separated by the movable part of the nasal septum. The sides of the external nose (*partes laterales nasi*) slope variously from the dorsum, laterally and posteriorly to terminate on each side in the *margin of the nose* (*margo nasi*), and inferiorly and posteriorly in the *expanded and convex wings of the nose* (*alæ nasi*).

The dorsum of the nose normally is straight, convex or concave; the apex in line with the dorsum, depressed or upturned. Differences in the proportion of breadth and length of the external nose distinguish three fundamental types of nose, indicated by the *cephalometric nasal index* (greatest breadth  $\times 100$  divided by the greatest length), one of the most distinctive racial characteristics. Individual and family characteristics are commonplace and of less value. However, for cosmetic reasons and purposes these cannot be ignored in plastic surgery (Fig. 3).

The framework of the external nose is composed of bone and hyaline cartilage. The bones that give shape and support to the lesser and superior portion of the external nose are the nasal bones (the nasal bridge), the frontal processes and the anterior nasal spines of the maxillæ, and the nasal part of the frontal bone. The latter occupies the ventral portion of the ethmoidal notch and projects beneath the nasal bones and the frontal processes of the maxillæ, thereby lending support to the bridge. The arch-like construction of the combined nasal bones and the frontal processes of the maxillæ makes for strength. The entire dorsum of the external nose is further supported by the unit action of the elements entering into the composition of the nasal septum. Especially should this be borne in mind in connection with the mobile and projecting portion of the external nose. Operative procedures should take cognizance of this natural and normal support.

#### THE NASAL BONES

The nasal bones (*os nasales*) vary much in size and shape. They are relatively large and prominent in the white races and small and less prominent and flat in the black and yellow races. They are not infrequently reduced in size and altered in shape and symmetry by the encroachment of the frontal processes of the maxillæ, even to complete replacement. Ape-like, the two nasal bones occasionally fuse and obliterate the internasal suture. Both nasal bones may be absent, the perpendicular plate of the ethmoid coming to the surface between the enlarged frontal processes of the maxillæ. Again, each nasal bone appears as several smaller elements. Supernumerary ossicles or plates of bone occasionally are added in the several suture planes. All of these variations are important when interpreting Roentgen plates of the region following an injury. The inferior free border of the nasal bones and the maxillæ form the osseous pyriform aperture (Fig. 4).

#### THE NASAL CARTILAGES

The nasal cartilages (*cartilagines nasi*) are located about the pyriform aperture and constitute the larger portion of the framework of the external nose, also enter into the composition of the nasal septum. There are five principal cartilages: superiorly, the two lateral nasal; inferiorly, the two greater alar, and the unpaired median (septal). Besides these there are the lesser alar, the sesamoid, and the vomeronasal cartilages. The septal and

vomeronasal, together with the medial crura (*vide infra*) of the greater alar cartilages form the cartilaginous portion of the nasal septum. All of the nasal cartilages, according to Schultz, show racial differences in form, and seem to be more developed in size in whites than in negroes (Figs. 4-6).

The **lateral nasal cartilages** (cartilagine nasi laterales) are triangular and more or less flat, lateral expansions of the nasal septal cartilage, placed on each side of the nose, immediately inferior to the nasal bones. Each cartilage presents a deep and a superficial surface and three margins. The medial margin in its superior third is confluent with the anterior margin of the septal cartilage and by way of the latter with its fellow of the opposite side. Inferiorly, a narrow cleft intervenes between the septal cartilage and the lateral nasal cartilages. The curved superior and lateral margin of the

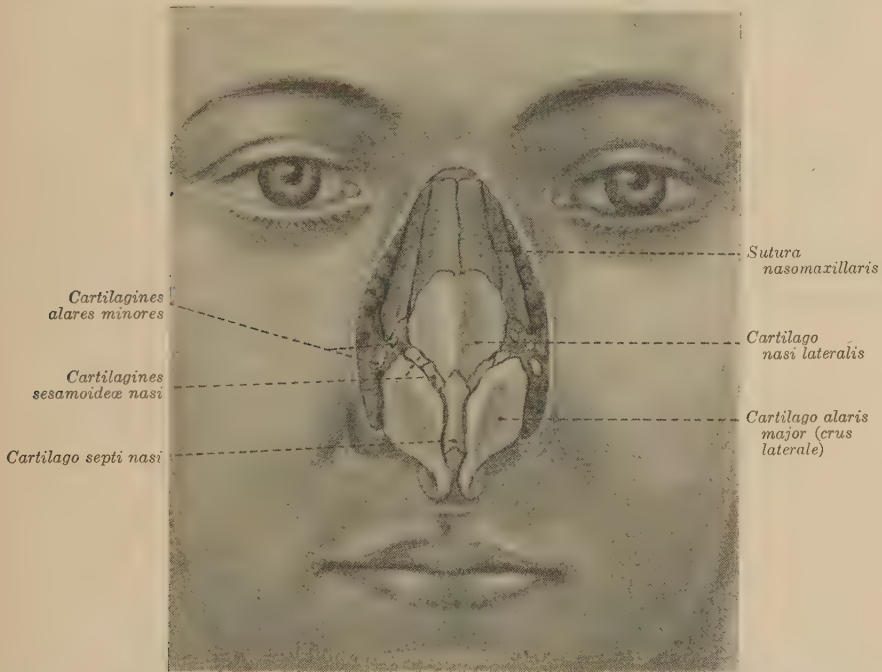


Fig. 4.—The cartilages of the external nose as displayed after the removal of the skin and muscles. Frontal view. (After J. Parsons Schaeffer, *The Nose, Olfactory Organ, and Accessory Sinuses*.)

lateral nasal cartilage is firmly attached by strong fibrous tissue to the nasal bone and the frontal process of the maxilla, and underlies these bones for a considerable distance. The inferior margin is attached to the greater alar cartilage, fibrous tissue and sesamoid cartilages intervening (Fig. 5).

The **greater alar cartilages** (cartilagine alares majores), variable in form, are located one on each side of the nose. They give shape and support to the base and tip of the nose and assist in keeping the nares and vestibules open. Each cartilage is thin and pliant and so curved that it forms a *medial* and a *lateral crus*. The *medial crus* is loosely attached to its fellow of the opposite side; the two, located below or distal to the septal cartilage, conjointly form the lower part of the mobile nasal septum and the tip of the nose. The *lateral crus* is confluent with the medial crus at the apex of the

nose and curves dorsally in the anterior and superior portion of the related wing. It is connected with the nasal margin of the maxilla by a mass of dense fibrous and fatty tissue, containing also the lesser alar cartilages. The greater and lesser alar cartilages together form an incomplete ring about the naris. The angle formed by the mergence of the crura of the greater alar cartilages (*angulus pinnalis*) varies with the shape and type of the nose (Figs. 4-6).

A variable number of small cartilages, the **lesser alar cartilages** (*cartilagine alares minores*), are embedded in the fibrous tissue of the *alæ*. In the space between the greater alar and the lateral cartilages are found the minute **sesamoid cartilages** (*cartilagine sesamoideæ*). These may be re-

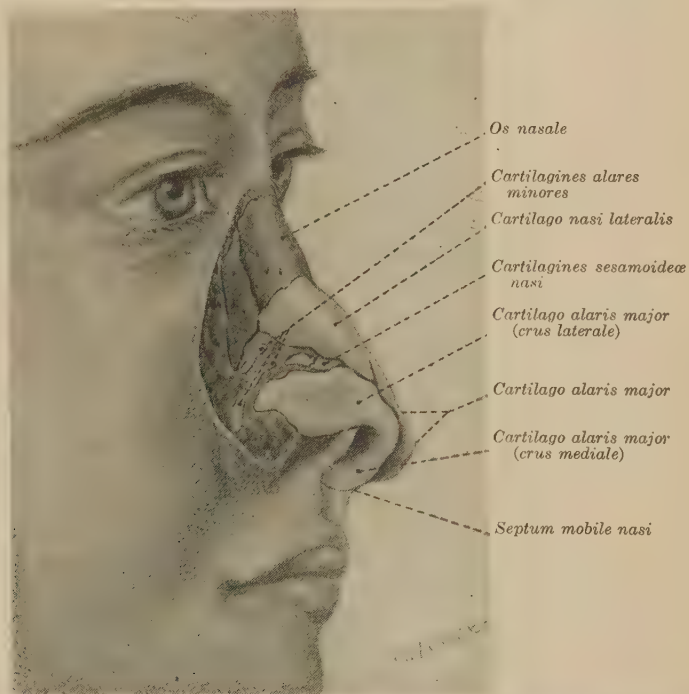


Fig. 5.—The cartilages of the external nose as displayed after the removal of the skin and muscles. Profile view. (After J. Parsons Schaeffer, *The Nose, Olfactory Organ, and Accessory Sinuses*.)

placed in whole or in part by the lateral crus of the greater alar cartilage (Fig. 5).

The **septal cartilage** (*cartilago septi nasi*) is an unpaired, medianly placed, irregular, quadrilateral plate of cartilage which forms the anterior part of the nasal septum, fitting into the triangular interval between the perpendicular lamina of the ethmoid and the vomer. Its anterosuperior margin in its upper part usually meets the internasal suture and projects forward below as far as the *dorsum nasi* to become confluent with and extend between the two lateral nasal cartilages. The most inferior part of this margin of the septal cartilage is located between the greater alar cartilages. The antero-inferior margin extends backward from the rounded anterior angle to the anterior nasal spine, gaining attachment to the medial

crura of the greater alar cartilages and aiding in the formation of the mobile nasal septum. The posterosuperior margin is attached to the perpendicular plate of the ethmoid and the posteroinferior margin to the vomer and the

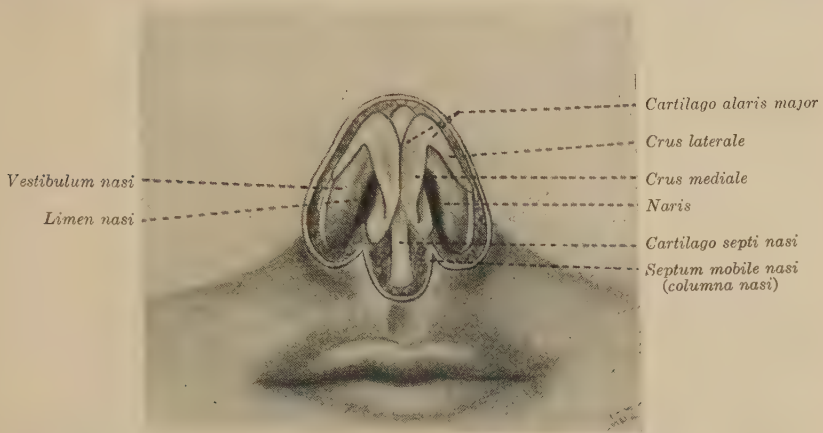


Fig. 6. —The cartilages of the nose in their relationship to the nares (anterior nares) or nostrils. (After J. Parsons Schaeffer, *The Nose, Olfactory Organ, and Accessory Sinuses*.)

anterior part of the nasal crest of the maxillæ and the anterior nasal spine. The size and shape of the septal cartilage varies with the degree of ossification of the osseous septum.

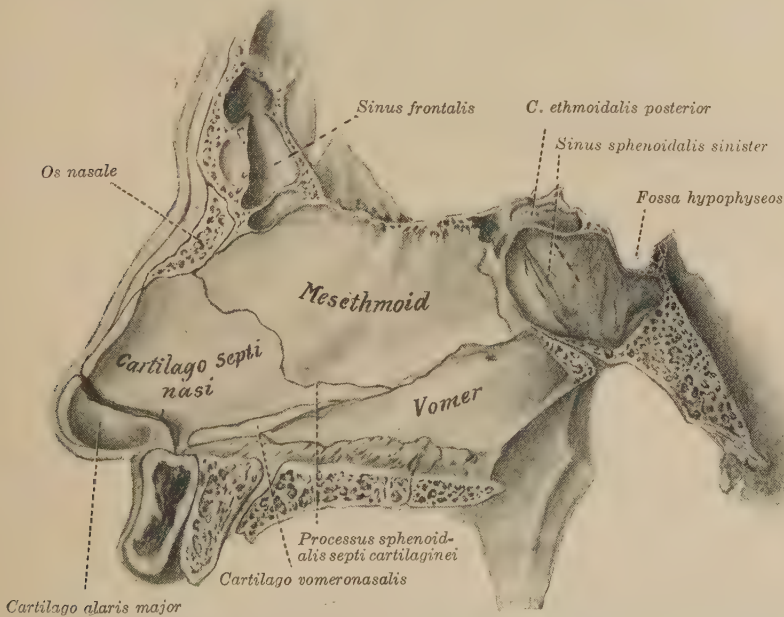


Fig. 7.—A dissection showing the elements that enter into the composition of the septum of the nose. (After J. Parsons Schaeffer, *The Nose, Olfactory Organ, and Accessory Sinuses*.)

The septal cartilage frequently extends between the vomer and the perpendicular plate of the ethmoid, forming the variable but important *spheno-*

*noidal process of the septal cartilage.* The latter may reach the sphenoid bone, especially in the young. It frequently is the seat of a ridge-like, horizontal projection into one or the other nasal fossa, leading to septal asymmetry (Figs. 6, 7).

The **vomeronasal cartilages of Jacobson** (cartilagine vomeronasales Jacobsoni) are two small longitudinal strips of cartilage which lie close over the anterior nasal spine along the anterior portion of the inferior border of the septal cartilage. They are related to the vomeronasal organ of Jacobson and in man reach the maximum development in the embryo.

### THE INTERNAL NOSE

The anatomy of the internal nose is that of the nasal cavity and a goodly number of ancillary and related structures and parts. The general nasal cavity (*cavum nasi*), located between the floor of the cranium and the roof of the mouth, is divided by a medianly placed nasal septum into two more or less symmetrical halves, the nasal fossæ (*fossa nasales*). These communicate with the exterior through the nares (anterior nares) and with the nasopharynx through the choanæ (posterior nares). The nasal fossæ are further divided into nasal meatuses (*meatus nasi*) by the nasal conchæ or

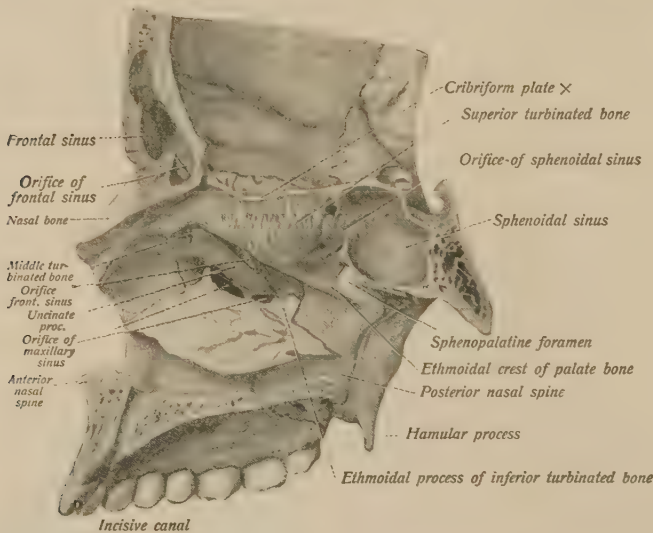


Fig. 8.—View of the lateral wall of the right nasal fossa, the nasal septum having been removed (§). (From Sobotta and McMurrich.)

turbinates (*conchæ nasales*) and are extended typically, and variously so, into neighboring bones as the paranasal (accessory) sinuses (*sinus paranasales*).

In accordance with the more specific functions and the important histological differences the nasal fossæ are divided into the *respiratory* and *olfactory portions* (*regiones respiratoria et olfactoria*). The former, the lower and greater portion of the nasal fossæ, has especially to do with the function of respiration, and the latter, the upper portion of the fossæ, is primarily concerned with the function of smell and may be considered the peripheral olfactory organ. With the exception of the anterior portion of the nasal

cavity, where the boundaries are completed by cartilages and membranes, the walls of the cavity are almost wholly of bone. The unpaired ethmoid, frontal, vomer and sphenoid bones, and the paired palate, nasal, lacrimal and maxilloturbinal bones, and the maxillæ participate variously in the composition (Fig. 8).

#### THE NASAL FOSSÆ

The paired nasal fossæ (*fossæ nasales*) are, generally speaking, triangular in the frontal plane. The narrow roof of each fossa being the apex of the triangle and the wider floor the base; the median or septal wall, normally even and approximately vertical, meeting the floor nearly at a right angle. The lateral wall or hypotenuse of the triangle is sloping and moulded and

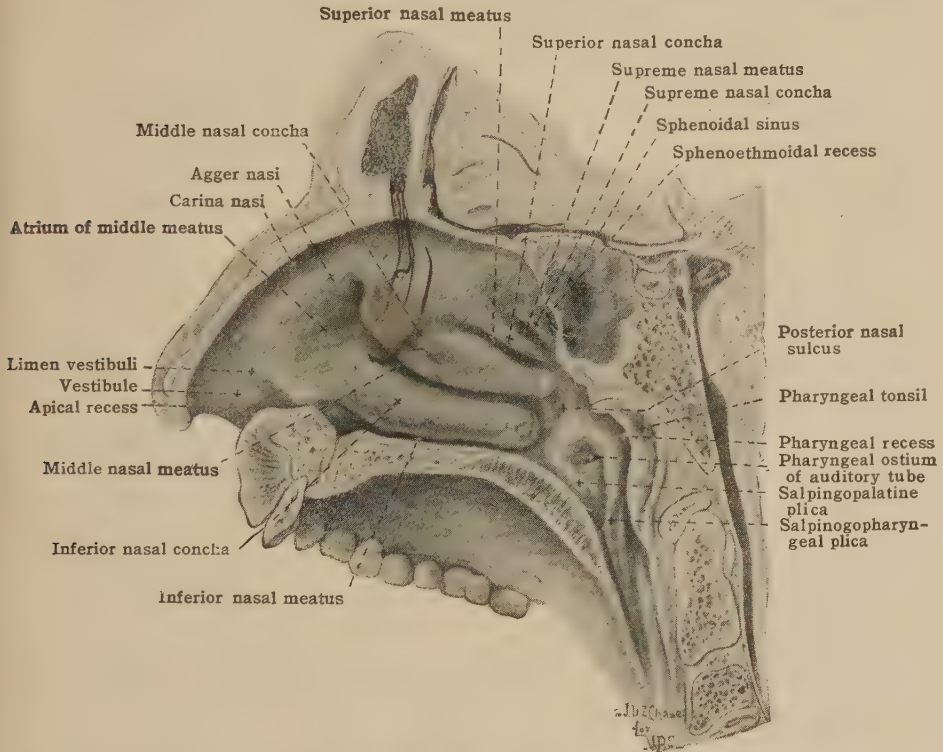


Fig. 9.—Lateral wall of the right nasal fossa and the nasal pharynx. (From Author's article in Morris' Anatomy.)

configured by the nasal conchæ and the meatus and the encroaching paranasal (accessory nasal) sinuses. In the sagittal plane each fossa is quadrangular in outline, the roof and floor roughly paralleling each other, the ventral or anterior side conforming to the profile of the external nose and the dorsal or posterior side passing in front of the body of the sphenoid, through the choana or posterior naris to the junction between the hard and soft palates. Laterally, the posterior limit of the nasal fossa is the posterior nasal sulcus and, medially, the posterior free border of the nasal septum.

The dimensions of the nasal fossæ vary. The following may be given as representative, based upon a series of individuals studied by the author:

The greatest sagittal diameter, measured from the most prominent part of the naris, along the floor of the nasal fossa to the posterior border of the hard palate, is 74 mm.; the greatest sagittal diameter, measured along the roof of the fossa, is 35 mm. or less; the greatest vertical diameter, measured from the cribriform plate to the floor of the nose, is from 40 to 45 mm.; the width of the roof (cribriform plate), 5 mm. or less; the width of the floor, measured at the greatest lateral expansion of the inferior meatus, varies from 12 to 25 mm.

**The Nares (Anterior Nares) and Vestibules.**—The vestibules are the antechambers to the nasal fossæ. They are located immediately ental to the nares, corresponding more or less in extent to the cartilaginous nasal wall from which they gain support. Each vestibule extends into the tip of the nose, forming the *recesses apicis*, and on the lateral wall is delimited from the rest of the nasal fossa by a distinct ridge, the *limen nasi* (limen vestibuli), corresponding to the superior margin of the greater alar cartilage. At the limen nasi the modified skin suffers a change into the mucous membrane of the nasal fossa proper. The skin of the vestibule is beset with large hairs (vibrissæ) and lodges sudoriferous and sebaceous glands. The nares look almost directly caudalward and present individual and racial variations in shape, size, and plane of direction.

**The Choanæ (Posterior Nares).**—The choanæ are the paired passages which communicate between the nasal fossæ and the ventral portion of the nasopharynx (Fig. 9). The apertures are oval in form, the vertical diameter greater than the transverse, and, due to the osseous boundaries, they stand permanently open and free for the ready ingress and egress of the air. The choanæ are located at either side of the free border of the nasal septum and are limited above by the body of the sphenoid and the alæ of the vomer, below by the line of junction of the hard and soft palates, laterally by the medial plates of the pterygoid processes of the sphenoid.

The vertical diameter of each choana varies from 24 to 33 mm.; the transverse diameter at the floor, from 12 to 17 mm.; at the roof, from 7 to 10 mm. The choanal index  $\left( \frac{\text{transverse diameter} \times 100}{\text{vertical diameter}} \right)$ , in the male, averages 61; in the female, 64.5.

Posterior rhinoscopic examination reveals the choanæ, the posterior extremities of the nasal conchæ, and the nasal and the nasopharyngeal meatus (Fig. 9).

**The Roof of the Nasal Fossa.**—The roof may be considered co-extensive with the cribriform plate of the ethmoid, or as cranially arched, with the cribriform plate, forming the *horizontal middle portion*; the body of the sphenoid, together with the wing of the vomer and the sphenoidal process of the palate bone, the *curved posterior portion*; and the frontal and nasal bones, the *curved anterior portion*. Cranially the cribriform plate supports the olfactory lobe of the brain and is perforated for the passage of the olfactory nerves with their meningeal investments and meningeal spaces. Anteriorly, close to the crista galli, is the longitudinal nasal fissure for the transmission of the anterior ethmoidal branch of the nasociliary nerve and the anterior ethmoidal vessels.

**The Floor of the Nasal Fossa.**—The bony framework of the floor of the nose is formed by the palatal processes of the maxillæ and the horizontal processes of the palate bones. The floor of each fossa is horizontal in the

sagittal plane, save for a mound-like elevation immediately inside the limen nasi, and concave in the frontal plane. The *nasopalatine canals*, funnel-shaped tubes of mucous membrane, are located about 2 cm. behind the inner margin of the nares. These are located in the *anterior palatine canals* of the hard palate. The lumen of the membranous canals is usually obliterated in the adult. However, they may be open and establish communication between the nasal and oral cavities.

**The Nasal Septum.**—The nasal septum, or the median wall of the nasal fossa, is formed posteriorly by a framework composed of osseous elements (*septum osseum*); anteriorly, by cartilaginous elements (*septum cartilagineum*); and antero-inferiorly, by integument and subcutaneous tela (*septum membranaceum* or *septum mobile nasi*) (Figs. 6, 7). The nasal mucous membrane covers all portions of both sides of the septum with the exception of the vestibular part, where skin continued through the nares from the exterior replaces it. The mucous membrane at places forms ridge-like elevations and protuberances, *e. g.*, the *tuberculum septi* and the *septal plicæ*. The latter usually regress after birth. However, they may remain and hypertrophy into tumor-like masses in the adult. The tuberculum is a fairly constant elevation on the septum opposite the ventral extremity of the middle nasal concha.

Asymmetry of the nasal septum is commonplace and may involve all of the constituent parts of the septum or be limited to the septal cartilage or the osseous parts, respectively. Generally speaking, septal asymmetry is due to septal deflection as a whole, to a fault of one of its major constituents, or to spurs, ridges, etc. The dorsal or free border of the septum is practically always in the median plane. It must, however, be remembered that the septum may be straight in essence, yet a marked ridge or spur on one or the other side produce an asymmetry. The articulations between the vomer and the septal cartilage and the mesethmoid and the vomer are particularly vulnerable points. This is added to when there is a goodly sized sphenoidal process of the septal cartilage (Fig. 7).

It would appear that incoördinated and unbalanced development and trauma, the latter not infrequently unrecognized at the time (say during birth and afterward), are the most frequent and important etiological factors in septal deformity.

**The Lateral Nasal Wall.**—The lateral wall of each nasal fossa is characteristically configured by three or four projecting and overhanging scroll-like laminae—the *nasal conchæ* or turbinates (*conchæ nasales*). The conchæ incompletely subdivide each nasal fossa into a corresponding number of primary, groove-like passageways—the *nasal meatus* (*meatus nasi*). The meatus are always located caudal and lateral to the corresponding conchæ. A goodly portion of each meatus is operculated by its related concha (Fig. 9). The space between the nasal conchæ and the nasal septum, into which the meatus open, is referred to as the *common nasal meatus* (*meatus nasi communis*). The region posterosuperior to the uppermost nasal concha and anterior to the body of the sphenoid is the *spheno-ethmoidal recess* (*recessus spheno-ethmoidalis*). This contains on its posterior wall the aperture of the sphenoidal sinus.

In front of the anterior extremity of the middle nasal concha is the *aggr nasi*, a ridge-like elevation and the rudimentary homologue of the nasoturbinal of mammals. The shallow depression in advance of the middle

nasal meatus is the *atrium of the middle nasal meatus* (*atrium meatus medii*). The narrow cleft-like space between the *agger nasi* and the inner surface of the *dorsum nasi* is the *carina nasi* or olfactory sulcus. It leads from the nasal vestibule to the roof of the nasal fossa, and if continued along the roof becomes confluent with the sphenothmoidal recess. The lateral wall of each nasal fossa is delimited posteriorly by the shallow *posterior nasal sulcus*. It extends from the body of the sphenoid bone to the juncture of the hard and soft palates. The general region extending from the posterior extremities of the inferior and middle nasal conchæ to the choanæ and limited by the adjacent lateral and medial walls of the nasal fossa, is the *nasopharyngeal meatus* (*meatus nasopharyngeus*) (Fig. 9).

**The Nasal Conchæ and Meatuses.**—The nasal conchæ extend and converge anteroposteriorly on the lateral nasal wall. They have a bony framework and are covered by the mucoperiosteum of the nasal cavity. The middle, superior, and supreme conchæ represent appendages or outgrowths of the ethmoid bone, while the skeleton of the inferior or maxillary concha is an independent osseous element. The nasal meatus are located below and lateral to the corresponding nasal conchæ, the inferior and middle meatus particularly being overhung or operculated by the related conchæ (Figs. 9, 11).

The *inferior nasal concha* (*concha nasalis inferior*), or maxilloturbinal, is an independent scroll-like lamina of bone covered by an exceptionally thick mucous membrane containing an extensive venous plexus, the *plexus cavernosi concharum*. It projects from behind the *limen nasi* to a point from 10 to 12 mm. in front of the choana and overhangs the inferior meatus. The *inferior nasal meatus* (*meatus nasi inferior*) is limited above by the arched and attached border of the inferior concha and below by the floor of the nose. It measures from 4.5 to 5.8 cm. in length, beginning variously from 3.5 to 3.7 cm. behind the tip of the nose. The inferior meatus is narrow anteriorly, expanding rapidly in width and height, to narrow again toward the choana.

On the anterior portion of the lateral wall of the inferior meatus, from 15 to 20 mm. behind the *limen nasi* and from 30 to 40 mm. behind the *naris*, is located the *ostium of the nasolacrimal duct*. Moreover, it is located either at the highest point of the inferior meatus or at varying distances (2–10 mm.) below this point. The ostium usually is a single opening. Duplication and triplication occur. If located high, it is usually wide mouthed and stands permanently open; if located lower, it is more likely to be slit-like, collapsed, or guarded by a fold of mucous membrane, the so-called valve of Hasner (Fig. 22).

The *middle nasal concha* (*concha nasalis media*) is large and hangs valve-like over the middle nasal meatus. It hides or operculates a number of secondary conchæ and furrows of the middle meatus. Its osseous lamina is a part of the ethmoid bone and is covered by a thick mucous membrane, erectile in character. The free border of the concha presents a marked knee, giving rise to a short vertical and a larger horizontal limb. The knee very commonly enlarges, forming a lobule which not infrequently is surmounted by a secondary nodule. Ethmoidal cells commonly pneumatize the concha media (Figs. 10, 30).

The *middle nasal meatus* (*meatus nasi medius*) is the most complex and important of the nasal meatus. For practical and descriptive purposes

it may be divided into *ascending* and *descending rami*. The *descending ramus* is spacious and arched and conforms to the contour of the middle and inferior conchæ. The *ascending ramus*, often referred to as the *frontal recess*, is less roomy and represents an extension frontalward of the middle meatus proper. Removal or turning upward of the middle nasal concha discloses on the lateral wall of the descending ramus immediately below the attached border of the middle concha a conspicuous bleb-like and cell-containing structure—the *ethmoidal bulla*. Below the latter is a sharp crescentic lamella, the *uncinate process*. Between the free border of the uncinate process and the ethmoidal bulla is the cleft-like (15-20 mm. long) *semilunar hiatus*, which, in turn, leads from the middle nasal meatus into a groove of variable depth (1-12 mm.) and dimensions—the *ethmoidal infundibulum* (Fig. 11).

The *ethmoidal infundibulum*, as a rule, ends blindly in the frontal region lateral to the frontal recess, in the form of one or more infundibular anterior ethmoidal cells. Posteriorly it either ends in a pocket or merges gradually with the middle meatus. Occasionally, the ethmoidal infundibulum is directly continuous anatomically with the nasofrontal duct (infundibulum of the frontal sinus) or, in the absence of the latter, with the frontal sinus proper. The ethmoidal infundibulum in its depth contains the apertures or ostia of the infundibular group of the anterior ethmoidal cells and the maxillary sinus (ostium maxillare).

The *suprabullar furrow* or recess, located between the ethmoidal bulla and the attached border of the middle nasal concha, contains the ostia of most of the bullar group of anterior ethmoidal cells, frequently classed as middle ethmoidal cells.

The lateral wall of the middle nasal meatus between the attached border of the uncinate process and the inferior nasal concha is at places *wholly membranous and undefended* by osseous tissue. It presents the *accessory maxillary ostium* (a direct communication between the middle meatus and the maxillary sinus) in from 25 to 40 per cent. of cases (Figs. 8, 10).

The frontal portion of the middle nasal meatus, or the *frontal recess*, is a pouch-like extension with which the frontal group of anterior ethmoidal cells and the frontal sinus communicate. Occasionally the ethmoidal infundibulum and the nasofrontal duct, or the frontal sinus proper, are continuous channels and groove the lateral wall of the frontal recess (Fig. 26). Usually, however, the ethmoidal infundibulum and the nasofrontal duct are anatomically discontinuous channels (Figs. 23, 24).

The *superior nasal concha* (concha nasalis superior) is a short, thin lamina of bone projecting from the lateral ethmoidal mass and slightly overhanging the superior nasal meatus. The mucous membrane of this concha is thinner and less erectile in character than that of the middle and inferior conchæ. The *superior nasal meatus* (meatus nasi superior) is a restricted, channel-like depression below the related concha and approximately half as long as the middle nasal meatus. An accessory concha occasionally moulds its lateral wall and divides the meatus into *superior* and *inferior recesses*. These recesses and the anterior end of the superior meatus receive the ostia of most of the posterior ethmoidal cells (Figs. 10, 15).

The *supreme nasal concha* (concha nasalis suprema) is the smallest of the conchæ and is found bilaterally or unilaterally in 60 per cent. of bodies. It overhangs but slightly the *supreme nasal meatus*. The latter is found in a

corresponding number of bodies, and in approximately 75 per cent. of these cases it contains the ostium (or ostia) of a posterior ethmoidal cell (Fig. 10).

The *spheno-ethmoidal recess* is located immediately above and behind the supreme concha, or the superior concha, when this is the uppermost one differentiated. The recess lies in the angle between the ethmoid and the anterior surface of the body of the sphenoid, and practically always receives the ostium of the sphenoidal sinus (Figs. 9, 10).

#### THE PARANASAL SINUSES

The paranasal (accessory) sinuses (*sinus paranasales*) have their origin and beginning from the third to the fourth month of fetal life as evaginations or outpouchings of certain areas of the mucous membrane of the nasal meatus proper (the inferior meatus excepted), and from the secondary

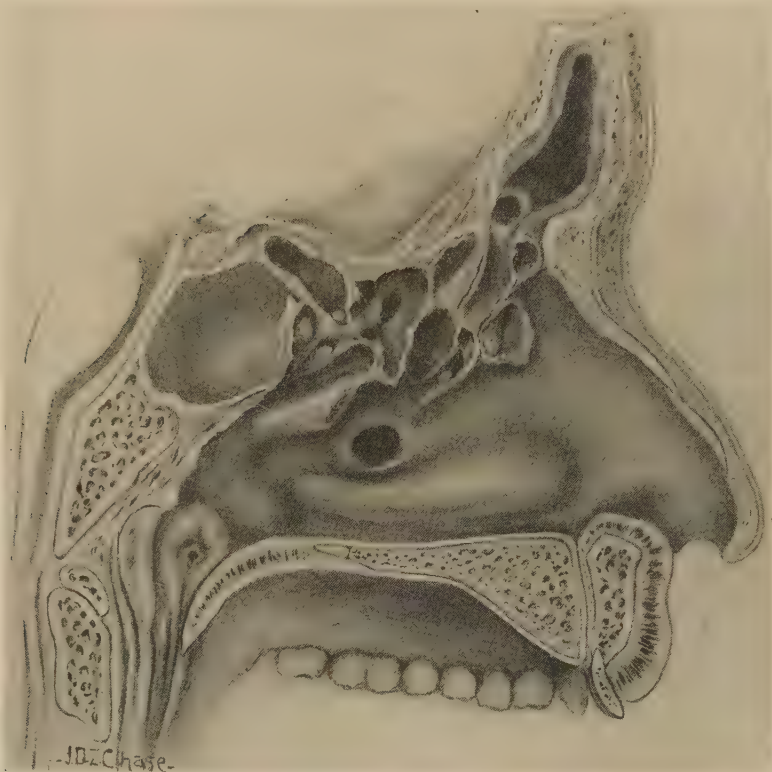


Fig. 10.—A dissection of the lateral nasal wall exposing the frontal sinus, the several groups of the ethmoidal cells, the sphenoidal sinus, and other structures of the lateral nasal wall. A large accessory maxillary ostium is present in the specimen.

meatus or furrows. The sphenoidal sinus is an exception, arising, in a sense, as a constriction from the posterior and superior region of the nasal fossa. It is not an outgrowth from a nasal meatus.

The mucous membrane sacs or early sinuses and cells wander into related bones of the nasal walls, and by growth of the sacs and absorption of bone make pneumatocystic large portions of the ethmoid, frontal, maxillary,

and sphenoidal bones in the formation of the ethmoid cells and the frontal, maxillary, and sphenoidal sinuses. Despite the fact that many of the paranasal or accessory sinuses grow far from the point of initial evagination, the primary points of outgrowth remain in the adult, although modified, as the ostia or apertures of communication between the nasal fossæ and the sinuses. The precise location, form, size, and relations of the ostia of the sinuses and cells differ in the dried skull and the wet or recent specimen and the living body. There is no constancy or ideal in the anatomy of the para-

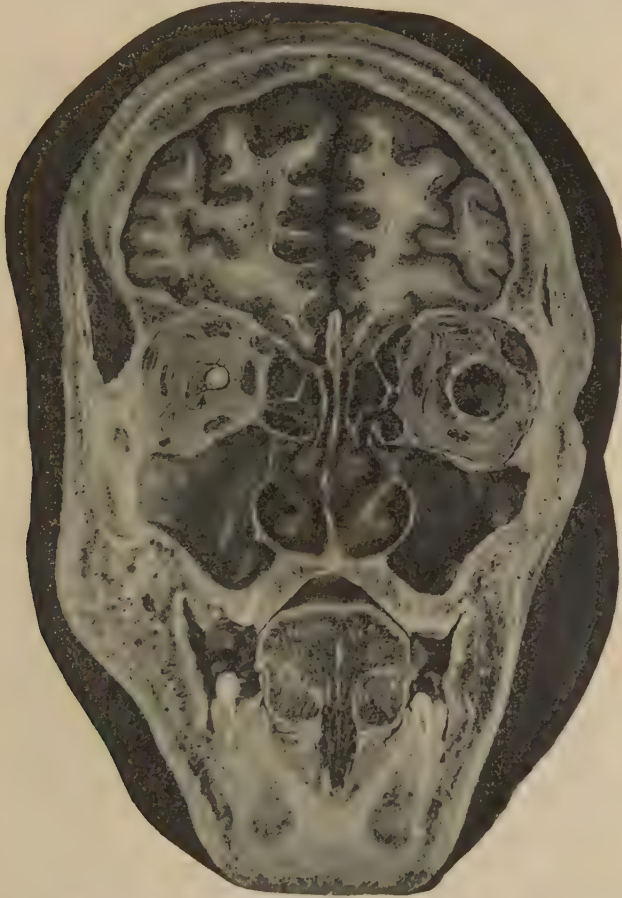


Fig. 11.—Photograph of a coronal or frontal section through the nasal fossæ, ethmoid labyrinth, the maxillary sinuses, and the orbits. On the left side the section passes through the eyeball, on the right immediately behind the eyeball, cutting the optic nerve. On the left side the section happens to pass through the ostium of the maxillary sinus.

nasal sinuses. Great variations in number, size, and type are commonplace. Strictly speaking, there is no so-called normal type. The clinical practice should be to view the problem of the anatomy of the paranasal sinuses from the basis of anatomic types and variations. Fewer errors will be made if the conventional and set type of sinus is forgotten.

With the withdrawal of the olfactory function from the accessory sinuses in the phylogenic history of man, the one conspicuous and probably dominant function remaining is that as an adjunct to respiration and warming

and humidifying the inspired air. In normal conditions there is an air change in the paranasal sinuses during respiration; good ventilation of them being essential to health. It is possible, should the sinuses be replaced by solid bone, that the poise and equipoise of the head would be interfered with. It is unlikely that the paranasal sinuses exert an important influence over vocalization. On the other hand, the nasal cavities profoundly affect this function.

**Maxillary Sinus.**—The maxillary sinus (*sinus maxillaris*, antrum of Highmore) is located in the maxilla and follows in the main the shape of the



Fig. 12.—Photograph of a transection through the eyeballs, ethmoidal labyrinths, and the sphenoidal sinuses. Especially note the intimate relationship of the optic nerves to the posterior ethmoidal cells and the sphenoidal sinuses. The erectile character of the mucous membrane of the nasal septum and the nasal conchæ and the lacrimal sacs is clearly brought out in the illustration.

body of this bone. The sinus may be considered as having three walls, a roof, and a floor. The medial wall forms part of the lateral wall of the nasal cavity and the apex extends variously into the zygomatic process of the maxilla and into the maxillary border of the zygomatic bone. The anterior or ventral wall of the sinus corresponds to the facial surface of the maxilla; the posterior or dorsal wall to the infratemporal surface; the roof to the orbital surface; and the floor to the alveolar process (Figs. 11, 16).

While there is considerable variation in size, average measurements may be given as follows: *posterosuperior diagonal*, 38 mm.; *anterosuperior diagonal*, 38.5 mm.; *supero-inferior*, 33 mm.; *anteroposterior*, 34 mm.; *medio-*



Fig. 13.—Photograph of a dissection showing the relations of the teeth in a child aged seven years.



Fig. 14.—Photograph of a semifrontal section of a child's face aged from sixteen to eighteen months. On the right side the plane of section is through the maxillary sinus. Note the shallowness of the sinus from side to side (not so from before back) and its relationship to the orbit and the infraorbital nerve. It is also worthy of note that the sinus can be reached at this early time from the inferior nasal meatus.

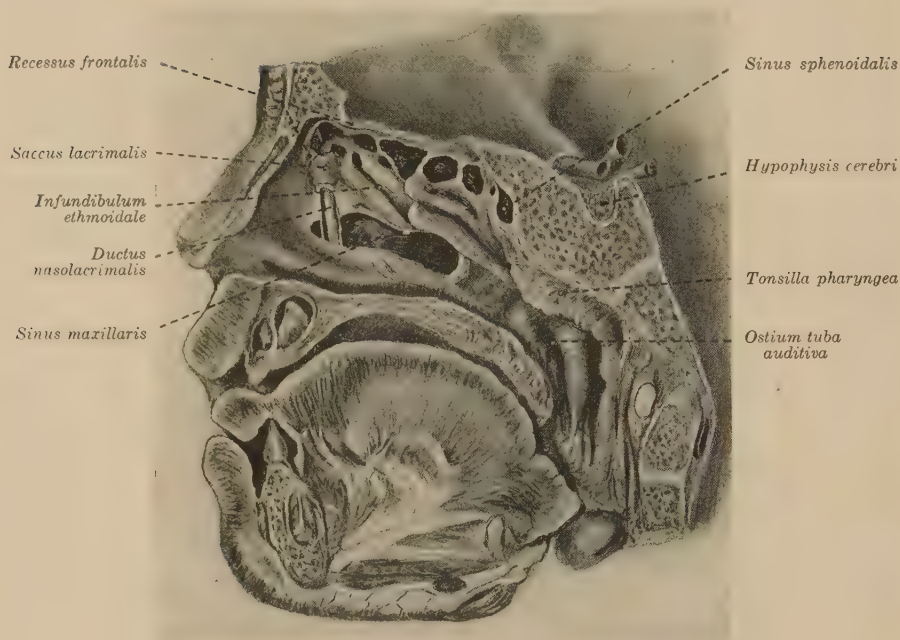


Fig. 15.—The paranasal (accessory) sinuses in a child aged sixteen months, as viewed from the medial side. (After J. Parsons Schaeffer, *The Nose, Olfactory Organ, and Accessory Sinuses*.)

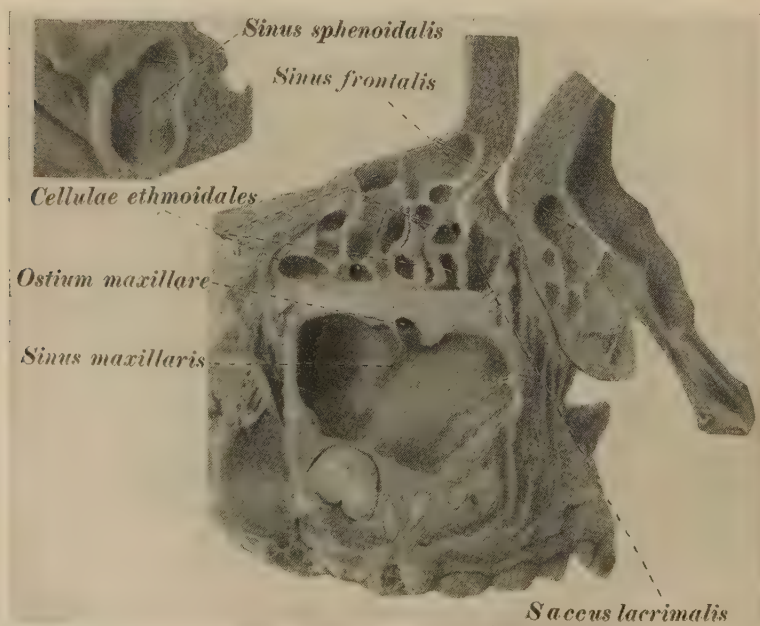


Fig. 16.—Photograph of a dissection of the paranasal sinuses of a child aged six years, six months, and fifteen days. (Dissection by Dr. Davis.) (After J. Parsons Schaeffer, *The Nose, Olfactory Organ, and Accessory Sinuses*.)

*lateral*, 23 mm. More extensive excavation of the alveolar, palatal, frontal, and zygomatic processes of the maxilla give an increased volume. This is added to at times by a definite and marked encroachment of the medial sinus wall on the lumen of the nasal fossa. Thick sinus walls, retention of teeth, lessened hollowing of the outlying processes of the maxilla, and encroachment of the facial wall lead to lessened capacity of the sinus



Fig. 17.

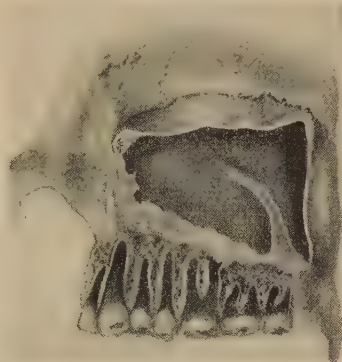


Fig. 18.

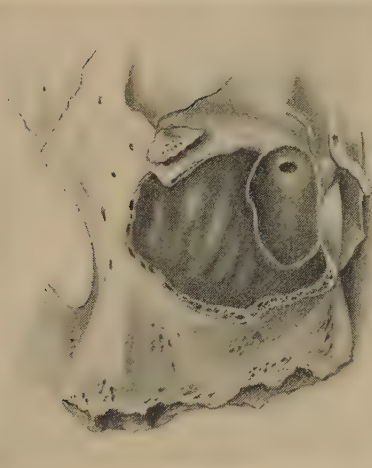


Fig. 19.

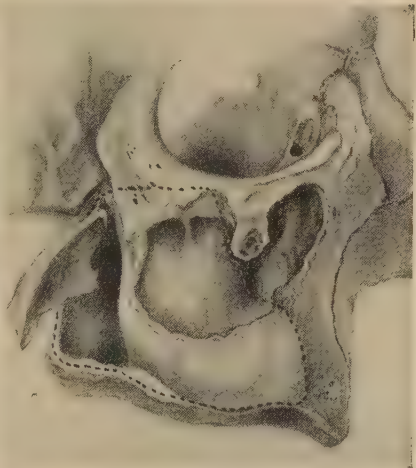


Fig. 20.

Figs. 17-20.—Drawings of four important types of maxillary sinuses.

In Fig. 17 the sinus is more or less even walled and without recesses. A molar tooth molds the floor of the sinus.

In Fig. 18 there is a large osseous septum partially dividing the sinus, leading to the formation of a goodly sized, dorsally placed recess.

In Fig. 19 a posterior ethmoidal cell impinges upon the more usual confines of the maxillary sinus and simulates a duplication of the sinus. Genetically, however, the impinging cell is ethmoidal.

In Fig. 20 there are two true maxillary sinuses, each with an independent aperture into the ethmoidal infundibulum. Operative procedures from the inferior nasal meatus would open into the dorsal maxillary sinus, indicated by dotted outline.

The encroachment of the facial wall and a prominent canine fossa are important in the surgical approach on the sinus from the inferior nasal meatus. In these cases the maxillary sinus is greatly restricted or wholly absent in the anterior and medial portion of the alveolar process (Fig. 21).

Not infrequently the maxillary sinus is incompletely divided into *sub-compartments* and *recesses* of various depths by membranous and osseous septa (Fig. 18). At times the compartments are of such magnitude that they require individual attention in diseased states. The clinician and the roentgenologist should bear the condition in mind in cases which fail to yield to the usual treatment. Rarely one meets with specimens in which *two completely separate maxillary sinuses* are found on one or both sides, each with its own ostium of communication with the infundibulum ethmoidale of the middle meatus (Fig. 20). The author observed four such cases. Each sinus is, of course, entirely independent of the other.

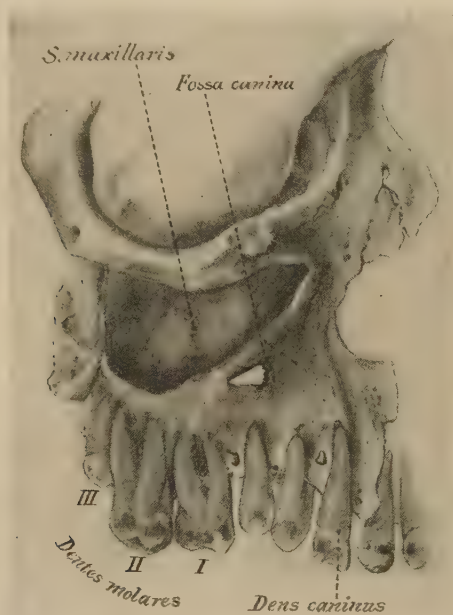


Fig. 21.—A dissection showing the relations of the permanent teeth to the maxillary sinus. The first molar and the two premolars are not intimately related to the floor of the sinus, owing to a very prominent canine fossa. A perforator pushed from the inferior nasal meatus wholly missed the maxillary sinus. This would not be so were the anterosuperior diagonal of the maxillary sinus greater, *e. g.*, the alveolar process of the maxilla more extensively pneumatized. (See Figs. 17 and 18 for comparison.) (After J. Parsons Schaeffer, *The Nose, Olfactory Organ, and Accessory Sinuses.*)

A fairly common condition is the growth into the maxilla and the encroachment on the maxillary sinus of a posterior ethmoidal cell—ethmo-maxillary cell (Fig. 19). The size and degree of encroachment vary greatly. Anatomically, the condition simulates duplication of the maxillary sinus, while clinically the similarity is even more striking. However, it must be borne in mind that the cell which looks and behaves like an additional maxillary sinus communicates with the superior meatus. This rules it out as a true maxillary sinus.

The maxillary sinus communicates with the deep aspect of the posterior half of the *ethmoidal infundibulum* by means of an oval or elongated aperture or ostium—the *ostium maxillare* (Fig. 11). The *semilunar hiatus* is the slit-like space between the free border of the uncinate process and the

ethmoidal bulla, connecting the middle nasal meatus with the ethmoidal infundibulum. The ostium of the maxillary sinus is disadvantageously placed as a drainage aperture, since it is located at the extreme upper part of the sinus and opens into the narrow, deep, and restricted ethmoidal infundibulum. The ostium may be double. It varies in size from a minute opening to a complete replacement of the floor of the infundibulum.

In approximately one-third of all individuals there is an additional aperture—the *ostium maxillare accessorium*—which communicates directly between the middle nasal meatus and the maxillary sinus. It is located either directly behind the ethmoidal infundibulum, even projecting into it, or, as is more customary, between the posterior third of the uncinate process and the related part of the inferior nasal concha. The accessory ostium is more advantageously placed as a drainage aperture, being lower, frequently larger and more direct than the constant and more normal ostium within the ethmoidal infundibulum. Moreover, it is the more accessible in exploration and irrigation of the maxillary sinus (Fig. 10).

The *number of teeth* that bear *direct relations* to the floor of the maxillary sinus varies with the degree of excavation of the alveolar process of the maxilla. The teeth most constantly in direct relation are the three molars and the second premolar, and, when the sinus is small, the second and third molars only (Figs. 13, 18, 21). The mucous membrane of the maxillary sinus is not infrequently pushed into relief by the roots of the teeth, this alike in the young and the old.

The majority of maxillary sinuses have their *floor below the level of the floor of the nasal fossa* and markedly below the usual point of surgical entrance from the inferior nasal meatus. At times almost one-half of the vertical extent of the sinus is caudal to the point of perforation, an important factor when one considers dependent drainage (Figs. 11, 13).

*At birth* the maxillary sinus is of relatively large size, not infrequently measuring  $8 \times 4 \times 6$  mm. It is important to note that at this time the sinus is slit-like in shape and hugs the lateral nasal wall closely, its greatest diameter being from before backward. The narrow transverse or medio-lateral diameter must ever be kept in mind in surgical procedures on the sinus in infancy (Fig. 14). At the end of the first year the sinus is still medial to the infraorbital foramen, at the end of the second year has reached this foramen, and at five years has extended beyond it. While the age of the child is an invaluable guide in the determination of the degree of development of the maxillary sinus into the alveolar process, when the sinus is operated upon by way of the nasal fossa it remains for the roentgenologist to give the surgeon the definite and precise information. The rudiments of both the deciduous and the permanent teeth, of course, also are contained within the body of the maxilla, and these may throw confusing shadows. At one year the sinus measures in length 16 mm.; in height, 6 mm.; in width, 5 mm.; at three years,  $23 \times 12 \times 10$  mm.; at six years,  $28 \times 17 \times 17$  mm.; and at eight years,  $29 \times 17 \times 18$  mm., comparable adult measurements being  $34 \times 33 \times 23$  mm.

**Frontal Sinuses.**—The frontal sinuses (*sinus frontales*) are located between the outer and inner tables of the frontal bone. They vary in size, shape, type and number, and are usually asymmetrical. Not infrequently one of the sinuses encroaches markedly upon the confines of its fellow, with a corresponding displacement of the intervening *septum sinuum frontalem*.

The frontal sinuses occasionally extend beyond the confines of the frontal bone into the sphenoid, the nasal, the parietal, and by way of the crista galli into the ethmoid.

The more usual frontal sinus is roughly pyramidal and occupies the squama frontalis or vertical portion of the frontal bone. However, commonly the shape is greatly modified by the marked extension of the sinus over the orbit into the pars orbitalis of the frontal bone (Fig. 29). Indeed, the vertical portion of the frontal bone may have less of the frontal sinus than does the horizontal part. Either part of the sinus may be absent.

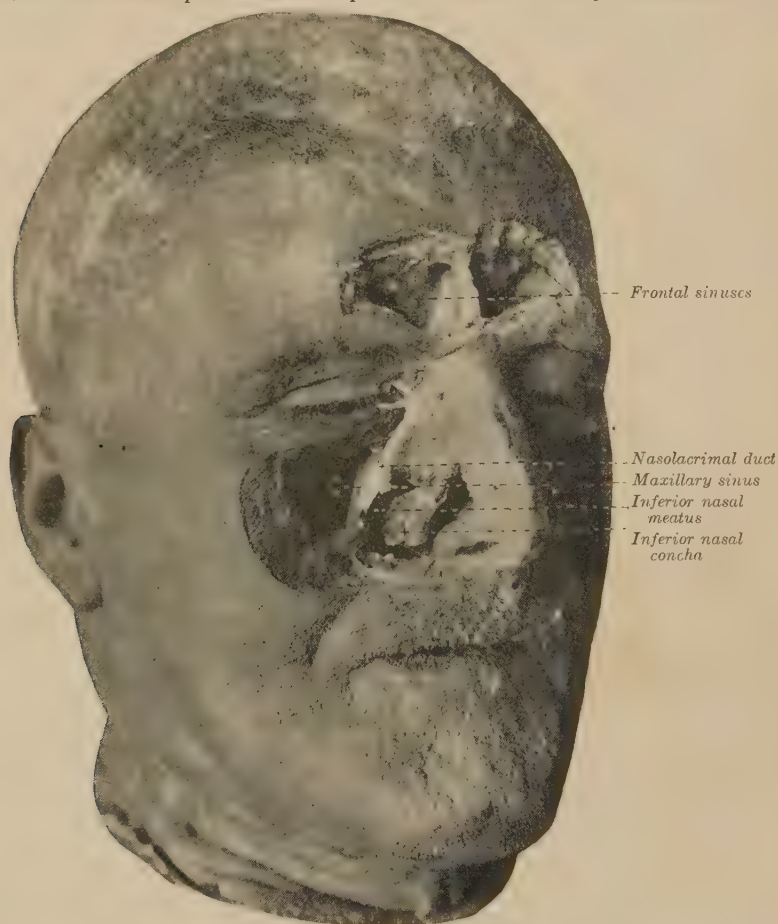


Fig. 22.—Photograph of a dissection of the frontal sinuses, the right maxillary sinus, the efferent lacrimal passageways, the inferior nasal meatus, and the inferior nasal concha.

The frontal sinuses vary from simple chambers to those in which partial subcompartments and recesses are formed by incomplete membranes and bony partitions. Supernumerary sinuses are common, each with its own connection with the nasal fossa. Schaeffer has observed as many as six frontal sinuses in one skull. Multiple frontal sinuses are variously placed: side by side, in the sagittal plane; one over the other, in the horizontal plane; or one behind the other, in the frontal plane (Figs. 27–29). Rarely one or both sinuses are wholly wanting.

The frontal sinus most commonly communicates with the *frontal recess* of the middle nasal meatus either by way of the *nasofrontal duct* (infundibulum of the frontal sinus), with proximal and distal frontal ostia, or by a relatively large, direct, single frontal ostium. Occasionally the nasofrontal duct or the frontal sinus proper is directly continuous with the infundibulum ethmoidale (Fig. 26). As a rule, however, they are anatomically discontinuous (Fig. 23). In approximately one-half of all cases the



Fig. 23.

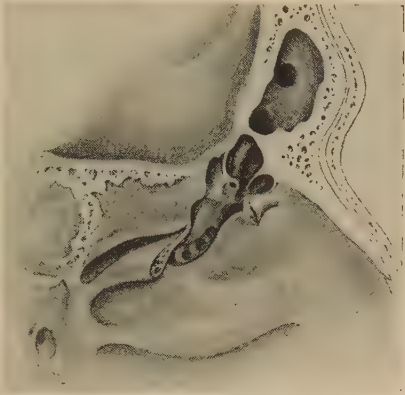


Fig. 24.

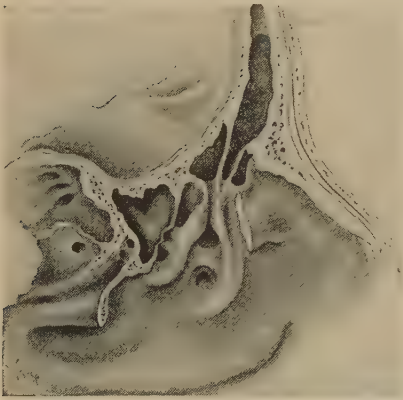


Fig. 25.



Fig. 26.

Figs. 23-26.—Types of nasofrontal connections.

In Fig. 23 there is indication that there were in the early embryonal period four frontal anterior ethmoidal cells. The second cell developed into the frontal sinus. The infundibulum ethmoidale and the nasofrontal duct are discontinuous channels.

In Fig. 24 frontal anterior ethmoidal cells encroach upon the communication of the frontal sinus with the frontal recess. The nasofrontal duct is narrow and tortuous.

In Fig. 25 there is no true nasofrontal duct, the frontal sinus proper coming well down to the region of the frontal recess. The ethmoidal infundibulum is widely open and ends blindly in an infundibular cell.

In Fig. 26 the nasofrontal duct and the ethmoidal infundibulum are in direct anatomic continuity.

relationship between the nasal end of the frontal sinus, or its duct, and the frontal end of the ethmoidal infundibulum is so intimate and of such a character that some or all of the secretion from the frontal sinus readily drains toward and into the ethmoidal infundibulum. Certain anterior

ethmoidal cells likewise drain into the ethmoidal infundibulum (*vide infra*) (Fig. 30). The location of the ostium of the maxillary sinus in the depth of the ethmoidal infundibulum makes it possible for infectious materials from the frontal sinus, etc., to drain into the maxillary sinus.

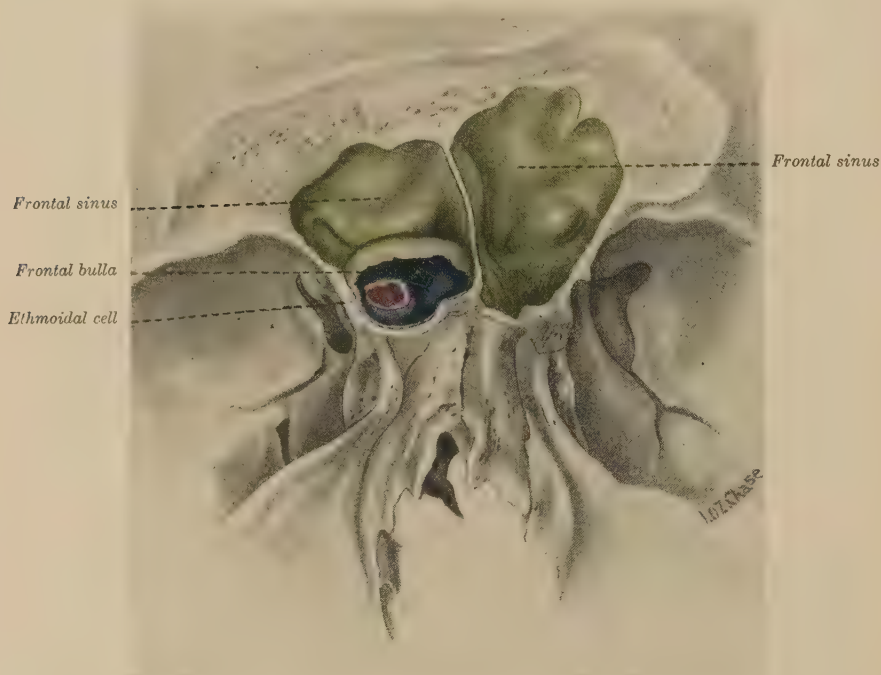


Fig. 27.—A dissection showing anterior ethmoidal cells arranged tier-like in the floor of the right frontal sinus. Should the frontal sinus proper of this side be absent, the frontal bulla would, of course, be classed as a frontal sinus. The same is true for the ethmoidal cell. All three air spaces on the right side of the specimen are topographically frontal sinuses. They all communicate with the frontal recess of the middle nasal meatus.

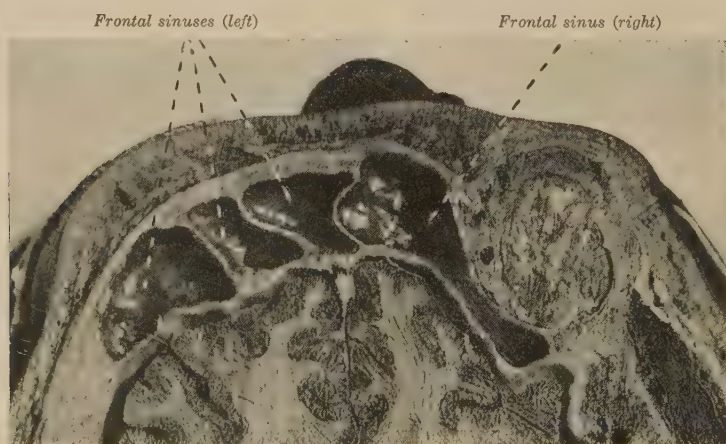


Fig. 28.—A semitranssection through the frontal sinuses. On the right side there is one frontal sinus, while on the left there are three frontal sinuses. It should be noted that the supernumerary frontal sinuses stand side by side in the sagittal plane and project into the vertical portion of the frontal bone.

The efficiency of the *nasofrontal duct* as a drainage or aëration channel is in direct ratio to its length, directness, and diameter. Not infrequently

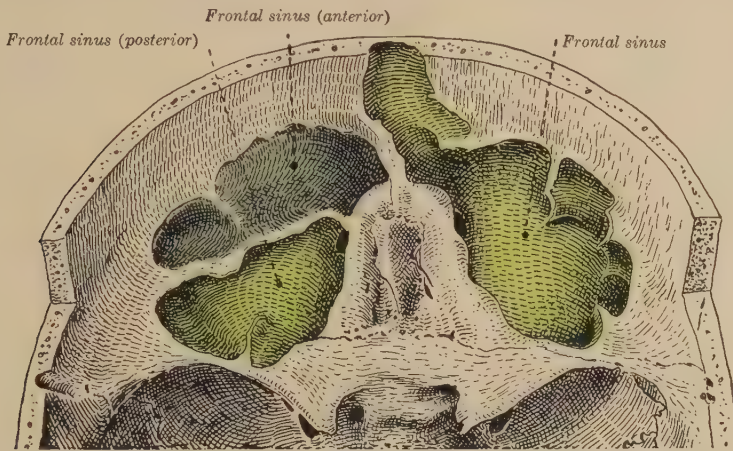


Fig. 29.—A specimen with duplication of the frontal sinus on the left side. Contrary to the specimens shown in Figs. 27 and 28, the sinuses appear one behind the other. (After J. Parsons Schaeffer, *The Nose, Olfactory Organ, and Accessory Sinuses*.)

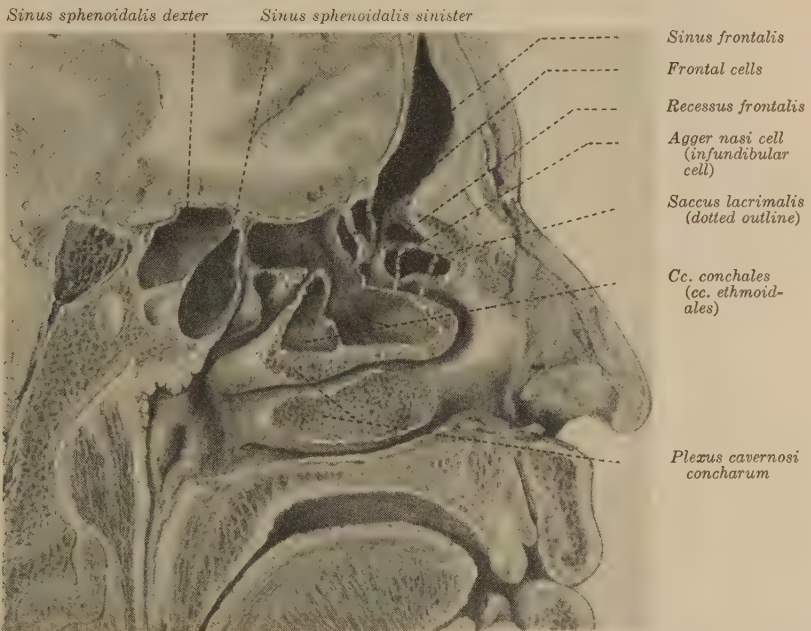


Fig. 30.—The lateral nasal wall with the paranasal sinuses exposed (the maxillary sinus excepted). Particularly note the extension of posterior ethmoidal cells into the middle nasal concha, forming conchal cells (*cellulæ conchales*). An agger nasi cell (*infundibular anterior ethmoidal cell*) overlays the lacrimal sac. In the endonasal approach of the lacrimal sac and the nasolacrimal duct ethmoidal cells of this type would be opened into before the sac is reached. (After J. Parsons Schaeffer, *The Nose, Olfactory Organ, and Accessory Sinuses*.)

it is constricted and sinuous due to the encroachment of some of the frontal anterior ethmoidal cells (Fig. 24). As said elsewhere, many frontal sinuses

have no true nasofrontal duct, the sinus itself opening into the frontal recess by a direct, single frontal ostium. Such sinuses are readily probed and usually have much freer drainage and better aëration.

Average measurements of the adult frontal sinus may be given as follows: Height, 28 mm.; width, 24 mm.; depth, 20 mm. The combined volume of the right and left frontal sinuses averages 14 c.c., with a range from 1 to 45 c.c.

The frontal sinus develops variously as a direct extension of the whole frontal recess of the middle nasal meatus, from one or more anterior ethmoidal cells which have their genetic points in the frontal recess, and occasionally from the anterior extremity of the ethmoidal infundibulum. The frontal sinus is, therefore, ethmoidal in topography before it is frontal. In the majority of cases one cannot be certain which of the potential

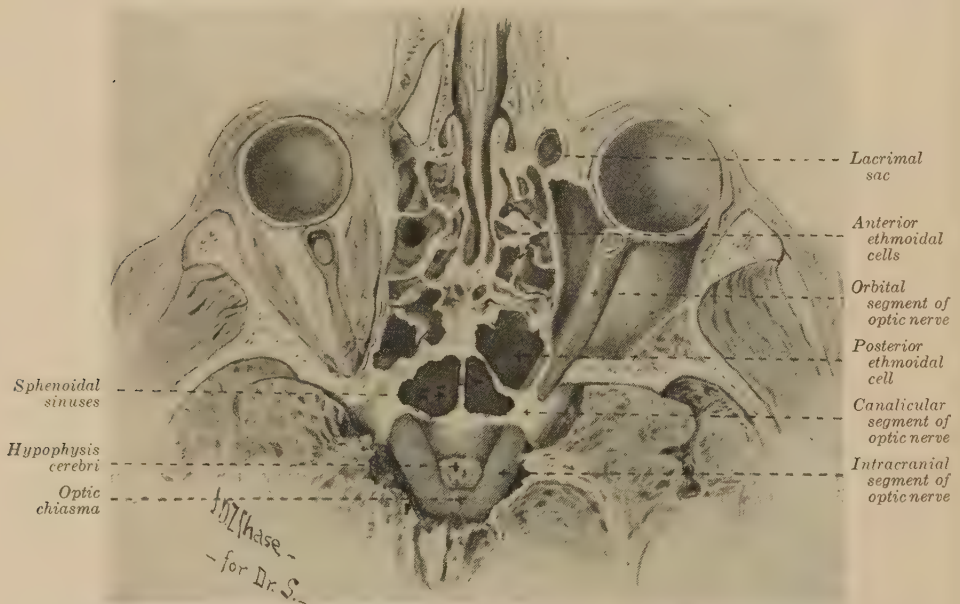


Fig. 31.—A dissection of the optic nerve and chiasma, the sphenoidal sinuses, and the ethmoidal labyrinths. (After J. Parsons Schaeffer, Tr. Sect. Ophth., Am. Med. Assn., 1921.)

rudiments are to form the frontal sinus until the latter half of the first or second year of postnatal life. By the end of the second year the frontal sinus has grown into the vertical portion of the frontal bone, measuring  $5 \times 3 \times 4$  mm. By the sixth year it measures  $8 \times 4 \times 6$  mm., and the cupola extends well above the nasion. At eight years, bearing in mind the normal variation, the sinus may be considered of adult dimensions.

**Sphenoidal Sinuses.**—The sphenoidal sinuses (sinus sphenoidales) hollow out the body of the sphenoid bone and not infrequently extend into the great wings, the pterygoid processes, and the rostrum of the sphenoid and the basilar process of the occipital bone. Rather commonly they replace certain posterior ethmoidal cells, occasionally coming far enough forward to establish immediate relationship with the maxillary sinus. The opposite also occurs, in which one or more posterior ethmoidal cells grow

into the body of the sphenoid bone and restrict the size of the sphenoid sinuses (Fig. 37).

The sphenoid sinuses are usually asymmetrical, with corresponding asymmetry of the intervening *septum sinuum sphenoidalium*. The sinuses vary much in size, shape, and type. Extremely rudimentary and excessively large sinuses are encountered. In conformity with the variations in size the capacity of the sphenoidal sinus varies from 0.5 to 30 c.c., with an average of 7.5 c.c. Average dimensions may be stated as follows: Height, 20 mm.; width, 18 mm.; length, 12 mm. Osseous dehiscences are not in-



Fig. 32.—Dissection through the ethmoidal labyrinth and the sphenoidal sinuses, looking against the roof of the latter. Particularly note the intimate relationship of the optic nerves to the roof and lateral walls of the sphenoidal sinuses.

frequently found, thereby exposing the mucous membrane of the sinus to the structures immediately ectal in relation with the cavernous sinus.

The great variations in size, shape and type, and the common asymmetry and the presence of incomplete septa, recesses, and diverticula profoundly influence the anatomical relationships and add to the difficulties which confront the rhinologist in diagnosis, prognosis, and treatment. In dealing with the sphenoidal sinus clinically one must recall the important structures immediately outside of the thin-walled cavity. Laterally is the cavernous sinus with the internal carotid artery and the abducens nerve coursing through it, and the oculomotor, the trochlear, the maxillary, and the

ophthalmic nerves in its lateral wall. In many cases the mandibular nerve as well has a sinus contact (Figs. 33, 35). In the upper and outer angle the optic nerve almost always pushes the wall of the sphenoidal sinus into a mound-like relief (Figs. 32, 35). This is also commonly so for one or more posterior ethmoidal cells.

In the floor of the sphenoidal sinus is the Vidian nerve and vessels, in the roof the hypophysis cerebri with the optic chiasma immediately over it. Prechiasmal optic pathway involvement, secondary to posterior ethmoidal and sphenoidal disease, is well established and is in keeping with the intimate relational anatomy. Thrombosis of the cavernous sinus also follows diseased states of the sphenoidal sinus and extensions from foci far removed, especially through the anastomoses of the ophthalmic veins with those of the nose and face.

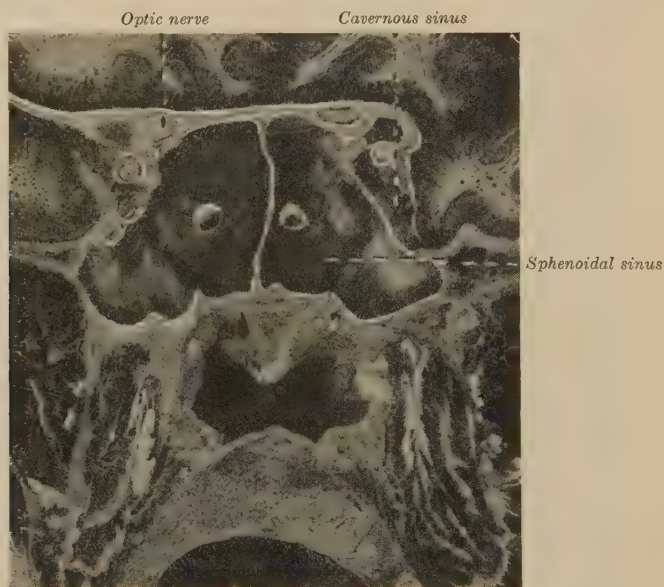


Fig. 33.—Photograph of a frontal section through the sphenoidal sinuses, looking ventrally. Particularly note the symmetry of the sinuses, their relation to the optic nerves, the cavernous sinuses and related structures.

Of great importance clinically is the presence of *depressions*, *recesses*, and *diverticula*. These make the sphenoidal sinuses the most irregular of all the paranasal chambers. Of course, we do find the ideal, that is, sphenoidal sinuses of average size with perfectly even walls.

Recesses, the result of osseous septa, of various size are frequently encountered. The smaller recesses, the result of small septa, probably, may be passed lightly. However, the deeper recesses are largely isolated, never wholly so, from the main sinus cavity (Fig. 34). Indeed, in cross-sections and at times in Roentgen plates one has to contend with what appears to be a duplication of the sphenoidal sinus. However, careful checking always shows that an osseous septum of large size cuts off a part of the main sinus and forms a blindly ending recess or pouch with a restricted connection with the main cavity. These recesses are of various size, and frequently the removal of the anterior sphenoidal wall would fail to establish the de-

sired drainage for them. An important and common recess extension is into the pterygoid process. This may be sufficiently deep to come in contact with the eustachian tube. The accompanying figures better illustrate



Fig. 34.—Frontal section through the sphenoidal sinuses and the left cavernous sinus. Particularly note the osseous septa in both the right and left sphenoidal sinuses, leading to the formation of blindly ending recesses and the appearance of multiple sphenoidal sinuses. However, the partially isolated recesses communicate with the main sphenoidal sinuses.

The inset shows the more usual relationship of the optic chiasma to the hypophysis cerebri and the sphenoidal sinuses.

than words can describe the significance and importance of osseous septa and recesses in clinical work (Figs. 34–36).

The sphenoidal sinus of each side communicates with the respective sphenothmoidal recess, above and behind the highest nasal concha dif-

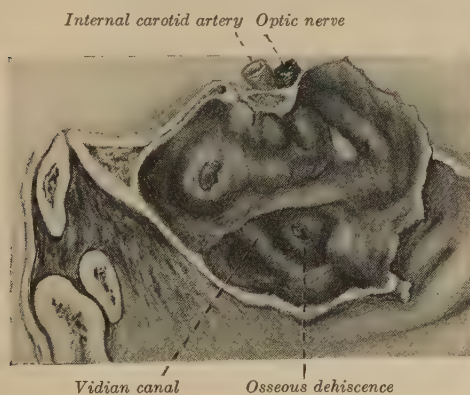


Fig. 35.

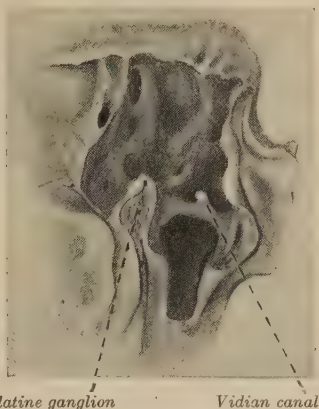


Fig. 36.

Fig. 35.—The lateral wall of the sphenoidal sinus, showing how important structures immediately ectal form mound-like elevations. Osseous dehiscences likewise are present in this case. In the floor will be noticed an elevation produced by the vidian canal and its contents.

Fig. 36.—A dissection showing the relationship of the sphenoidal sinus to the sphenopalatine ganglion. Note the many recesses and the great extension of the sinus into the pterygoid process of the sphenoid. The vidian canal with its osseous wall forms a conspicuous ledge across the pterygoid recess of the sinus.

ferentiated, by means of the *sphenoidal ostium* (apertura sinus sphenoidale). The aperture is large in the dried skull and much reduced in the recent and living state by the respiratory mucous membrane. It is located in the ventral wall of the sphenoidal sinus, from 3 to 20 mm. above the floor. It is disadvantageously placed as an efficient drainage aperture owing to its great distance from the floor of the sinus, averaging 14 mm. (Fig. 33).

The sphenoidal sinus arises primarily in relation with the posterior cupola or terminal nasal sinus of the cartilaginous nasal capsule, the wall of which gives the foundation for the sphenoidal concha or ossicle of Bertin. Strictly speaking, the terminal sinus is the primitive sphenoidal sinus. After the ossicle of Bertin fuses with the body of the sphenoid bone (fourth year of infancy) the sphenoid sinus begins to excavate the body of the sphenoid. The average sphenoidal sinus at birth has a capacity of from 6 to 8 c.mm. By the second year it measures  $4 \times 3.5 \times 2$  mm.; by the fifth year,  $7 \times 6.5 \times 45$  mm.; and by the ninth year,  $5 \times 12 \times 10$  mm.

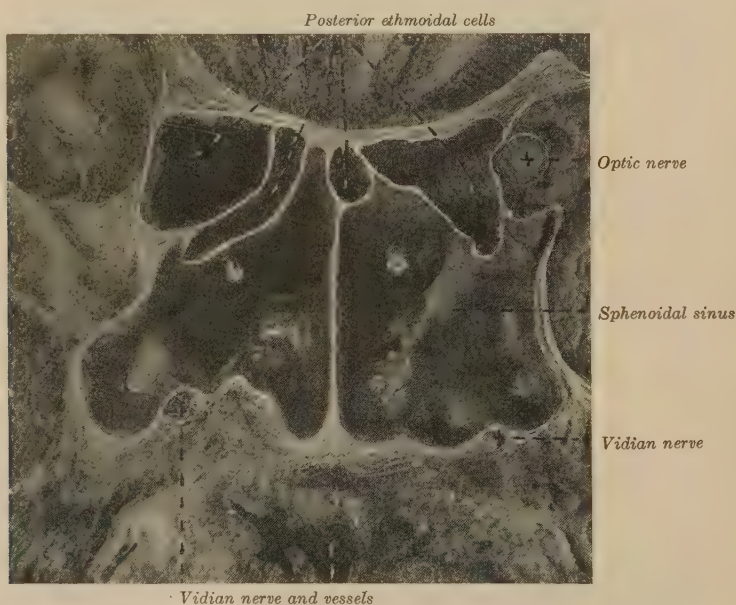


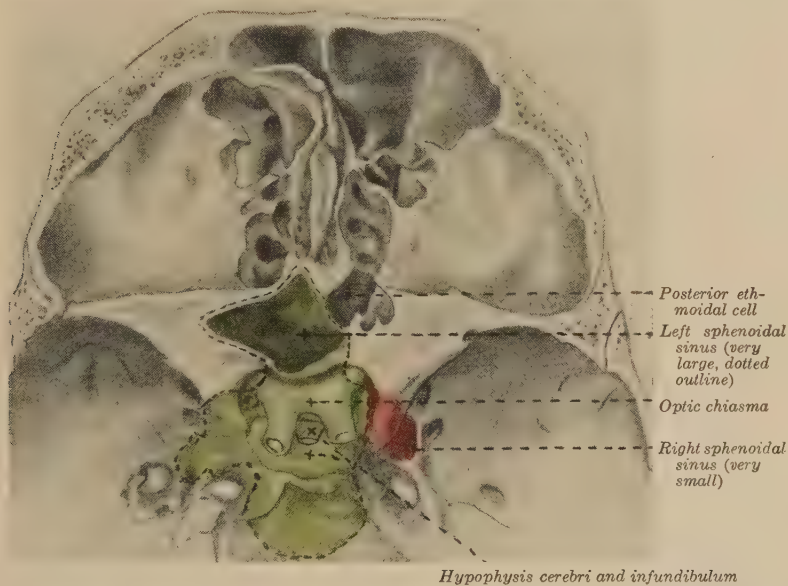
Fig. 37.—Photograph of a frontal section through the region of the sphenoidal sinuses. Note that four posterior ethmoidal cells have grown backward into the body of the sphenoid bone and over the sphenoidal sinuses. In this case the optic nerve is related to a posterior ethmoidal cell and not directly to the sphenoidal sinus. The relation of the vidian canal and its contained nerve and vessels is clearly indicated.

**Ethmoidal Cells.**—The ethmoidal cells (*cellulæ ethmoidales*) honeycomb the lateral masses of the ethmoid bone and collectively form the paired *ethmoidal labyrinths*, located between the upper part of the nasal fossæ and the orbits. The number of cells that compose each labyrinth varies from three to fifteen. Since the entire ethmoidal field is occupied by the related labyrinth, the individual cells are small when many appear and large when few constitute the groups.

It is commonplace for the ethmoid cells to extend beyond the confines of the lateral masses of the ethmoid bone into the ethmoidal appendages, that is, the middle, superior, and supreme nasal conchæ and the ethmoidal

bullae and the uncinate process, forming the *conchal cells* (*cellulæ conchales*); and into the neighboring bones and structures forming the *ethmofrontal*, the *ethmomaxillary*, the *ethmosphenoidal*, the *ethmopalatine*, and the *agger nasi cells*. As a rule, these are merely extensions and enlargements of the cells of the ethmoidal labyrinth proper (Fig. 30).

The ethmoidal cells are divided anatomically, and to a large degree clinically as well, into two primary groups—the *anterior* and *posterior ethmoidal cells*. The *anterior group*, from two to eight in number, communicate with the various parts of the middle nasal meatus. This group is subdivided anatomically into secondary groups, that is, the *frontal anterior ethmoidal cells*, opening into the frontal recess of the middle nasal meatus; the *infundibular anterior ethmoidal cells*, opening into the ethmoidal infundibulum



*Hypophysis cerebri and infundibulum*

Fig. 38.—Dissection exposing the paranasal sinuses from the intracranial side. Note the marked asymmetry of the sphenoidal sinuses (left sinus in green, right in red). The left sinus has a right as well as a left optic nerve contact. It is in cases such as this that sphenoidal sinus disease of one side may produce a temporal narrowing of the visual field on both sides. (After J. Parsons Schaeffer, Tr. Sect. Ophth., Am. Med. Assn., 1921.)

ulum of the middle meatus; and the *bullar anterior ethmoidal cells* (frequently designated the middle ethmoidal cells), opening into the middle meatus, below, above, or upon the ethmoidal bulla, the latter made pneumatocystic by them (Figs. 10, 11). The *posterior group*, from one to seven in number, have their ostia or apertures above the attached border of the middle nasal concha and in communication with both the superior and supreme nasal meatuses. The supreme meatus is found in 60 per cent. of adults, and in 75 per cent. of these it receives the ostium of a posterior ethmoidal cell. The supreme meatus, almost always differentiated, constantly gives origin to posterior ethmoidal cells. Many of the ethmoidal cells have no gravity drainage in the more usual postures of the body, and in this regard are like the maxillary and sphenoidal sinuses. Some simulate the frontal sinus and have gravity drainage (Figs. 10–12).

The position and relations of the *ethmoid labyrinth* are important in diseased states of the part. It is intimately related to the orbit, the thin orbital plate or lamina papyracea alone intervening. Congenital dehiscences are frequently encountered. The anterior group of cells is closely related to the lacrimal fossa and sac. Ethmoidal veins pass from the labyrinth to the cranial cavity and the meninges. The dural cavernous sinus may be secondarily involved by way of the ophthalmic vein, and the superior sagittal sinus via a small vein which traverses the foramen cecum (especially in children).

The extensions of the ethmoidal cells into the primary and secondary nasal conchæ and into neighboring and related bones should always be remembered in clinical work. There is much overlapping of the primary groups of cells, however; the location of the ostia, either above or below the middle concha or turbinate, clearly separates them anatomically and clinically. The rhinoscopic and roentgenographic examinations must go hand in hand.

The ethmoidal cells are primarily extensions or outgrowths of the mucous membrane from the middle, superior, and supreme nasal meatus or from their secondary furrows or recesses. In Roentgen plates of the early postnatal period it is difficult, if not impossible, to distinguish the frontal and infundibular groups of anterior ethmoidal cells from the frontal sinus. In the child at birth the combined anterior group measures, on the average,  $5 \times 2 \times 2$  mm., and the posterior group,  $5 \times 4 \times 2$  mm.; at five years the anterior group,  $8 \times 7 \times 6$  mm.; the posterior group,  $8 \times 10 \times 7$  mm. In the adult the anterior group averages 24 mm. high, 23 mm. long, and 11 mm. wide; the posterior group, 21 mm. high, 21 mm. long, and 12 mm. wide.

#### THE NASAL MUCOUS MEMBRANE

The nasal mucous membrane (*membrana mucosa nasi*) may be considered as related to the three fundamental divisions of the general nasal cavity; *e. g.*, the *nasal vestibule*, the *nasal fossæ*, and the *paranasal sinuses*.

The lining mucous membrane of the *vestibule* is continued from the skin at the naris or nostril and retains for a short distance the surface characteristics of a stratified, flat epithelium, with superficial horny cells. The skin characters are lost at the middle of the vestibule, changing to stratified flat epithelium, devoid of horny cells, resting upon an underlying connective-tissue propria. The fibrous tunica propria is richly provided with elastica, coarse stiff hairs (*vibrissæ*), and sebaceous and sudoriferous glands. In the deeper part of the vestibule mixed seromucous glands replace the sweat glands.

In the *nasal cavity proper*, and in conformity with its double function, there are two areas of the lining mucous membrane that differ in structure—the *pars respiratoria* and the *pars olfactoria*. The latter contains olfactory elements and is limited to the upper third of the nasal septum, nearly the whole of the superior nasal concha, and a small portion of the middle nasal concha. The remaining portion of the mucous membrane is devoid of olfactory cells and is classed as purely respiratory.

The *respiratory portion* of the nasal mucous membrane contains a stratified ciliated cylindrical epithelium. It is intimately attached to the periosteum and the perichondrium of the bones and cartilages of the nose and varies much in thickness in the several parts of the nasal fossa (4–0.5 mm.). Migratory leukocytes are numerous among the epithelial elements. The

tunica propria is composed of fibro-elastic tissue rich in cells and collections of lymphocytes which resemble lymphatic nodules. Numerous glands, mucus secreting, purely serous, and mixed, are encountered.

An extremely rich blood-supply in the tunica propria characterizes the respiratory mucosa of the nose. The relatively great thickness of the mucosa over the inferior and middle conchæ and related portion of the nasal septum is largely due to the abundant cavernous plexuses or blood sinuses (*plexus cavernosi concharum*). The respiratory mucous membrane readily thickens under pathological conditions, often attaining a thickness four or six times normal.

The *olfactory portion* of the nasal mucous membrane consists of a surface neuro-epithelium beneath which is a tunica propria. Strictly speaking, there is no basal membrane. The neuro-epithelium is composed of the specific sensory or olfactory cells (the perceptive elements), the sustentacular cells (the supporting elements), and the basal cells. Fibrous tissue and elastica, lymphocytes, and tubulo-acinar serous glands (olfactory glands of Bowman) are contained in the tunica propria. The glands of Bowman are believed to elaborate a specific secretion.

The perceptive olfactory cells are bipolar and extend through the entire thickness of the neuro-epithelium. Their peripheral processes are short and pass to the surface of the mucosa, where each process ends as a hemispherical vesicle surmounted by six to eight so-called olfactory hairs. The central processes, slender and often tortuous, become grouped into approximately twenty bundles, usually called the olfactory nerves. The latter pass through the apertures in the cribriform lamina of the ethmoid bone and, after piercing the meninges, enter the olfactory bulb of the brain and establish synaptic relationships with the dendrites of the mitral cells.

The olfactory mucosa is less vascular than the respiratory mucosa and is delimited by its yellowish color and greater number of nuclei.

The *paranasal sinuses* are lined by a mucous membrane directly continuous through the several ostia with the lining mucosa of the nasal fossæ. In general, the mucosa of the paranasal sinuses is like that of the respiratory portion of the nasal fossæ. However, it is much thinner, less vascular, and contains less elastica and fewer glands. The current produced by the cilia of the epithelium is always toward the ostium of the respective sinus or cell. It will be recalled that the movement of the cilia of the nasal fossæ is toward the choanæ and the nasopharynx.

### THE BLOOD-SUPPLY

The **arteries** of the nose and the paranasal sinuses are derived from both the external and the internal carotid systems. Branches of the ophthalmic artery (internal carotid system) and branches of the internal and external maxillary arteries (external carotid system) participate in the supply (Figs. 39, 40).

The *sphenopalatine artery* (terminal branch of the internal maxillary) is the main arterial supply to the nasal fossa and its appendages. It enters the nasal fossa through the sphenopalatine foramen which is located on the lateral nasal wall near the dorsal extremity of the superior nasal meatus. The sphenopalatine artery is accompanied through the foramen by the corresponding vein and the sphenopalatine nerve. Immediately after the artery has made its appearance in the nasal fossa it gives off a branch to

the sphenoid sinus and a variable branch which may replace the pharyngeal artery.

The main trunk of the sphenopalatine artery divides into *medial* and *lateral* branches. The *lateral branch* divides into the *lateral posterior nasal arteries*, which supply the nasal conchæ and meatus and the maxillary sinus and the ethmoidal cells, and anastomoses with the anterior and posterior ethmoidal arteries and the lateral nasal branch of the external maxillary (facial) artery. The *medial branch* of the sphenopalatine artery, often called the *nasopalatine artery*, crosses the roof of the nasal fossa dorsally to reach the medial nasal or septal wall. After reaching the septum the nasopalatine artery divides into the *posterior nasal septal arteries*, some

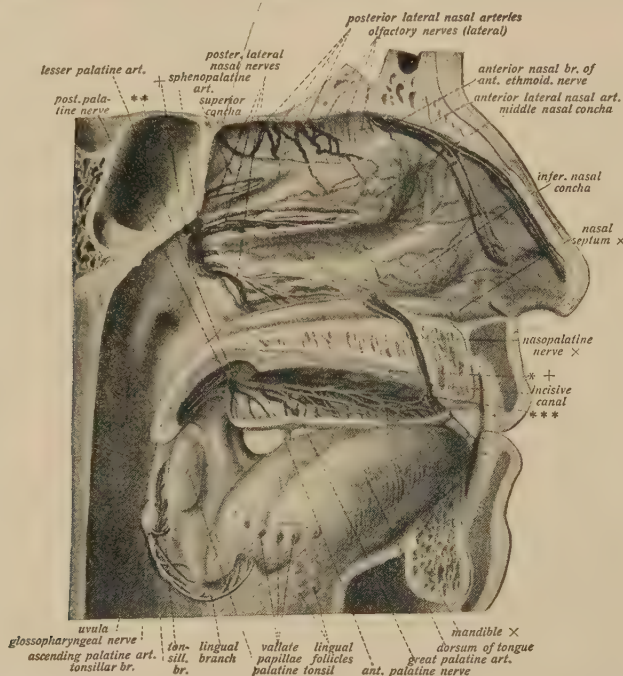


Fig. 39.—The nerves and arteries of the outer nasal wall and of the palate. The tongue has been drawn out, all of the nasal septum except its lower portion removed, and the mucous membrane of the faucial isthmus divided along the glossopharyngeal nerve and the ascending palatine artery. \*\* = Sphenoidal sinus. + = Divided branches to nasal septum. \*\*\* = Anastomosis between nasopalatine and anterior palatine nerves. \*+ = Mucous membrane of hard palate. (Sobotta and McMurich.)

of which accompany the nasopalatine nerve across the septum to the incisive foramen and beyond.

The *anterior* and *posterior ethmoidal arteries* arise from the ophthalmic artery as it courses along the medial wall of the orbit. The *anterior ethmoidal artery* passes through the anterior ethmoidal foramen into the anterior cerebral fossa, accompanied by the anterior ethmoidal branch of the nasociliary nerve. Here it gives branches to the frontal sinuses and the anterior ethmoidal cells. It enters the nasal fossa through a slit-like foramen at the side of the crista galli. Its terminal branch passes along the under surface of the nasal bone and emerges on the external nose between the nasal bone and the lateral nasal cartilage. In the nasal fossa it supplies *medial*

and *lateral nasal branches* to the anterior and upper part of the nose and the related air sinuses and cells. The *posterior ethmoidal artery*, smaller and less constant than the anterior ethmoidal, passes through the posterior ethmoidal foramen and supplies the posterior ethmoidal cells and the related portions of the nasal septum and the lateral nasal wall. The posterior ethmoidal branch of the ophthalmic anastomoses with the branches of the sphenopalatine from the internal maxillary, forming a plexiform network.

The *descending palatine artery* arises from the internal maxillary, descends in the pterygopalatine canal with the anterior palatine branch of the sphenopalatine (Meckel's) ganglion, and in its course supplies small branches to the dorsal portion of the nasal fossa. Its terminal branch, the

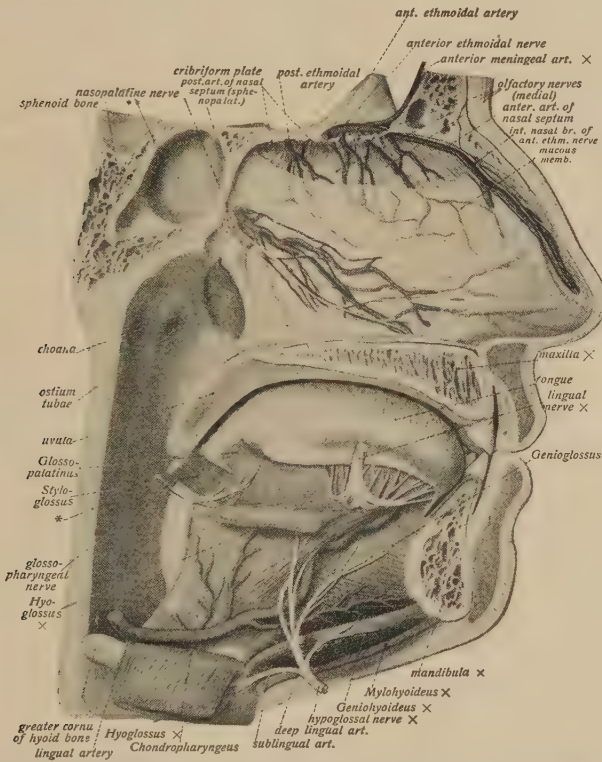


Fig. 40.—The nerves and arteries of the nasal septum and of the tongue. \* = Divided posterior pharyngeal wall. \*\* = Sphenoidal sinus. (Sobotta and McMurrich.)

great palatine artery, emerges at the greater or posterior palatine foramen, passes forward into the roof of the mouth, and some of its branches pass into the nasal fossa through the incisive foramen and anastomose with the nasopalatine artery.

The *pharyngeal (pterygopalatine) artery*, a slender branch of the internal maxillary, accompanied by the pharyngeal branch of the sphenopalatine ganglion, courses downward and through the pharyngeal canal to the roof of the pharynx. The artery gives small branches to the dorsal and upper part of the nasal fossa, the sphenoidal sinus, the auditory tube, and the pharynx.

The *posterior and anterior superior alveolar arteries* and the *infra-orbital*

*artery*, all branches of the internal maxillary, give additional supply to the maxillary sinus and to the external nose.

The *external maxillary* or facial artery supplies lateral nasal branches to the nostril and the wing of the nose and a branch from the superior labial to the nasal septum. The *angular artery* of the external maxillary and the *dorsal nasal artery* of the ophthalmic form an anastomotic network for the supply of the dorsum and side of the external nose.

The **venous blood** from the plexiform and cavernous network of the nose is returned by three main pathways: dorsally, into the *sphenopalatine vein*; ventrally, into the *anterior facial vein*; cranially, into the *ethmoidal veins*. The latter communicate with the ophthalmic vein, the veins of the dura, and the superior sagittal sinus. Frequently an anterior ethmoidal vein passes through the cribriform plate and connects up with the venous plexus of the olfactory bulb and the frontal lobe of the brain.

#### THE LYMPHATIC SUPPLY

The **lymphatics** of the nasal cavity and the paranasal sinuses ramify the connective tissue of the tunica propria of the mucoperiosteum in the form of a dense capillary network. At places the mucosa is infiltrated with lymphocytes. Numerous minute solitary nodules are encountered. The main collecting vessels of the capillary network form ventral and dorsal groups.

The *ventral group of the lymphatics* courses in the groove between the triangular cartilage and the related bones, and between and outside the several external cartilages. They unite in the subcutaneous tissue and form large ultimate distributing trunks which end in the facial and submaxillary (submandibular) groups of lymph-nodes. There is a constant and considerable anastomosis between the anterior group of nasal lymphatics and those of the skin over the external nose.

The *dorsal group of the lymphatics* drains in the direction of the region ventral and caudal to the pharyngeal ostium of the auditive (eustachian) tube. From here go forth the large collecting trunks to terminate in the deep cervical lymph-nodes and the aberrantly placed retropharyngeal group of nodes.

The major lymph drainage from the paranasal sinuses appears to be into the retropharyngeal nodes.

The drainage from the external nose is into the parotid, facial, and submaxillary nodes. The external nasal lymphatics may also drain into the lymphatics of the nasal mucosa.

The subdural space communicates indirectly with the extracranial lymphatics and directly with the perineural spaces of the olfactory nerve fila. Key and Retzius succeeded also in injecting the olfactory perineural sheaths by way of the subarachnoidal space of the brain. The true lymphatic capillaries appear not to communicate directly with the olfactory perineural sheaths. However, there appears to be absorption and interchange of fluid and cellular elements from one to the other. The relationship probably is like that of the blindly beginning lymph capillaries and the tissue spaces elsewhere in the body.

There are those who believe that lung abscess and bronchiectasis result from nasal and paranasal sinus disease by extension by way of lymphatic pathways. The lymph returning from the head into the deep cervical regional

nodes and from there into the large veins at the base of the neck would, of course, establish continuity of pathway and make possible the carriage of infectious matter from the nose and sinuses into the right side of the heart and from there to the lungs. In the lungs it would, in all probability, in a large measure be held in the capillary bed, making a pathological condition of the lung possible secondary to nasal disease.

Interesting in this connection is the observation of Winternitz that, in addition to draining into the tracheal and bronchial lymph-nodes, the lymphatics of the tracheal submucosa establish a direct pathway of infection to the lung by anastomosing with periarterial and peribronchial lymphatics at the bifurcation of the trachea. If a similar condition can be established for the lymphatics of the nose and accessory sinuses, *e. g.*, direct lymphatic continuity from these chambers into the lung, we will have anatomic support for the clinical observation that lung abscess and bronchiectasis result from nasal and paranasal sinus disease wholly by way of lymphatics. However, at present these pathways are not definitely established anatomically and further study is necessary along these lines. One must always bear in mind that infectious materials from the upper respiratory passages may be aspirated; also, that the veins from the nose return blood to the right heart, and from there the pulmonary artery conveys it to the capillary bed of the lungs.

In a paper on non-tuberculous diseases of the apex of the lung Pal makes mention of several cases of catarrh of the apex in persons with homolateral affections of the nasal sinuses. Râles disappeared and reappeared together with the nasal affection. The anatomic pathway is not clear. Infection by aspiration may apply here. One also thinks of the lymphatics and the blood-stream. However, the homolateral involvement is disconcerting. Is it possible that the parietal and visceral pleuræ, because of a pathology, become fused at the apex, and new lymphatic capillaries sprout and connect up the deep neck lymphatics with the pleural and the pulmonary lymphatic channels?

#### THE NERVE SUPPLY

The nerves of common sensation to the nose and the ancillary structures are derived from the ophthalmic and the maxillary divisions of the trigeminal nerve, that is, from the *ophthalmic* and the *maxillary nerves* respectively. The *common sensory nerves* to the nose and related parts are the peripheral processes of T-fibers which spring from nerve-cell bodies located in the semilunar (gasserian) ganglion (Fig. 41).

The *posterior superior* and *posterior inferior nasal nerves*, the *nasopalatine nerve*, and the *anterior palatine nerve* are derived from the maxillary nerve. They come to the internal nose by way of the *sphenopalatine nerves* and the *sphenopalatine ganglion* of Meckel and its branches. The *anterior ethmoidal nerve* from the nasociliary branch of the ophthalmic supplies the *medial and lateral anterior nasal nerves* to the forward portion of the internal nose. The *posterior, middle, and anterior superior alveolar* (dental) *nerves*, branches of the maxillary, supply additional fibers to the maxillary sinus and the external nose. The terminal part of the maxillary nerve, that is, the *infra-orbital*, also sends fibers to the external nose.

Nerve impulses which determine the caliber of blood-vessels and control the mechanism of secretion of the glands in and about the nasal fossæ are transmitted by *sympathetic efferent fibers*. Both the *cranial* and the *thorac-*

*columbar* sympathetics have to do with the nasal and paranasal supply. The cranial sympathetics are contained in the *great superficial petrosal nerve*, a branch of the pars intermedia (glossopalatine nerve) of the facial, and the thoracolumbar sympathetics are supplied by connections between the upper thoracic segments of the spinal cord and the superior cervical sympathetic ganglion and extended headward by way of the *great deep petrosal nerve*. Both the great superficial petrosal and the great deep petrosal nerves form the *Vidian nerve*, and by it are connected with the *sphenopalatine ganglion*. The cranial sympathetics to the nose are generally vasodilator and secretomotor in function; an antagonistic action coming from the thoracolumbar sympathetics. Moreover, it is believed by some workers that certain impulses from the nasal mucous membrane reach the cerebrospinal centers by way of *visceral afferent nerves*.

It is a well-established fact that after removal of the semilunar ganglion (gasserian ganglion) pressure-pain may persist in the face and tongue. Maloney and Kennedy (1911) believe "that the head sympathetic subserves a general crude sensibility to pressure-pain of the protopathic type with high threshold which may persist after the removal of the semilunar ganglion." Deep pain of the face is served by fibers of the facial nerve whose cell bodies are located in the geniculate ganglion (Loyal E. Davis, 1923).

The **sphenopalatine ganglion** (ganglion sphenopalatinum), also known as Meckel's, the nasal or sphenomaxillary ganglion, is a small, triangular, reddish-gray (in fresh state) body, and is a component of the group of sympathetic ganglia found in the head region of the body. It is located in the sphenopalatine fossa and very close to the sphenopalatine foramen and is suspended from the maxillary division of the trigeminal nerve. The sphenopalatine ganglion is, therefore, more or less intimately related topographically to the lateral wall of the nasal fossa, the sphenoidal sinus, and certain of the posterior ethmoid cells. In its histologic make-up it consists of an interlacement of nerve-fibers and stellate nerve-cell bodies. The nerve-fibers forming the ganglionic branches leading from the ganglion (post-ganglionic neurons) participate in supplying the nasal fossa and the related parts. Moreover, many of the branches of the maxillary division of the trigeminal nerve to the nose pass by way of the sphenopalatine ganglion and its branches. However, most of these fibers pass outside the ganglion, others traverse it. A few terminate in it for the supply of the capsule. This relationship of the sensory trigeminal nerve to the sympathetic sphenopalatine ganglion must not be forgotten in clinical work. Especially is this so when it is believed that the ganglion of Meckel is at fault in certain head and face pains. Significant also is the fact that certain fibers of the facial nerve mediate deep pain of the face and that the sympathetic nerves of the head apparently subserve a general crude sensibility to pressure-pain.

The **olfactory nerve** (*nervus olfactorius*) is the peripheral organ of smell. Approximately 20 non-medullated olfactory nerve filaments issue from the olfactory mucosa, which is limited to the cephalic third of the nasal septum, nearly the whole of the superior concha, a small portion of the middle concha, and to supernumerary conchæ that may be differentiated above the supreme concha. These filaments (the olfactory nerves, collectively the olfactory nerve) pass through the foramina of the cribriform lamina in two rows, medial and lateral, become ensheathed by the meningeal layers, enter the

primary olfactory center in the olfactory bulb, and synapse with the dendrites of the mitral cells in formations known as the olfactory glomeruli. The several portions of the secondary olfactory center are olfactory reflex stations. The cortical area for smell is located in the uncus and the hippocampus.

The **terminal nerve** (nervus terminalis) is a slender and variably plexiform nerve, containing both medullated and non-medullated fibers. It is ganglionated. The terminal nerve is found in many classes of vertebrates from fishes to man. Its peripheral twigs are found distributed to the mucous membrane of the nasal septum and to the mucosa joining the olfactory region proper on the lateral wall. The exact ending of its peripheral twigs

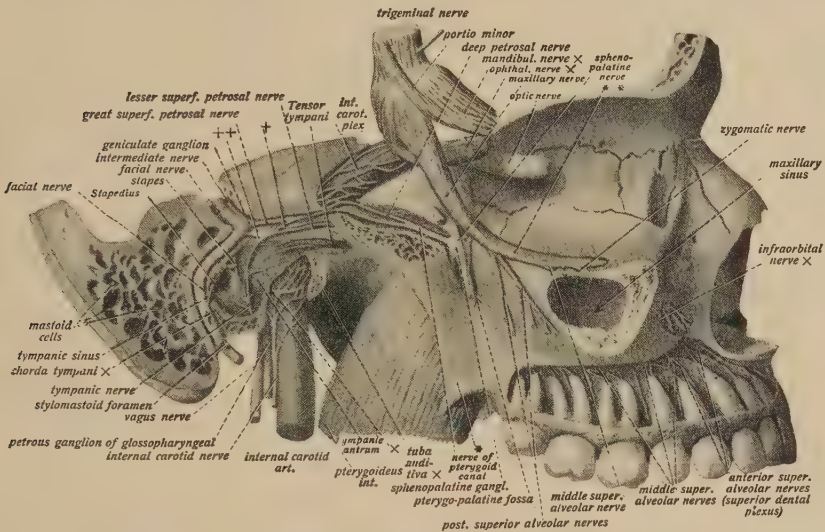


Fig. 41.—The second branch of the trigeminal nerve, the sphenopalatine ganglion, the intracranial portion of the facial nerve, and the tympanic nerve.

The orbit has been opened by a sagittal section through its outer portion, its contents removed, the pterygoid canal opened, and the temporal bone sawn obliquely, exposing the tympanum and the facial canal. \* = External pterygoid plate. \*\* = Infra-orbital nerve with the overlying zygomatic nerve. + = Caroticotympanic nerve. \*+ = Anastomosis between great superficial petrosal and tympanic nerves. ++ = Anastomosis between facial and tympanic nerves. (From Sobotta and McMurich.)

is unknown. Centrally the nerve passes through the cribriform plate. The function of the terminal nerve is obscure.

The **vomeranase nerve** (nervus vomeronasalis) is composed of central processes of cell bodies located in the mucosa of the vomeronasal organ of Jacobson. Since the latter is rudimentary and probably functionless as an olfactory organ in adult man, the vomeronasal nerve is absent. In adult man the vomeronasal region seems to be for general sensation only and is, therefore, supplied by the trigeminal and the sympathetic nerves common to the mucosa of the general nasal fossa. However, Read has shown that in man a branch of the olfactory nerve passes to the vomeronasal organ of Jacobson, at least at the time of birth. Indeed, some would consider the vomeronasal nerve as a special slip of the olfactory nerve.

J. PARSONS SCHAEFFER.

## INFLAMMATORY DISEASES OF THE NOSE, ACUTE AND CHRONIC

## GENERAL PRINCIPLES OF LOCAL TREATMENT AND TECHNIC

**Treatment.**—The mucous membrane of the nose is a very delicate structure and should be treated so skilfully that no damage is done to it by the treatment. If the applications are made with large pledgets of cotton, which are forced through between inflamed tissues, the membrane may be excoriated or bruised, and the consequent inflammatory reaction adds to the discomfort of the patient and retards the natural recovery.

In making applications in the nose choose an applicator with a pledget of cotton which is small enough to slip through easily between the septum and the tissues on the lateral nasal wall. Be sure that the end of the applicator is well protected and that the cotton is fluffy, not wadded.

Make the application skilfully and gently and thus avoid adding injury to an already inflamed membrane. Practically all of the necessary nasal applications can be made without pain. If the patient complains of being hurt, the complaint is a just criticism of lack of skill in making the application.

In removing the cotton from the applicator small squares of paper folded several times protect the fingers from contamination better than cotton or cloth. As most of the pathologic lesions in the nose are infectious, the rhinologist must be constantly on guard to avoid contaminating his fingers and the instruments.

**Technic.**—The mucous membrane in the nose and throat has established for itself a considerable resistance to bacterial infection, due to its constant exposure to the numerous bacteria in the air, but this is no excuse for otolaryngologists failing to use reasonable precaution and at least as good technic as any other surgeon.

In most of the operations in the nose it is impossible to make the operative field sterile, but we can avoid introducing new infections in both operative and office work, and it is our duty to do so. All instruments should be sterilized every time they are used, whether for simple treatment or for operative work. A mirror clip of some sort, which can be easily applied, removed, and sterilized, should be used constantly in the office and in the operating room.

In all operative work the general preparation and surgical technic which every good surgeon advocates should be used. If it is necessary to touch the head mirror, or anything else which is contaminated, protect the gloves or hands with a large sterile sponge. The same care in preventing the introduction of new infections should be observed in the dressings and after-treatment of operative wounds.

## INFECTIONS IN THE NASAL VESTIBULE

**Definition.**—A localized infection in the vestibule, almost invariably beginning in a hair follicle.

**Synonyms.**—Folliculitis, furuncle, boil, pimple.

**Etiology.**—The *Staphylococcus pyogenes aureus* is the most common organism found, but there may be a mixed infection. The *Streptococcus*, fortunately, is rarely the cause of these infections.

The pernicious habit of picking the nose, pulling out the hairs, or prick-

ing an indurated area with an unclean instrument or needle, and then squeezing it with fingers which are contaminated, open up channels through which infections enter. Debilitating constitutional diseases are predisposing causes.

**Prophylaxis.**—The skin and the hairs in the nasal vestibule are constantly exposed to all of the organisms and dust in the inspired air. Careful removal of this debris with a clean, moist handkerchief, or spraying the vestibule gently with a normal saline, or some other mild alkaline solution, and then using the handkerchief, removes the threatening organisms and practically eliminates the danger of infections in the vestibules.

**Pathology.**—The pathology is the same as that of an infection anywhere in the skin. After the infective organism penetrates the skin or hair follicle its growth causes inflammation, induration, and pus formation.

**Symptomatology.**—The first symptoms are redness, swelling, slight discomfort, and pain which steadily increases. The pain is aggravated by the density of the tissue in the vestibule.

The tip of the nose becomes inflamed and swollen and the swelling may extend well up over the bridge of the nose, even involving the eyelids. There is usually some rise in temperature. After a few days a typical furuncle with a yellow center, surrounded by induration, shows in the vestibule.

**Diagnosis.**—The diagnosis is easily made from the typical symptoms and the appearance of the infected follicle.

**Prognosis.**—The prognosis is usually good, but should always be guarded on account of the serious danger of any infection in the skin of the nose and upper lip extending through the veins to the orbit and cavernous sinus.

**Treatment.**—The skin around the point of infection should be carefully cleaned with little pledgets of cotton dipped in hydrogen peroxide or some other non-irritating solution, then dried, and painted with alcohol. The application of alcohol should be repeated every three hours. In a slight, circumscribed infection this may be all that is it necessary to do.

The patient should be told that these slight infections in the skin of the nose are sometimes extremely dangerous and cautioned against picking or irritating it in any way.

If the inflammation and swelling increase, and show externally, then hot, moist, boric acid dressings should be applied over the nose to relieve the pain and keep the surrounding skin clean. No attempt to open the little abscess should be made until it points and the pus can be seen under the skin. Then, with strict aseptic precautions, a small incision should be made within the infected area so as to avoid opening new lines of infection. No pressure or squeezing to expel the pus should be applied, but drainage should be encouraged by the use of the hot boric dressings, and careful washing of the vestibule with small pledgets of cotton dipped in a 10 per cent. boric acid solution. The application of the yellow oxide of mercury ointment over the incision protects the inflamed area and prevents crust formation.

Occasionally an infection, beginning in the vestibule, may burrow along the alar cartilage and point externally. In such cases there is some danger of necrosis of the cartilage and deformity of the ala. Two or more small incisions in the skin may give better drainage, with less scar formation, than one large incision.

Care should be used, even in washing the face, not to disturb the

inflamed area. The patient should be kept quiet in bed until the temperature has remained normal for at least two days.

If infections in the vestibule recur, either an autogenous vaccine, or the stock furunculosis vaccine, which contains the *Staphylococcus aureus* only, should be given. The initial dose of the vaccine is 0.1 c.c., and the injections should be made at intervals of three or five days, increasing each successive dose 0.1 c.c. until eight injections have been given.

**Complications.**—The veins of the upper lip and the nasal vestibule and the external nose all communicate with the superior ophthalmic veins and the cavernous sinus, and any infection in these regions may cause a thrombosis, which is liable to extend to the cavernous sinus (Fig. 42).

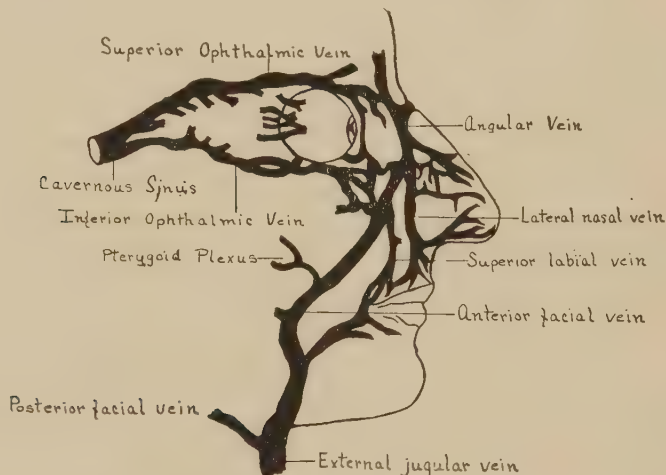


Fig. 42.—Drawing (slightly diagrammatic, from Author's specimens) showing the anastomoses of the superficial veins of the nose and lips with the ophthalmic veins and the external jugular vein.

The angular vein leads directly into the superior ophthalmic vein, which runs backward in the upper portion of the orbit, through the superior orbital fissure, and terminates in the anterior portion of the cavernous sinus.

The lateral nasal vein communicates with the superior ophthalmic vein and the anterior facial vein.

The majority of the veins of the nasal fossæ, both from the septum and the lateral walls, unite and form the sphenopalatine vein, which terminates in the pterygoid plexus, and this plexus of veins also communicates directly with the cavernous sinus, and with the anterior facial vein.

These veins also communicate with the external jugular vein through the anterior facial vein, and through the jugular vein metastasis in the lungs and heart may develop.

Any infection in the nasal vestibule, or in the skin around the nose, which is accompanied by redness and swelling extending up over the bridge of the nose and involving the eyelids should be regarded with apprehension.

The history of a chill, with headache, septic temperature, swelling of the eyelids, and an exophthalmus, which begins on one side and in one or two days involves the other eye also, are pathognomonic symptoms of cavernous sinus thrombosis.

Ligation of the anterior facial vein and also the angular vein, just above the internal canthus of the eye, is recommended by some authors.

Good drainage with as little trauma as possible, no manipulation or

squeezing, hot boric dressings, and absolute rest are probably the best methods of treating such complications.

### ACUTE RHINITIS

**Definition.**—An acute inflammation of the mucous membrane of the nose. Secondarily, the mucous membrane lining the accessory nasal cavities is always somewhat involved.

**Synonyms.**—Acute coryza. A cold.

**Etiology.**—No specific germ has been proved to be the etiologic factor, but the typical symptoms which an acute rhinitis produces are as clear cut and distinct as the symptoms in many of the acute infectious diseases in which the specific organism has been isolated and identified.

Clinical and epidemiologic experience both indicate that it is an acute infectious disease.

The experimental studies of the incitant of acute rhinitis, made by Olitsky and McCartney,<sup>1</sup> show quite definitely that acute rhinitis may be transmitted from man to man and that the rhinitis produced experimentally may be transmitted to a second individual.

Probably in the near future some specific organism will be discovered and proved to be the causative agent of acute rhinitis, and we shall then be able to produce an immunity to this disease with a vaccine, as successfully as we do now in typhoid fever.

The micro-organisms which are probably factors in the cause of acute rhinitis are the *Micrococcus catarrhalis*, the influenza bacillus, the *Bacillus mucosus capsulatus*, the *Staphylococcus albus* and *aureus*.

The bacteriologic examination of secretion from the nose, during an attack of acute rhinitis, usually shows the predominance of one organism, most frequently the *Staphylococcus albus*.

All of these germs are more or less constantly present in the air and may usually be found in the nasal vestibule, but if the nasal mucous membrane is healthy, and the general physical condition is good, the normal resistance of the mucous membrane combats and usually overcomes the infective organisms. If the normal resistance has been decreased or abolished by any cause, then the infecting organism survives and produces a definite train of symptoms.

Frequent attacks of acute rhinitis in children are usually caused by hypertrophied and pathologic adenoid tissue and tonsils, which harbor infections and cause constant irritation in the nasal mucous membrane and thus make the membrane more susceptible to infection.

Any pathologic condition in the nose, as well as fatigue, dissipation, overindulgence in food or alcohol, insanitary or improperly ventilated rooms, lack of proper exercise, chemical irritants, and prolonged exposure to dust, all tend to lower the normal resistance and are predisposing factors. Chilling of the whole body or some part of it, as the feet or the head and neck, puts an extra strain upon the heat-regulating mechanism and evidently decreases the bodily resistance and increases the susceptibility to infections, especially in the mucous membrane of the respiratory tract. This is particularly so in people of sedentary habits, who live and work in overheated rooms.

Exposure to cold alone cannot be the only factor in acute rhinitis, for arctic explorers are practically immune while they are in the uncontaminated

polar regions, but usually develop a coryza if they return to the contaminated atmosphere of our cities during the winter season when acute nasal infections are prevalent.

In other words, anything which decreases the normal resistance of the body and the nasal mucous membrane makes it easier for an infection to invade the membrane, just as an abrasion in the skin predisposes to an infection there.

The most important factors in the etiology of acute rhinitis are the general resistance of the whole body, the amount of resistance of the nasal mucous membrane to infection, and the virulence of the invading micro-organism.

**Prophylaxis.**—*General Principles.*—As pathologic conditions in the nose, and in the tonsils and adenoid tissue, are always predisposing causes of acute rhinitis, such conditions should be rectified if possible. The differential diagnosis between a pathologic condition and an anomaly, such as a small septal spur causing no symptoms, must always be carefully made before any operative treatment is recommended.

The cold shower or plunge produces a very stimulating reaction in many people, and such a reaction helps to maintain the general bodily resistance. In some people the reaction is slow, or entirely lacking, and such people should take a moderately warm shower followed by a brisk rub.

The proper amount, and kind, of clothing is an individual question and should be intelligently decided for each person. Too much clothing is as bad as too little.

Proper attention to ventilation, diet, elimination, recreation, and exercise are all essential factors in decreasing the susceptibility to disease.

**Vaccine.**—During the past few years the value of the catarrhal vaccine, as a prophylactic measure, has been reasonably well established. The vaccine is made from the following organisms, *Micrococcus catarrhalis*, *Bacillus mucosus capsulatus*, *pneumococcus*, *influenza bacillus*, *streptococcus*, *Staphylococcus albus*, and *S. aureus*. It is distinctly a prophylactic measure and should not be used during an attack of acute rhinitis.

The object of the vaccine treatment is to produce an immunity or at least increased resistance to infections. As attacks of acute rhinitis occur most frequently in the fall and winter the first injection of the vaccine should be given in September.

Four subcutaneous injections are given at intervals of one week, and the injections are usually made under the loose skin, on the back of the arm, about midway between the elbow and the shoulder, alternating from one arm to the other each week.

The first dose for adults is 5 minims (0.308 c.c.); the second dose,  $7\frac{1}{2}$  minims (0.461 c.c.); the third dose, 10 minims (0.616 c.c.); the fourth dose,  $12\frac{1}{2}$  minims (0.769 c.c.). The immunity obtained by these four injections lasts about one month, and this immunity can be maintained by the subsequent injection of  $12\frac{1}{2}$  minims every four weeks during the winter and spring.

If marked local or constitutional reaction follows any dose, the subsequent dose should not be increased. The dosage should be decreased for children and for those known to react unfavorably to foreign proteids.

**Pathology.**—The capillaries of the nasal mucous membrane become dilated and the serous glands are stimulated, causing a serous or watery dis-

charge. This is soon followed by engorgement of all of the blood-vessels, especially of the inferior turbinal and the anterior end of the middle turbinal, and an infiltration of lymphocytes into the tissue, producing variable swelling and nasal obstruction. The increased blood-supply stimulates the mucous glands and the secretion then becomes thick and mucopurulent. Then there is a gradual return to normal in one to three weeks.

**Symptomatology.**—The first symptoms are a slight feeling of irritation or fullness in the nose, frequently accompanied by sneezing, and soon followed by a serous discharge which increases rather rapidly. This serous discharge may be irritating, and with the frequent use of the handkerchief it often produces excoriations around the nasal vestibule and on the upper lip.

Nasal obstruction is always present in the early stage of an acute rhinitis and in some patients this is one of the most annoying symptoms. Breathing through the mouth naturally follows the nasal obstruction, and causes dryness and irritation of the pharynx and larynx. On account of the nasal obstruction the sense of smell is less acute and, consequently, there is also some interference with the sense of taste. The voice loses its normal nasal resonance and has what is commonly called a nasal twang.

In nursing children the nasal obstruction makes it difficult for them to get the proper amount of nourishment. In adults there is usually little or no increase in the temperature, but in children some increase in temperature is almost always present.

**Complications.**—The membrane of the nasal accessory cavities is always somewhat involved in an acute rhinitis, but usually the inflammation is so slight that it causes no symptoms. In the more virulent infections, especially influenza, the accessory cavities are acutely inflamed and sensitive, frequently causing headache and pain. If the ostia are obstructed, the pain becomes severe and increases until drainage is reestablished.

Inflammation and swelling in the eustachian tubes produces a feeling of fulness in the ears and variable deafness. The infection may extend through the tubes, or it may be forced into the middle ear by violently blowing the nose and thus cause an acute otitis media. Secondary infections in the larynx, trachea, and bronchi are rather common complications.

The eyes are frequently slightly inflamed and occasionally a severe inflammation may extend to them through the nasal ducts.

**Diagnosis.**—The diagnosis is easily made from the nasal obstruction and increased discharge, and from an examination which will show the nasal fossæ more or less completely occluded by the inflamed and swollen turbinal tissue. The pharynx and nasopharynx will also show signs of inflammation.

In children and young adults an acute rhinitis, especially if it is accompanied by constitutional symptoms, should always suggest the possibility of some general systemic infection, such as the exanthemas.

**Prognosis.**—The prognosis is invariably good in uncomplicated cases. In young children and elderly people the danger of bronchitis and pneumonia will influence the prognosis.

**Treatment.**—In the beginning of an attack of acute rhinitis, when practically the only symptoms are a little discomfort and irritation in the nose, the prompt use of a camphor and menthol solution, or ointment, may often stop or relieve the symptoms very markedly.

For this purpose the following prescriptions will serve as samples:

			Gm. or c.c.
R.	Camphor.....	} āā gr. ij.....	0.15
	Menthol.....		
	Liquid petrolatum.....	ad. ʒj.....	30.00
M. S.—A few drops in each nostril as directed.			

The easiest way to use this solution in children is with a medicine dropper, putting a few drops in each nostril every three or four hours. Adults usually prefer to use the medicine in an atomizer.

The same ingredients may be made into an ointment and dispensed in a collapsible tube with a tapering nasal tip.

			Gm.
R.	Camphor.....	} āā gr. ij.....	0.15
	Menthol.....		
	Petrolatum.....		ad. ʒj.....
M. S.—Apply a small quantity in each nostril as directed.			

The patient should be told to squeeze a little of the ointment from the tube into each nostril and then distribute the ointment over the membrane by a few quick inspirations.

This camphor and menthol solution, or ointment, usually is more efficacious in the early stages of an acute rhinitis than the various preparations of silver. If, for any reason, a silver solution is preferred, a 5 to 10 per cent. solution of neosilvol (colloidal silver iodide compound) used in a dropper, or an atomizer, every four hours may be prescribed. The neosilvol solution does not stain, is not irritating, and does not deteriorate as rapidly as some of the other colloidal silver solutions. A fresh solution should be prepared at least once a week.

When the nose is obstructed by the swelling of the turbinal tissue the application of adrenaline will give marked relief for several hours. The stock solution (1 : 1000) should be diluted with 9 parts of normal saline solution and used in an atomizer. The camphor and menthol application should always be made after using the adrenaline. The oily preparation acts as a protection to the mucous membrane and prolongs the effect of the adrenaline. The adrenaline spray will deteriorate in seven to ten days and will then not only lose its specific action, but may become an irritant. Some patients may have an idiosyncrasy for adrenaline, and its use may cause symptoms quite like an attack of vasomotor rhinitis. In such patients adrenaline should not be used.

Bismuth subnitrate powder dusted on the nasal mucous membrane with a powder blower, either after the use of adrenaline or without its use, if it is not well tolerated, is one of the most soothing applications that can be made in an acutely inflamed nose. This powder sticks to the membrane for hours, and not only protects the membrane but also decreases the amount of secretion.

The adrenaline may be combined with chloretone in an ointment. The chloretone acts as a mild anesthetic and tends to decrease any reflex symptoms from the adrenaline.

			Gm.
R.	Adrenaline.....	gr. $\frac{1}{2}$ .....	0.03
	Chloretone.....	gr. x.....	0.65
	Petrolatum.....	ʒj.....	30.00
M. S.—Put in collapsible tubes and label: To be used as directed.			

Often patients who cannot use the adrenaline spray may use a 1 per cent. ephedrine hydrochloride spray. In all cases in which adrenaline is indicated ephedrine hydrochloride may be used.

The adrenaline may also be combined with the neosilvol solution if there is no idiosyncrasy to contraindicate its use.

	C. c.
R. Adrenaline.....	3.70
Neosilvol sol. 10 per cent. ad.....	30.00
M. S.—Use in a dropper or atomizer every three or four hours.	

Cocaine should never be used as a local application in acute rhinitis. The effect of its application is not as beneficial as adrenaline and the danger in using it is infinitely greater.

Any of the drugs which tend to decrease the amount of secretion from the mucous membrane are of some benefit in the early stages of an acute rhinitis, and may be used during the first few days of the attack, but not after the secretion becomes thick and mucopurulent.

The extract of belladonna,  $\frac{1}{8}$  grain (0.008 gm.), or atropine,  $\frac{1}{120}$  grain (0.00054 gm.), or rhinitis tablets (half strength) consisting of: Camphor, gr.  $\frac{1}{4}$  (0.016 gm.), quinine, gr.  $\frac{1}{4}$  (0.016 gm.), and fluidextract of belladonna, min.  $\frac{1}{8}$  (0.007 c.c.), may be given every two or three hours until the drying effect is felt in the nose and throat, and then continued, three times a day, for two or three days.

Discomfort or pain over the frontal or maxillary sinuses indicates inflammation of the membrane lining the sinuses and is usually relieved by hot or cold applications. In such cases the patient should remain in the house until these symptoms disappear, using one of the prescriptions containing adrenaline as often as is necessary to maintain good drainage.

## REFERENCE

### *Acute Rhinitis*

1. Studies of the Nasopharyngeal Secretions from Patients with Common Colds. P. K. Olitsky and J. E. McCartney: Jour. Exper. Med., xxxviii, 1923, p. 427.

## RHINITIS OF THE EXANTHEMAS

One of the early symptoms of the exanthemas is an acute inflammation of the mucous membrane in the nose and throat. It is often difficult, or even impossible, to make a differential diagnosis between such an inflammation and the inflammation of an acute rhinitis during the first days of the infection.

Generally in the exanthemas the inflammation in the nose is a little more severe, the rise in temperature is greater, the conjunctiva is more inflamed, headache is more frequent, and general malaise is more constant than in a simple rhinitis.

All of these symptoms precede the appearance of the rash in measles and scarlet fever, and when they are present in children and young adults they should always suggest the possibility of the onset of some systemic infection.

**Measles.**—It is generally admitted that the infective agent in measles is present in the discharge from the nose and eyes, and that perhaps the most infectious period is during the early stage, before the typical symptoms become manifest.

*Symptomatology.*—Koplik's spots are minute bluish-white spots, surrounded by a bright red areola, showing in the buccal membrane opposite the molar teeth. They do not appear on the palate or pharynx and may be easily overlooked. Good, bright daylight is the best light for demonstrating them.

They are one of the earliest positive symptoms of measles and they may show when there is only a very slight coryza, before there is any rise in temperature.

As the disease progresses the inflammation in the eyes and nose increases. The nasal discharge becomes purulent and quite profuse, and the paranasal sinuses are often infected. The mucous membrane in the mouth and pharynx is congested and the palate presents a mottled appearance.

**Scarlatina.**—The rhinitis in scarlet fever is usually less severe than it is in measles.

*Symptomatology.*—The vivid red erythema of the mucous membrane which shows during the first few days is confined to the oropharynx, and the patient complains more of some soreness in the throat than of any irritation in the nose. Another early manifestation of scarlatina is well-marked punctations on the soft palate and at this time there is usually some irritation in the nose and a serous discharge. Later the nasal mucous membrane becomes more inflamed and there is a persistent, irritating, purulent discharge. In a great majority of the cases the accessory sinuses are also involved, but unless there is some obstruction of the ostia the symptoms of sinusitis are masked by the general depression of the patient. The inflammation in the nose and sinuses gradually subsides as the general condition improves.

The infection in the nose and sinuses in severe cases of scarlet fever involves all of the layers of the mucous membrane, including the periosteum, and may produce necrosis and osteomyelitis.

**Treatment.**—As the pathologic conditions in the nose are practically the same in measles and scarlet fever, the same methods of treatment are applicable in both diseases.

The adrenaline and neosilvol solution will relieve the nasal obstruction, promote drainage, avoid the retention of secretion in the nose and nasopharynx, and thus decrease the danger of complications.

	C. c.
R. Adrenaline.....	3.70
Neosilvol sol. 10 per cent. ad.....	30.00
M. S.—Use in an atomizer or with a medicine dropper every three or four hours.	

When the nasal obstruction and acute inflammation have subsided, the adrenaline may be omitted, but the 10 per cent. neosilvol solution, or the solution of camphor and menthol,  $\frac{1}{2}$  per cent., in liquid albolene, should be used three or four times a day, until all the symptoms of inflammation in the nose and nasopharynx have disappeared.

The careful use of a nasal douche, containing a warm normal saline solution, is recommended by some rhinologists, but the danger of infecting the ears more than counterbalances the benefit derived from the douche.

If the nasal discharge persists after all of the other symptoms have disappeared, it is probably due to an infection in the nasal accessory sinuses, or pathologic conditions in the tonsil and adenoid tissue, or both.

The removal of the tonsil and adenoid tissue usually cures the sinus

infection also. When such foci of infection have been removed, proper attention to diet and hygiene and, if necessary, a few months spent in a warm, dry climate will cure practically all cases of uncomplicated sinusitis following measles and scarlatina in children.

**Complications.**—In measles and scarlatina, infections in the ears and tracheitis, bronchitis, and pneumonia are fairly frequent complications.

Patients who have had either disease are particularly susceptible to diphtheria on account of the lowered resistance of the mucous membrane in the nose and throat. For this reason an attack of laryngitis in such patients should always be considered carefully, with the idea of either establishing the diagnosis of diphtheria or excluding it. Any indications of infection in the ears should be watched closely, and the proper treatment instituted promptly.

**Influenza.**—An attack of influenza usually begins with an acute rhinitis. The nasal symptoms are practically the same as those of an ordinary acute rhinitis, but the constitutional manifestations are much more evident. The rise of temperature, general depression, and exhaustion easily differentiate influenza from a simple rhinitis. The nasal accessory sinuses are almost always infected and are the cause of the headache and pain in the frontal and maxillary regions.

**Treatment.**—The treatment of the nasal inflammation should be the same as in an acute rhinitis. The adrenaline and chloretone ointment or the adrenaline-neosilvol solution should be used as often as is necessary to maintain nasal respiration and good drainage. The patient must be cautioned particularly against blowing the nose vigorously, on account of the danger of infecting the ears.

Hot applications over the sinuses help to relieve the pain there, especially if they are supplemented with phenacetin or some similar preparation. Absolute rest in bed until the temperature becomes normal is one of the most essential things in the treatment of influenza.

#### HYPERTROPHIC RHINITIS

**Definition.**—A chronic inflammatory thickening of the nasal mucous membrane with an increase in the connective tissue, especially on the inferior turbinal.

**Synonyms.**—Chronic rhinitis; chronic catarrh. (The word catarrh is objectionable because it is used by the laity, and even by the medical profession, in a very inaccurate way to apply to many pathologic lesions. It is used here, however, and also in atrophic rhinitis on account of the almost universal employment of the word in these diseases.)

**Etiology.**—No single, specific cause for hypertrophic rhinitis can be demonstrated. Any condition which produces repeated attacks of irritation and inflammation in the nasal mucous membrane will cause hypertrophy of the membrane and the underlying tissue. Pathologic conditions in the tonsils and especially in the adenoid tissue, if neglected, are predisposing causes. Repeated attacks of acute rhinitis, and occupations in which dust or chemical irritants are constantly inspired, will produce a chronic inflammation and hypertrophy.

Chronic sinusitis will cause hypertrophy, but proper attention to the sinus infection will eliminate this condition.

Septal spurs and deviations are frequently accompanied by hypertrophy

of the turbinal tissue in the wider naris. This is a compensatory hypertrophy and often is not pathologic. Spurs and deviations which press against the structures on the lateral nasal wall, or interfere with respiration and drainage, are etiologic factors. Climatic conditions, improper diet, lack of exercise, unhygienic living, and constitutional diseases, such as rheumatism, gout, diabetes, and anemia, are all influential factors in hypertrophic rhinitis.

The disease is more common in men than in women and usually occurs between the ages of fifteen and forty-five years.

**Prophylaxis.**—One of the most important prophylactic measures is the elimination of other pathologic conditions in the nose and nasopharynx. In this connection special emphasis is placed upon the words *pathologic conditions*. By this is meant conditions which are actually pathologic and cause demonstrable symptoms. Anomalies in the septum and nasal structures, which cause no symptoms, are not pathologic and do not need treatment of any kind.

The treatment of constitutional diseases and proper attention to diet, hygiene, and exercise decrease the tendency to the development of hypertrophic rhinitis.

**Pathology.**—The hypertrophy begins with a dilatation of the blood-vessels. This increased blood-supply causes infiltration in the connective tissue, with thickening of the walls of the blood-vessels. The interstitial tissue also gradually thickens and increases in density, so that the contractile power of the turbinal tissue is interfered with or lost.

The hypertrophy usually involves most of the tissue covering the inferior turbinal bone, but it may be limited to the anterior or posterior ends of the inferior turbinal. Occasionally the anterior end of the middle turbinal is also hypertrophied and, less frequently, the tissue on the septum, opposite the anterior end of the middle turbinal, may be thickened.

**Symptomatology.**—Nasal obstruction is the most common symptom. It varies in degree, depending upon the amount of hypertrophy and climatic conditions, being worse in damp weather, and gradually increases until it becomes constant in cases of marked hypertrophy. It is usually worse at night and more noticeable in the lower nostril, when the patient is lying down, probably due to the blood gravitating to the lower side of the head and thus increasing the swelling in the hypertrophied tissue.

On account of the nasal obstruction mouth breathing is necessary, especially at night, and a secondary inflammation develops in the nasopharynx and pharynx, and often extends to the eustachian tubes, producing a sense of fulness in the ears and deafness.

The irritation and accumulation of sticky secretion in the nasopharynx, particularly during the night, is often one of the most annoying symptoms, and the patient's efforts to remove the secretion frequently cause nausea or vomiting.

Pain is never present, but the discomfort caused by the obstruction is very annoying, especially in nervous patients.

Hypertrophy of the middle turbinals interferes with the drainage and aëration of the frontal and ethmoidal sinuses, and causes frontal headache, which may be so severe that any mental work is difficult. Sneezing is also a common symptom when the middle turbinals are involved. Decrease in the acuteness or loss of the sense of smell, anosmia, on account of the ob-

struction of the olfactory cleft, is another symptom which may accompany hypertrophy of the middle turbinals.

The voice loses its normal resonance and is often hoarse, as a consequence of the nasal obstruction and the secondary inflammation in the larynx.

Hypersecretion is also a constant symptom in hypertrophic rhinitis. The secretion is usually thick and tenacious and, on account of the nasal obstruction, it is difficult for the patients to get rid of the secretion. Such patients develop the habit of blowing the nose vigorously and picking away the dried secretion in the anterior nares, often causing excoriations on the septum and epistaxis.

Odor, usually slight and not offensive, is due to fermentation in the retained secretion and is caused by saprophytic bacteria. This fermentation adds to the irritation in the nose and tends to increase the hypertrophy.

Purulent secretion and foul odor are never due to hypertrophic rhinitis alone, and always indicate some complication, such as sinus infection, ulceration and necrosis, syphilis, or a foreign body in the nose.

**Diagnosis.**—The examination of the nose should be systematic and complete. Looking into the nares, the anterior portion of the inferior turbinal stands out more or less prominently, depending upon the amount of hypertrophy. If the hypertrophy is large it may be the only turbinal tissue which can be seen.

Gentle pressure on the prominent tissue with a probe helps to determine the relative firmness of the swelling. If the swelling is due to turgescence of the membrane, the probe easily makes a depression in it, which disappears as soon as the probe is removed. This indicates either an acute transitory swelling or an early stage of the disease. If the tissue shows a slight resistance to the probe and the depression fills in slowly, it indicates actual hypertrophy.

The posterior ends of the turbinals should be examined next with the nasopharyngeal mirror. Posterior hypertrophies are usually mulberry-like growths which project out into the nasopharynx. They may be smooth and quite regular, and in either case they often fill the inferior half of the posterior nares. Occasionally the mulberry hypertrophies develop on the lower border of the inferior turbinal.

The color of the mucous membrane is a dusky red, lacking the pink color of the normal membrane, and the bright red of an acute inflammation.

After examining the anterior and posterior nares, a 2 per cent. cocaine solution should be applied on the turbinals, and then, in three to five minutes, the nose should be examined again. If there is no true hypertrophy, the application of the cocaine solution will cause the contraction of the turbinal tissue and the normal contour of the bones will show through the membrane. The nasal fossæ appear quite roomy and the nasopharyngeal wall may be seen through the nose.

Hypertrophies will show as thickened areas, obscuring the normal outlines of the bones, and when touched with the probe will still show some indentation.

Even after the application of the cocaine the lower border of the inferior turbinal may rest upon the nasal floor and may be moved laterally with the probe. Thick mucous secretion is often found between this pendulous hypertrophy and the lateral nasal wall.

**Differential Diagnosis.**—The careful use of the probe, after the application of cocaine, easily differentiates the lesions of hypertrophic rhinitis from septal deformities, foreign bodies, tumors, syphilis, and other constitutional infections.

**Prognosis.**—The prognosis is always good, with proper treatment, unless the patient's occupation and environment are causative factors which cannot be changed. Even in such cases temporary relief may be obtained by treatment.

**Treatment.**—The amount of hypertrophy and the severity of the symptoms should determine the method of treatment in each case.

In mild cases the use of an alkaline spray helps to dislodge and wash away the thick secretion. The best spray for such a purpose is normal saline solution. For psychologic purposes some one of the standard preparations may be added to the normal saline solution. Such preparations color the saline solution and give it a pleasant odor.

If the solution is prepared by the patient, specific instructions should be given about the quantity of the ingredients to be used, so as to avoid making a strong solution which will be irritating rather than beneficial. The normal saline tablets provide an easy way for preparing the saline solution.

After using the alkaline spray some oily preparation should always be used to protect the mucous membrane. The camphor and menthol ointment is one of the best preparations for this purpose and the collapsible nasal tube facilitates its use.

R.	Camphor.....	} āā gr. ij.....	Gm.
	Menthol.....		0.15
	Petrolatum.....		3j..... 30.00

M. S.—Put in collapsible tubes and label: To be used as directed.

The patient should be instructed to apply a little of the ointment in each nostril, then take two or three short, quick inspirations, and in this way distribute the ointment over the nasal membrane.

Often the menthol ointment alone, without the use of any spray, will give very material relief and will be used more regularly by a busy patient than when combined with a spray.

The local application, by the physician, of a 1 per cent. zinc chloride solution on the inferior turbinates two or three times a week, supplemented by the use of the menthol ointment, will frequently cure the mild cases of hypertrophic rhinitis.

**Cautery.**—If these conservative methods of treatment, after faithful trial for three or four weeks, fail to give material relief, the electric cautery should be used.

The use of acids for cauterizing the nasal tissue is not advisable, because it is impossible to control their action, and the fused bead on an applicator may drop off and do much damage to the normal tissue.

It should always be remembered in using the cautery that the contraction of the scar tissue decreases the swelling in the hypertrophied mucous membrane and that the object of the cauterization is to provide better breathing space by means of this contraction, with the destruction of as little tissue as possible. The electrode should be a narrow one with a blunt point. The current should be regulated so as to heat the electrode to a cherry-red color, not to a white heat.

*Cocainization.*—After gently spraying the nose with a normal saline solution, wipe out the vestibule with small pledgets of cotton. Dip a small applicator in a 10 per cent. cocaine-adrenaline solution, and after shaking off any excess of the solution make an application on the tissue to be cauterized and on that part of the septum directly opposite. After waiting about three minutes make a second application with the same applicator, without dipping it in the cocaine-adrenaline solution. After waiting again about three minutes, with a fresh applicator make a third and fourth application in the same way as the first two were made.

Then, with fairly firm pressure, touch the tissue with a probe to determine the amount of anesthesia. Usually after four applications there is no sensitiveness to pain.

*Technic of Cautery.*—Wipe the membrane dry with small pledgets of cotton, insert the cold electrode in the nasal fossa, carrying it back to the posterior end of the hypertrophy, complete the electrical contact to heat the point, and apply the hot point to the tissue, drawing it anteriorly in a straight line. Remove the point, while it is still hot, from the tissue, otherwise it will adhere to and tear the tissue.

The depth of the cautery should depend upon the amount of hypertrophy and varies from light cauterization in mild cases to a deep groove, extending down to the bone, in thick, firm hypertrophies.

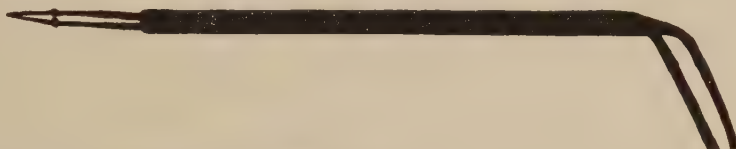


Fig. 43.—Type of electrode, with a narrow point, to be used in cauterizing turbinal hypertrophies.

After completing the cautery, insufflate enough bismuth subnitrate powder to cover the cauterized area. This is usually the only after-treatment that is necessary or advisable. A thick grayish eschar forms and the nasal obstruction may be temporarily increased by the inflammatory reaction. The eschar will slough off in one to two weeks, and the contraction of the scar tissue will increase the breathing space.

If the cautery has touched the membrane on the septum also, the inflammatory swelling is liable to bring the two cauterized spots together and adhesions will form unless the burned surfaces are kept separated for at least one week. To prevent such adhesions a small piece of gauze, covered with oxide of zinc ointment, should be inserted between the two surfaces daily until the new mucous membrane forms.

*Localized Hypertrophies.*—The pendulous hypertrophies along the inferior border of the inferior turbinal and localized hypertrophies, usually on the anterior or posterior end of the inferior turbinal, or on the anterior end of the middle turbinal, should be removed with the cold wire snare.

*Technic.*—The same surgical technic which is employed in all modern intranasal operations should be used. All instruments, cotton, tampons, and gauze should be sterile. The nasal fossa should be sprayed with a normal saline solution, and the vestibule and skin on the nose and surrounding area of the face should be carefully wiped off with alcohol before beginning the operative work.

The operative field should be cocaineized in the same manner as described for cauterization.

Then surround the hypertrophy with the snare loop, holding the cannula firmly against the tissue just anterior to the portion to be removed, and slowly close the snare. The excised tissue may cling to the snare and be removed with it, but if the mass remains in the nose, remove it with nasal forceps.

If the hypertrophy of the inferior border of the inferior turbinal extends along the whole length of the turbinal, cut the anterior portion free with nasal scissors, making the incision parallel with and just below the lower border of the turbinal bone, and then complete the excision of the whole mass of hypertrophied tissue with the snare.

Frequently there may be some difficulty in placing the snare loop around hypertrophies on the posterior end of the turbinals. Bending the loop slightly, so as to make it curve toward the hypertrophy and careful manipulation to avoid bleeding and obscuring the field, help to make this part of the operation successful.

After the hypertrophy has been removed, insufflate bismuth subnitrate powder freely to cover the operative field. As the turbinal tissue is very vascular, some packing must be applied for at least twenty-four hours, even though the hemorrhage at the time of the operation may be slight, due to the use of cocaine and adrenaline.

The Simpson nasal tampons are the best and most convenient form of packing. They are made of cotton compressed between two layers of gauze and increase in thickness as the cotton absorbs moisture. When saturated with moisture they become at least twice as thick as when they are dry, and allowance must be made for this increased thickness before placing one in the nose, so as to avoid excessive pressure. To decrease the thickness of the tampon split it and remove a few layers of cotton from the central portion, leaving the gauze covering intact on the outside of each piece of the tampon. This prevents the cotton from fraying and pulling apart when the tampon is removed.

The tampon may be easily trimmed to any desired size and shape with bandage scissors. It should be large enough to produce a little firm, even pressure beyond the site of operation in all directions. If it is to be placed under the turbinal, it should be bent at a right angle, along the long axis of the tampon, so as to slip into position easily. Before inserting the tampon in the nose smear both sides liberally with the oxide of zinc ointment. This makes its removal easier and thus helps to prevent secondary hemorrhage.

If the posterior end of the turbinal has been excised, the tampon should be long enough to project out into the nasopharynx at least  $\frac{1}{2}$  inch beyond the septum. This free end will mushroom around the turbinal and cover it.

The tampon should be left in position from twenty-four to thirty-six hours. At least one-half hour should be devoted to removing the tampon, moving it very slightly at intervals of five to ten minutes, or even longer intervals if there is any bleeding. This slowly relieves the pressure and allows new clots to form and usually prevents secondary hemorrhage. After removing the tampon, insufflate bismuth subnitrate powder freely, caution the patient against vigorously blowing the nose, and do nothing more in the way of after-treatment unless complications arise which necessitate it.

Gauze packing, instead of the tampon, may be used if for any reason it is preferred. Sterile gauze,  $\frac{1}{2}$  inch in width, selvaged on both edges, should be used. It may be dipped in sterile vaseline or zinc ointment. The first portion of the gauze should be carried well back into the posterior superior part of the nasal fossa, packing from above downward and forward with fairly firm pressure. From  $\frac{1}{2}$  to 1 yard of gauze will be needed. It should be carefully removed, a small portion at a time, at intervals of five or ten minutes in the same manner as described for the removal of the tampon.

Fortunately the postoperative reaction, after such a nasal operation, is usually slight if the operative technic has been good, and healing takes place promptly if the operative field is not molested. Sprays and douches and local applications should not be used.

Occasionally secondary hemorrhage may make it necessary to repack the nose. More rarely, excessive bleeding posteriorly may necessitate packing the nasopharynx also. This is always accompanied by the danger of an infection in the eustachian tubes and ears, and the packing in the nasopharynx should never be left in position more than twenty-four hours.

Never cauterize or operate in both nasal fossæ at the same time. Wait until the operative field in one fossa has healed before doing any operative work in the other fossa.

#### ATROPHIC RHINITIS

**Definition.**—An atrophy of the nasal mucous membrane and the underlying soft tissue, eventually involving the turbinal bones.

**Synonyms.**—Rhinitis sicca; ozena; dry catarrh.

**Etiology.**—The etiology of this disease is unknown. Many theories have been proposed and in recent years much bacteriologic work has been done in trying to determine its etiology, but no pathologic condition or specific organism has ever been proved to be the causative factor.

In 1889 Perez described an organism which he called the *coccobacillus foetidus ozenæ* and attributed the odor to the action of this bacillus. This opinion has since been affirmed by some men and denied by others.

Atrophic rhinitis occurs more frequently in poorly nourished patients who live in unhygienic surroundings, but this simply means that such patients are more susceptible to this as well as to other diseases. It may be hereditary, that is, the tendency may be passed along from one generation to another, but this also gives no clue to the primary etiology.

Syphilitic lesions in the nose, which destroy the major portion of the mucous membrane and the turbinal structures, produce large, roomy nasal fossæ, lined with scar tissue instead of normal mucous membrane, and the end-result is practically the same as in atrophic rhinitis.

Overzealous and indiscriminate intranasal operations also cause similar conditions, but neither of these conditions is true atrophic rhinitis.

**Prophylaxis.**—Atrophic rhinitis usually begins between the ages of ten and twenty years, and proper attention to diet, hygiene, and exercise, with careful, persistent treatment of the nose, if there are any indications of this disease, are the best prophylactic measures.

**Pathology.**—The first manifestation of the disease appears in the epithelial layer of the mucous membrane, which decreases in thickness, and the cilia are lost. The glands degenerate, the secretion becomes thick, and as it dries it accumulates and forms crusts. The arteries and veins decrease in

size and in number, and the tissue under the mucous membrane loses its vascularity and becomes very dense and thin. The nourishment of the turbinal bones is interfered with, and the inferior and middle turbinals atrophy and finally are left as mere stumps.

**Symptomatology.**—Nasal obstruction and crust formation and irritation in the nasopharynx, pharynx, and larynx are the symptoms of which the patient complains. The nasal obstruction varies according to the amount of accumulation of crusts in the nose, and disappears when the nasal fossæ are kept clean.

The accumulation of the crusts in the nose, especially on the septum, annoys the patient, and the constant picking at them and removal of them with the fingers often causes excoriations on the septum which may be followed by epistaxis, ulceration, and perforation of the septum.

The irritation in the rest of the respiratory tract is due to the interference with the normal respiratory function of the nose. The inspired air is not warmed, cleaned, and saturated with moisture, and, consequently, the mucous membrane in the throat is inflamed and dry and irritable. Crust formation in the pharynx and larynx, and even in the trachea, may develop in the latter stages of the disease.

The odor from the retained secretion and crusts is exceedingly foul and disagreeable. Chaucer's old English word "stink" probably describes the odor better than any other single word. Ozena is more euphonious, but the meaning is the same.

The atrophy and other changes in the mucous membrane destroy the terminal filaments of the olfactory nerves and the patient loses the sense of smell and is unaware of the odor.

**Diagnosis.**—Examination of the nose will show an accumulation of greenish-black crusts, more or less completely filling both fossæ, depending upon the severity of the disease and the length of time which has elapsed since the crusts were removed. The mucous membrane of the nasopharynx and pharynx appears dry and stiff, with spots of crust formation on it. When the crusts in the nose have been removed, a thick mucopurulent discharge will be seen covering the mucous membrane.

The fossæ are roomy and the size of the inferior and middle turbinals varies according to the amount of atrophy. In cases of marked atrophy the ostia of some of the accessory sinuses may be easily seen and the eustachian prominences and nasopharyngeal wall also. The disagreeable odor helps to confirm the diagnosis.

**Differential Diagnosis.**—*Sinusitis.*—If in addition to the atrophic rhinitis, some of the paranasal sinuses are infected, the roomy fossæ often make the location of the source of the purulent discharge easier, otherwise the diagnosis of sinus infection should be established in the routine way.

*Syphilis.*—An active tertiary syphilitic lesion with necrosis in the nose will produce a foul odor with crust formation, but the site of the lesion, usually localized on the septum or the floor of the inferior turbinal, surrounded by a zone of intense hyperemia, and the history of its rather rapid development accompanied by pain and some bloody discharge, will differentiate it from atrophic rhinitis.

Extensive syphilitic destruction of the intranasal structures may produce large, wide fossæ in which crusts form, but the scar tissue in the nose, usually causing external deformity also, and the irregular destruction of the

nasal tissue distinguishes this condition from the uniform, symmetrical loss of tissue caused by atrophy.

The Wassermann test, the history of a primary sore, and other manifestations of syphilis, all help to make the differential diagnosis in these cases.

*Foreign Bodies.*—Any such bodies which have remained in the nose for a long time will cause crust formation and a bad odor, but careful examination with a probe, after applying cocaine and adrenaline, and the presence of granulation tissue will establish the diagnosis in such cases.

**Prognosis.**—The prognosis, as to the curing of atrophic rhinitis, is bad. The fetor, nasal obstruction, and crust formation may be relieved or even eliminated by treatment if the patient will faithfully co-operate with the physician.

**Treatment.**—A nasal douche should be used carefully to soften and remove the crusts and thick secretion. An ordinary douche bag fitted with the blunt nasal tip, or any receptacle with an outlet to which the nasal tip can be attached with a rubber tube, will serve the purpose. While using the douche, the patient should keep the head bent forward and breathe through the mouth. The quantity of the solution to be used will depend upon the amount and hardness of the crusts. A quart or more should be used, if necessary, to thoroughly cleanse the nose. The douche should be used regularly at least twice a day as long as there is any crust formation and odor. One teaspoonful of salt (sodium chloride) and two of baking soda (sodium bicarbonate) dissolved in a pint of water will make a normal saline solution which is inexpensive and can be easily prepared by the patient. Such a solution is not irritating and is as efficacious as any.

The use of a nasal douche is always accompanied by the possibility of an infection being carried into the eustachian tube and middle ear. The patient should be told of this danger, and cautioned against elevating the douche bag more than 2 or 3 inches above the level of the nose, and also against blowing the nose vigorously. During the cold weather the patient should remain in the house for at least fifteen minutes after using the douche.

Following the douche, an oily spray or ointment should be used. One containing  $\frac{1}{2}$  or 1 per cent. of menthol makes an agreeable application, which also decreases or masks the odor.

The application, by the physician, of some stimulating solution to the mucous membrane, two or three times a week, for a period of four to six weeks, and then repeated again at intervals of two or three months, benefits these cases very materially and in mild cases may even stop the formation of crusts and prevent the odor. Before making such an application the nasal fossæ should be carefully cleaned with small pledgets of cotton on an applicator, or in a pair of forceps, removing the crusts and thick secretion. Then one of the following solutions should be applied gently but thoroughly:

Silver nitrate solution, 5 or 10 per cent.

A 2 per cent. solution of ichthyol in glycerine.

A 1 per cent. solution of iodine and potassium iodide in glycerine.

All of the nerve-endings in the mucous membrane are obtunded by the atrophy, and the applications of these solutions are usually not painful. If they do cause pain, the strength of the solution should be reduced.

If the patient will co-operate with the physician, the disagreeable symptoms in atrophic rhinitis can be controlled, but as the condition improves the patient is very liable to neglect the treatment and the symptoms soon return.

Paraffine injections to build out the lateral nasal walls and operation to advance the walls and make the fossæ narrower have been tried, but the results have not been satisfactory.

#### FOREIGN BODIES IN THE NOSE

**Animate Foreign Bodies, Nasal Myiasis.**—Parasites and worms in the nasal fossæ are comparatively rare in temperate and cold climates, but are not uncommon in the tropics. Screw-worms (*Chrysomya macellaria*) and maggots are the most common variety of parasites found in the nose.

They are practically never found in the normal fossæ. Any of the pathologic conditions in which there is pus formation, and especially those lesions which produce an odor, attract flies and parasites and provide an ideal incubator for the development of the eggs.

*Symptomatology.*—Irritation, sneezing, and increased discharge are the first symptoms. Later, the discharge is usually streaked with blood and the irritation and inflammation in the nose becomes intense and very painful. Externally there is swelling of the nose and surrounding tissue of the face.

*Diagnosis.*—The larvæ usually develop in large numbers within two or three days after the eggs have been deposited in the nose, and they may be seen in the fossæ or even in the vestibule.

If the number of larvæ is small, the diagnosis may be difficult, but a sudden, unusually severe inflammation, developing after sleeping out-of-doors, should suggest the possibility of a parasitic infection, and repeated careful examinations, after applying cocaine and adrenaline, should be made in searching for the larvæ.

*Treatment.*—A solution containing 20 per cent. of chloroform and 80 per cent. of warm water should be instilled into the fossæ repeatedly, with a medicine dropper, or the same solution may be used as a nasal douche. This will stupify the larvæ so that they may be removed with forceps, or with a douche of warm water.

**Inanimate Foreign Bodies.**—Such foreign bodies are found most frequently in young children. Their character is limited only by the size of the body. Any small article which a child finds may be pushed into the nostril. Insane patients manifest the same proclivity. Foreign bodies in the mouth and pieces of food may be regurgitated and lodged in the posterior nares. Buttons, seeds, and pieces of wood are the objects most commonly found.

In adults metallic splinters, from accidents in factories, and bullets are the usual type of foreign body. These objects enter the fossa through the external walls and the diagnosis is established at once.

*Symptomatology.*—The foreign body causes irritation, inflammation, and ulceration of the surrounding mucous membrane. At first there is a serous discharge usually accompanied by sneezing and lacrimation. The secretion soon becomes purulent and is often slightly tinged with blood. Later, it may become fetid and suggest atrophic rhinitis.

Nasal obstruction steadily increases in the affected nostril, and usually in the other nostril also, on account of the inflammatory reaction.

A unilateral nasal discharge in a child should always suggest the possibility of a foreign body.

*Diagnosis.*—In children a general anesthetic is necessary for a satisfactory examination. Nitrous oxide gas is the ideal anesthetic for such a purpose, or if this is not available, the primary stage of ether anesthesia.

If the foreign body has been in the nose only a few days, it may be seen lodged usually in the inferior meatus, less frequently in the middle meatus. If several weeks, or a longer time, have elapsed since it was introduced, it may be covered with granulations, lime-salts, or thick secretion. The character of the secretion may resemble the membrane seen in nasal diphtheria, but diphtheria is usually bilateral, accompanied by a membrane in the throat, bilateral enlargement of the glands, and constitutional symptoms.

In such cases adrenaline should be applied and the secretion removed with small pledgets of cotton, without causing any bleeding, if possible, so as not to obscure the field. If the foreign body cannot be seen, gentle manipulation with a probe will locate it. Extreme caution should be exercised in using the probe, not to push the foreign body into the nasopharynx and thus avoid the possibility of its being inspired into the trachea or bronchi.

In adults the search for a foreign body should be made under cocaine and adrenaline anesthesia, but the same precautions should be used as in a child. If the foreign body is a metallic one or some other substance which will cast a shadow, a roentgenograph will confirm the diagnosis, but such things as seeds or small pieces of bone cannot be identified in this way.

*Treatment.*—Complete control of the patient, so that there may be no jumping or sudden movement of the head, and careful manipulation by the physician are the secrets of successful removal of foreign bodies. In children a general anesthetic is necessary; in adults cocaine anesthesia is preferable unless the patient is intractable or the foreign body is firmly embedded. In either case adrenaline should be carefully applied. If the foreign body has been in the nose only a short time, the contraction of the tissue, after the adrenaline application, will bring it into view and it can be teased out with a small hook or bent probe or removed with forceps.

If the object is hidden by granulation tissue, this tissue should be removed, controlling the hemorrhage with adrenaline. When the foreign body has been exposed, it should be grasped with forceps and removed. In such cases if the foreign body, or its removal, has produced excoriations on the septum and the turbinal tissue, the raw surfaces should be separated by a small gauze packing, dipped in oxide of zinc ointment. This packing should be renewed daily until there is no danger of adhesions forming.

Bullets and pieces of metal embedded in the nasal fossæ usually carry some infection in with them and they should be removed promptly. If they are located in the upper portion of the nose, meningitis frequently develops, the infection reaching the meninges through some fracture, usually in the cribriform plate.

The removal of such foreign bodies may necessitate the amputation of the middle turbinal, an ethmoidal exenteration, or an external operation.

#### RHINOLITHS

**Definition.**—Rhinoliths, or nasal calculi, are concretions, resembling stone, which form in the nasal fossæ. Their chemical composition varies, but they usually contain calcium phosphate or calcium carbonate. Other

salts may enter into their composition. They are rough and irregular in outline and may become quite large if they are allowed to remain in the nose for a long time.

**Synonyms.**—Nasal calculus. Nasal stone.

**Etiology.**—The salts from the nasal secretion are deposited around a nucleus, which is probably always a neglected foreign body. In cases of long standing it may be difficult to identify the foreign body if it happens to be a bean or some soft substance, but a hard body, such as a cherry stone, may be recognized easily.

**Symptomatology.**—The rhinolith grows slowly in all dimensions by the addition of irregular concretions, and is a constant source of irritation, causing hypersecretion and nasal obstruction. The discharge is mucopurulent or purulent and is frequently slightly bloody.

In cases of long standing the growth of the rhinolith produces pressure necrosis, and it may extend through the septum or destroy part of the turbinal tissue. In such cases pain and reflex neuralgia are frequently present. Infections in the maxillary or other sinuses may develop.

**Diagnosis.**—After the application of cocaine and adrenaline, and the removal of the secretion, it may be possible to see the rhinolith lodged, usually in the middle or inferior meatus.

If it is partly or entirely hidden by granulation tissue, the careful use of a probe helps to establish the diagnosis. The peculiarly hard, rough, stone-like feel to the probe will usually differentiate a rhinolith from necrotic bone.

**Prognosis.**—The prognosis is good. If the rhinolith is entirely removed it never recurs.

**Treatment.**—The application of cocaine and adrenaline is usually necessary to establish the diagnosis. When this has been done, more cocaine may be applied if necessary, and the rhinolith should then be removed with a pair of forceps.

If the stone is so large that it cannot be removed without considerable injury to the surrounding tissue, it should be crushed with a pair of strong forceps, and removed in pieces.

The formation of adhesions should be prevented in the manner described under Foreign Bodies.

JOHN M. INGERSOLL.

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## THE SEPTUM

**Embryology.**—In order to understand the embryological development of the nasal septum it is necessary to review briefly, in part, the evolution of the embryonic face. For a more detailed account the current works on embryology must be consulted. During development the nasal septum passes through three stages: The membranous, cartilaginous, and, finally, the mixed cartilaginous and osseous or that of the adult.

*Development of the Early Nasal Septum.*—The nasal septum is developed from the mesenchymal mesoderm. The earliest rudiments of the face are formed by the development of the first pair of branchial or visceral arches and the frontonasal process. The former grow ventrally and each divides

into two parts: A mandibular process which unites with its fellow of the opposite side to form the posterior mouth boundary; and a maxillary process which unites with the frontonasal process to form the anterior mouth boundary.

The frontonasal process makes its appearance in the third week of development as a localized thickening of the mesodermic tissue on the ventral wall of the primary forebrain vesicle. During the fifth week it thickens greatly and by the concomitant development of the nasal pits becomes divided into two parts—a lateral and a medial nasal process. The lateral nasal processes really bud out from the initial thickening, one on each side, above the nasal pits, and growing downward form their external boundary.

It is before the end of the third week of development that the pits make their appearance as a localized thickening of the ectoderm just in front of the oral fossa. By ingrowth of this thickened area the ectoderm gives origin to the olfactory epithelium.

The nasal pits from the first communicate with the oral fossa by a relatively broad groove. Each lateral nasal process, as stated above, forms the outer boundary of a pit and is separated from the maxillary process of the same side by the lens pit and a groove; the latter is the naso-optic groove which begins in the lens pit and opens into the nasal pit. It is destined to become the future nasal or lachrymal duct. The lateral nasal processes become the *alæ nasi*.

The medial nasal process thickens greatly during the fifth week along the inner margins of the nasal pits, forming the globular processes, the depressed area between the latter finally becomes transformed into portions of the upper lip and movable part of the septum (Fig. 44). During the sixth week the frontonasal process which bounds the oral fossa anteriorly is joined on each side by the united maxillary and lateral nasal processes. This brings about a division between the oral fossa or cavity and the nasal pits and forms, as yet crudely, the external nose. The definite formation of the external nose is indicated about the fifth week. The orifices of the nasal pits have become the anterior nares, the pits behind them have become short canals which, by deepening, finally open into the primitive mouth cavity, forming the early choanæ. The canals of the open nares at times become temporarily closed by proliferating epithelial cells. This obstruction is most common between the fiftieth and sixtieth days of embryonic life (Schaeffer).

As indicated above, the portion of the frontonasal process between the nares forms the central part of the upper lip, the premaxillary process and the *primary nasal septum*, while the superficial parts give rise to the tip and dorsum of the nose. At this time the mouth and nasal cavities communicate behind the anterior portion (Figs. 44, 45).

Prior to the development of the hard palate the oral cavity and nasal fossæ communicate broadly, and the developing tongue projects toward the primary septum and between the early palatal processes (Figs. 45, 46). The latter continue to grow toward the median plane. This results, with their union with each other and with the developing septum. It is in this way that the nasal fossæ and mouth cavity become definitely separated from each other, the choanæ now opening into the pharynx. This is completed by the end of the third month.

The *primary nasal septum*, as described above, grows dorsally toward the pharynx and caudally toward the mouth in the formation of the secondary

nasal septum, ultimately fusing with the nasal surface of the palate, as already mentioned, and forming a free border dorsally between the definitive choanæ. It is, therefore, obvious that the definitive or final nasal septum is made up of primary and secondary portions, both derivatives of the mesial part of the frontonasal process. With these developments the nasal septum is formed not as a paired structure, but from the ingrowth of the mesoderm.

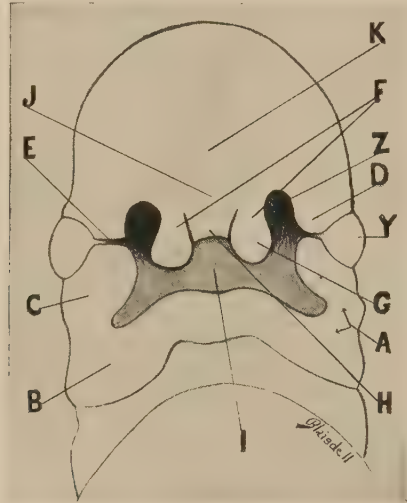


Fig. 44.—Face of a human embryo of 8 mm. Note carefully the nasal pits and frontonasal process.

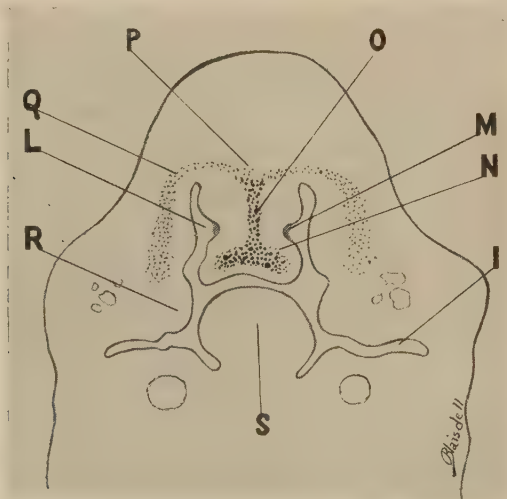


Fig. 45.—Section through the head of a human embryo of 18.5 mm. Showing membranous stage of the nasal septum. Mouth and nasal fossæ in continuity.

**Membranous Stage of the Nasal Septum.**—The membranous nasal septum arises as a condensation of the mesenchymal tissue within the primary nasal septum. It is a part of the differentiating membranous rudiment of the skeleton of the head and is particularly connected with the development of the nasal capsule (Fig. 45).

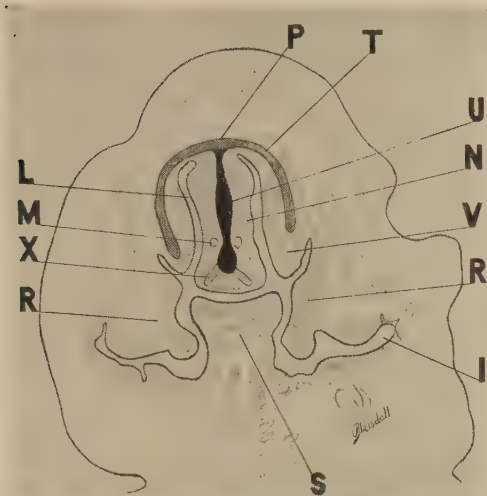


Fig. 46.—Section through the head of a human embryo of 2 mm. greatest length. Showing cartilaginous nasal capsule; mouth and nasal fossæ in continuity.

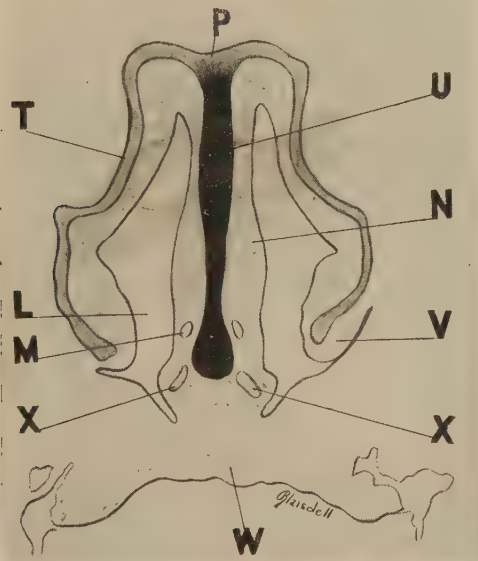


Fig. 47.—Frontal section through the nasal fossæ and hard palate of a human fetus of 4.2 cm. sitting height. Mouth cavity closed off from the nasal fossæ.

Explanation of Figs. 44-47: A, First visceral arch dividing into (B) mandibular and (C) maxillary processes; D, lateral nasal processes; E, naso-optic groove—future lacrimal duct; F, frontonasal process or median nasal process; G, processus globularis; H, infra-nasal area, which becomes transformed into philtrum, portion of upper lip, membranous septum, or septum mobile; I, oral fossa (J), to become tip of nose; K, area triangularis, which becomes the dorsum naso; L, nasal fossa; M, Jacobson's organ; N, nasal septum; O, differentiating membranous septum (deep black); P, ethmoidal region; Q, lateral plate of differentiating membranous capsule; R, palatal process (becomes hard palate); S, tongue projecting into nasal fossa toward nasal septum between the palatal processes; T, lateral plate of cartilaginous nasal capsule; U, cartilaginous nasal septum (deep black); V, inferior concha; W, hard palate—roof of mouth; X, Jacobson's cartilage; Y, eye; Z, nasal pit.

The membranous capsule includes the ethmoid region with two lateral plates and a median perpendicular portion or membranous nasal septum. By the middle of the third month of embryonal life the nasal capsule is undergoing a cartilaginous change. As stated above, up to this period the oral and nasal cavities communicate widely and the tongue is in relation with the nasal septum between the palatal processes which are gradually advancing inward and reducing the intervening space. The primary nasal septum is at first thick, separating widely the nasal fossæ. The mesenchyma within it and not concerned in the formation of the membranous septum becomes the connective tissue and corium of the mucous membrane, the epithelium being of ectodermic origin. Two epithelial invaginations develop in its walls, one on each side, as the rudiments of Jacobson's organ. These, during the membranous stage of the septum, become surrounded by condensed mesenchymal tissue or membranous capsule.

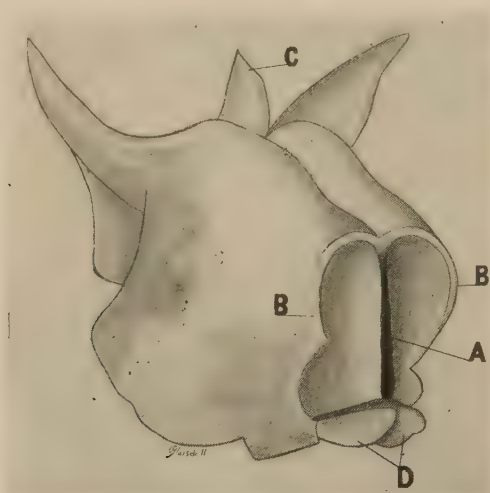


Fig. 48.—A reconstruction of the cartilaginous nasal capsule of a human fetus of about twelve weeks, seen from in front and partly from the side: A, Nasal septum; B, lateral wall; C, crista galli; D, Jacobson's cartilage. (Modified from Kallius, from Bardeleben's Handbuch.) (See Figs. 45-47.)

**Cartilaginous Stage of the Nasal Septum.**—The development of the cartilaginous nasal septum must be correlated with the development of the cartilaginous nasal capsule. Figures 46 to 48 will contribute to this end.

By the middle of the third month of embryonal life the nasal septum is in a precartilaginous stage and, by ingrowth of cartilage from the body of the sphenoid, gradually becomes converted into a cartilaginous plate. The primary septum, originally thick, becomes relatively thin as it passes into its secondary stage. The septum in its definitive state becomes thinner and thinner, the nares coming to occupy positions nearer the midplane as the laminar plate of cartilage develops in its substance. The cartilaginous septum is surrounded by perichondrium.

The mucosa of the septum is at first fairly heavy, and may present well-developed folds in the region of the later vomer. These folds increase in size until about the seventh or eighth month; they disappear in early in-

fancy as a rule. By the continued development of the epithelial invaginations mentioned above a paired tubular organ in the ventrocephalic portion of the septum, immediately dorsal and cephalic to the incisive foramen, becomes the vomeronasal or Jacobson's organs.

The glands of the septal mucosa develop as solid epithelial ingrowths of the surface epithelium and do not mature until after birth. While the nasal septum is still cartilaginous, the palatal processes grow toward each other until in the eighth week their union begins anteriorly, the tongue receding from between them as the lower jaw develops more fully. By the ninth week union has taken place as far back as the extent of the hard palate, uniting with the ventral border of the nasal septum.

The original continuity of the cartilaginous nasal septum becomes interrupted by ingrowth of connective tissue along the lines of the parts to be derived from it (Fig. 49).

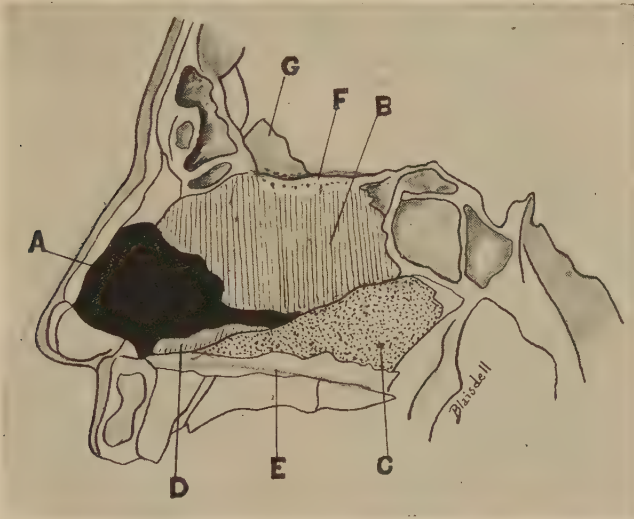


Fig. 49.—A diagram of a dissection showing the osseous and cartilaginous septum of the nose. Adult: A, Cartilago septi nasi; B, mesethmoid; C, vomer; D, cartilago vomeronasalis; E, superior maxilla; F, cribriform; G, crista galli. (Modified from Schaeffer.)

**The Mixed Cartilaginous and Osseous or Definitive Stage of the Nasal Septum.**—As just stated, the continuity of the cartilaginous nasal septum becomes broken into subdivisions by the ingrowth of connective tissue along the lines of inherent limitations of the parts derived from it, namely: The perpendicular plate of the ethmoid, vomer, vomeronasal, and septal cartilages.

**Perpendicular Plate of the Ethmoid.**—The greater part of the posterior portion of the cartilaginous nasal capsule becomes the ethmoid. The perpendicular plate is derived from the cephalic portion of the cartilaginous septum, and it is here that the ossification begins during the first year of postfetal life from a single center. If Fig. 48 be studied, it will be seen that the cartilaginous nasal septum is continuous dorsally with the median part of the ethmoid region of the cartilaginous nasal capsule. The union of the perpendicular plate with the cribriform plate probably takes place late in

the sixth year, and undergoes complete ossification about the end of the seventeenth year.

*Vomer.*—The true definitive vomer develops on each side of the caudal and dorsal portion of the cartilaginous nasal septum from a pair of ossification centers during the eighth week of fetal life. These centers unite beneath the caudal border of the septal cartilage, but grow headward on each side of the septum as two plates, thus enclosing the cartilage. These bilateral plates of the vomer unite forward at expense of the imprisoned cartilage, union being completed by the fifteenth year (Schaeffer).

The vomer of the young infant shows clearly its formation in two plates. In the adult the bilateral development is indicated merely by the groove between the alæ and by the groove on the lower part of the ventral border where it receives the triangular septal nasal cartilage. It is to be classed as a membrane bone. Ultimately, at about the forty-fifth to the fiftieth year, the vomer and the perpendicular plate of the ethmoid unite (Schaeffer).

*Septal and Vomeronasal Cartilages.*—Septal and vomeronasal cartilages are portions of the cartilaginous nasal septum which remain throughout life. The periosteum of the perpendicular plate of the ethmoid and vomer are not continuous, but interrupted at articulations, where it is united as a thin sutural membrane between the two; neither is the perichondrium of the septal cartilages continuous with the periosteum of the vomer and the perpendicular plate without interruption and interposition of a fibrous membrane.

It is to be remembered that there is no sharp borderline between the different developmental stages or periods given above, but that there is an overlapping, the growth processes begun at one stage being carried over and completed in the next or later stage.

**Anatomy.**—The septum may be divided broadly into a movable and a fixed portion.

The movable portion of the septum is composed of the triangular cartilage and the medial crura of the curved alar cartilages; between these there is fibrous tissue. These parts are easily felt with the thumb and index-finger. The fixed or bony part of the septum comprises the vertical plate of the ethmoid, the vomer, and the anterior and posterior spines of the superior maxilla. The triangular cartilage may extend in a long fibrous process between the vertical plate of the ethmoid and the vomer, even reaching to the body of the sphenoid. The posterior edge of the vomer which presents toward the nasopharynx is more or less vertically placed in the adult. It occupies an almost horizontal position at birth. This is a fact to be remembered in removing adenoids in young infants.

The mucous membrane of the septum is of the columnar ciliated type, except in the olfactory region where the cilia are wanting. It is of medium thickness and is intimately connected with the underlying perichondrium and periosteum. It is usually spoken of as a mucoperichondrium and mucoperiosteum. The superficial layers can be separated to a slight extent from the perichondrium or periosteum. This sometimes causes trouble to the operator in doing a submucous resection, leading him to think that he has raised the entire mucoperichondrium. The mucous membrane in the upper anterior part of the septum, opposite the anterior end of the middle turbinate, sometimes contains an unusual number of mucous glands which thicken it materially.

Jacobson's organ, a small cartilaginous embryological rudiment, lies on the cartilaginous septum immediately above the anterior nasal spine. While closely associated with the olfactory centers in lower vertebrates, it is of no known significance in man.

The anterior part of the movable septum is armed with a number of hairs corresponding to those in the rest of the vestibule.

**Arteries of the Septum.**—The sphenopalatine artery, one of the terminal branches of the internal maxillary artery, enters the nasal fossa at the sphenopalatine foramen. It runs across the under surface of the body of the sphenoid to reach the septum. From this point it runs downward and forward along the septum to the floor. Here it anastomoses with the terminal branch of the descending palatine artery through the anterior palatine canal and

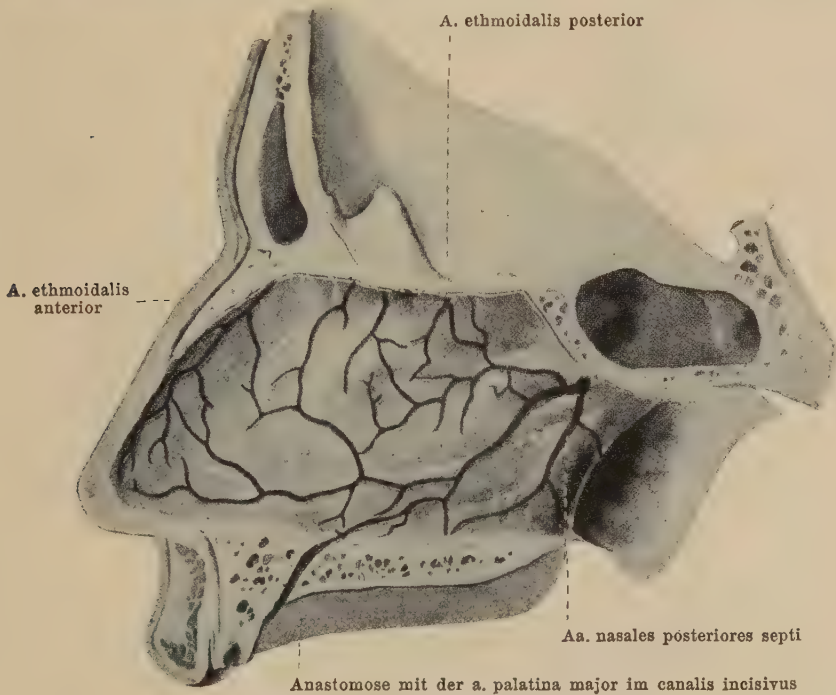


Fig. 50.—Arteries of the nasal septum (Spalteholz).

the inferior artery of the septum, a branch of the superior coronary artery, which enters the nose from the face.

The anterior ethmoid artery, a branch of the ophthalmic artery, enters the nose through the cribriform plate and supplies the upper part of the septum. All of these arteries anastomose freely.

The mucous membrane covering the lower part of the cartilaginous septum is especially well supplied with blood, and this region is called Kieselbach's area. Veins of like names accompany these arteries.

**Nerves of the Septum.**—The sphenopalatine nerve follows the artery of the same name, as does the anterior ethmoid nerve.

The olfactory nerves are composed of bundles of fibers extending centrally from the olfactory cells. These cells, with supporting cells and Bowman's glands, make up the mucous membrane of the olfactory region of the

nose. This region is marked in the fresh specimen by a yellowish coloration due to pigment in the supporting cells. The olfactory cells reach the surface of the mucous membrane between the supporting cells and present hair-like filaments toward the nasal chamber.

The olfactory fibers are gathered together to form about twenty nerve bundles which pass through the cribriform plate into the olfactory lobe. They are encased in sheaths from the dura and pia mater.

The olfactory region on the septum is not absolutely determined, but probably occupies an area that extends downward to about the level of the lower border of the middle turbinate.

**Lymph Vessels of the Septum.**—Some of these channels carry the lymph anteriorly and through the pyriform opening, eventually draining into the

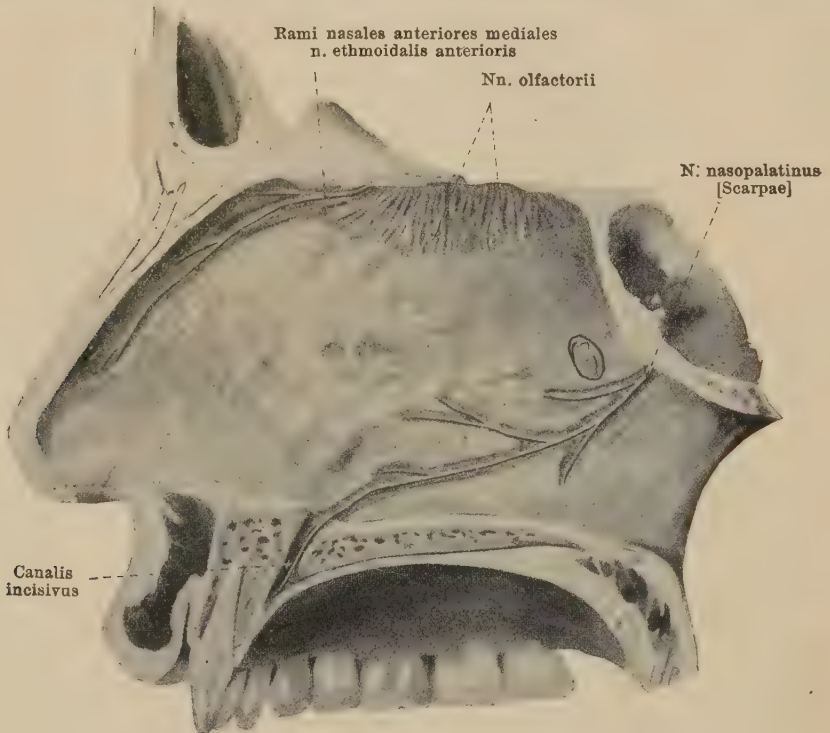


Fig. 51.—Nerves of the nasal septum (Spalteholz).

submaxillary and superficial cervical lymph-glands. Vessels also drain posteriorly and, with the lymph from the other parts of the nose and nasopharynx, penetrate the fascia and the musculature of the pharynx at the fossa of Rosenmüller to enter the retropharyngeal glands and the deep cervical glands.

#### DEFLECTIONS OF THE SEPTUM

Abnormalities of the nasal septum are the cause of much of the pathology that occurs in the upper air passages and in the ears. While the direct effect upon the eustachian tube and middle ear is open to argument, the part played in the determination of chronic nasal sinus infection and the resultant ear and upper respiratory disease is not questioned. The ideal

septum, a thin partition in the middle line dividing the nasal chambers into symmetrical halves, is rarely seen. Irregularities of the form and thickness, however, are only to be considered harmful when they interfere with the normal function.

**Etiology.**—The cause of irregularities of the septum is not definitely established. Traumatism and developmental anomalies are considered to be the principal etiological factors.

Deflections of the septum are said to be rare before seven years of age. However, a check up in the author's clinic would tend to cast some doubt on this assertion. About 40 per cent. in a group of 100 cases, in children of seven years or less, showed some deflection of the septum.

Infants and young children fall frequently when learning to walk. However, this is undoubtedly compensated for by the soft, elastic character of the parts of the nose and its lack of anterior development.

Later in life trauma certainly plays an important part in the causation of septal deflections. Most of the force of the blow is communicated to the cartilaginous septum. Where the nasal bones and nasal processes of the superior maxillary bones are depressed, there may be involvement of the bony septum. Even this may be considered to be rare, except where complete flattening of the bony framework has taken place. Ordinarily the bony sep-



Fig. 52.—Types of septal deformities. (From Pratt, "Intranasal Surgery," F. A. Davis Co., Publishers.)

tum is not in apposition with the nasal bones, and the force of the blow transmitted through the cartilaginous septum would hardly cause fracture of the vertical plate of the ethmoid or the vomer.

The deflections found in the septum due to trauma are easily recognized. They are mostly confined to the cartilaginous part. This may be luxated and block both nares or be thickened by the apposition of several layers due to the comminution resulting from repeated battering such as boxers sustain. These traumatic deviations are found naturally more frequently in males than in females. Trauma cannot be made to assume the chief rôle in the causation of deflections of the septum.

By far the greater number of septal irregularities must be referred to lack of precision in the developmental processes. These developmental anomalies may possibly be induced by trauma. The nasal septum occupies, and is confined by, the space limited above by the ridge of the nose, the cribriform plate and the body of the sphenoid, and below by the palatal process of the superior maxillary bone and the horizontal part of the palate bone. Shortening of this diameter, unless the septum be correspondingly diminished, must result in bending or overlapping of the component septal parts. It is shown that the cribriform plate occupies a lower position in

some skulls than in others, while the difference in the height of the palatal arch is a matter of frequent observation.

The elevation of the palatal processes seems to be influenced by post-natal factors. Mouth-breathing, caused by adenoid or other obstruction, favors the development of this high arched palate. The mouth is held closed normally largely by atmospheric pressure. When this force is nullified by an open mouth, the full weight of the jaws and musculature exert pressure on the sides of the alveolar processes, resulting in elevation of the roof of the mouth. The tongue also exerts a beneficial side pressure upon the alveolar processes when the mouth is closed. This pressure is lost in mouth-breathing.

Ossification of the septum takes place from behind forward. The vomer ossifies in two bony laminae at first separated by cartilage. The overnourishment of one of these laminae, or the meeting of more resistance by one than by the other at the suture lines may be the cause of septal deformities. This and traumatism may account for the fact that most septal deviations are at the anterior part of the septum and at the suture lines.

Deviations may also be caused by crowding from an unusually over-developed middle turbinate, polypi, benign or malignant neoplasm.

Heredity may influence the structure of the septum. It is not unthinkable to attribute an abbreviated space to the inheritance from one parent and a large septum, that must go into it, to another progenitor.

**Classification of Septal Deformities.**—Septal deformities may be classed under three heads. The first variety may be represented by a septum of fairly uniform thickness, found bent to one side or the other.

The second variety embraces the thickenings or overgrowths that occur on the septum. The most common deformity of this type is found as a ledge or crest following the superior border of the vomer upward and backward. Sometimes it is developed into a pyramidal-shaped or hooked point that extends across the nasal cavity, impinging against the lateral wall or turbinates.

The third type may be considered to be a combination of the other two. It is by far the most common, as there are few simple deflections without some piling up of the tissue at the suture lines.

The maxillary spines play an important obstructive rôle. There may be thickening of the septum in the upper part opposite the anterior end of the middle turbinate due to the fact that the developmental tissue is not reduced in the thinning process that ordinarily takes place. There may also be an abnormally abundant collection of glands in this region.

**The Symptoms Arising from Deflection of the Septum.**—All ill effects may be absent and the patient have no complaint to make whatever. Usually, however, depending upon the extent of the abnormality, there is more or less obstruction to respiration. This symptom manifests itself mostly at night, when conscious effort is relaxed. There may be noisy respiration and symptoms of irritation of all the mucous membrane of the upper and even lower air passages due to mouth-breathing.

Probably the most important symptoms associated with the deflected septum are those arising directly or indirectly from sinus infection. The sinuses are usually inflamed as a part of a general nasal infection. The cure of such an acute process depends almost solely upon the surgical principle of adequate drainage. The drainage from the sinuses is poor enough where the septum is as it should be. Where crests, spurs, or deviations encroach

upon the space in the nose, the interference with the spontaneous cure of the sinus infection is unquestionable. Acute sinus disease in the face of bad deflections sometimes gives rise to very serious problems. The deformed and moulded appearance of the middle turbinate that greets the eye of the operator after he has removed a high septal deflection gives mute evidence of the crowding that has been present. He can almost imagine seeing the parts unfold as a blanched tuber stem greets the sun when released from a covering stone.

There is a variety of opinion as to the effect upon the ears of a septal obstruction. It seems probable that the mechanical effect of mouth-breathing upon the ears has been exaggerated. However, if a chronic sinus infection, especially ethmoid and sphenoid involvement of low grade, is a cause of middle-ear disease, then the septal obstruction is certainly important. Theoretically it would seem probable that the air currents passing into the trachea would lead to lessening of atmospheric pressure in the nasopharynx behind a nasal obstruction. This would be accompanied, following the same reasoning, by hyperemia and congestion of the mucous membrane. This, with any protective hyperplasia of connective tissue that would result, would lead to changes about the orifices of the eustachian tubes, causing deafness. The best opinion, however, is inclined to minimize such direct mechanical effect upon the ears. It has been pointed out that in congenital stenosis of the posterior nares there may be no ill effects upon the ears.

Headache and distant reflex symptoms may arise as a result of septal deflections. However, here we are dealing with a problem where we may easily be led astray.

Irregularities in the septum may so deflect the air current passing through the nose that, with its content of more or less irritating substances, it impinges upon the septum. This may lead ultimately to such changes in the mucous membrane that bleeding and even ulceration and perforation may result.

**Diagnosis.**—A septal deflection can usually be easily determined by simple inspection. It is well always to have a clear view of all the septum, and to this end it may be necessary to shrink the mucous membrane slightly. For such a purpose a whiff of ephedrine spray is effective. Adrenaline is only used where necessary, as the after-effects are sometimes annoying.

One must be prepared to recognize a hematoma, a septal abscess, or syphilis of the septum. A probe will help in the differentiation from these conditions. They are more or less soft and yielding or putty-like to the probe. In a simple deflection the mucous membrane is thin and the probe easily demonstrates the underlying cartilage or bone.

**Indications for Operation for Deflected Septum.**—These are qualified under *symptoms*:

1. Nasal obstruction.
2. Sinus infection.
3. Where necessary to reach parts of the nose otherwise inaccessible in operating.
4. Ear conditions.
5. Septal hemorrhage or ulceration.
6. Headache or reflex distant disturbances.

**Treatment.**—Submucous resection has gradually taken the place of all other measures for the correction of the deviated septum.

*Preparation of the Patient.*—Local anesthesia is usually employed. The less the routine habits of the patient are disturbed, the better. He is allowed a light breakfast. The temperature is taken to guard against existing acute disease. One hour before operation he receives hypodermically scopolamine gr.  $\frac{1}{150}$  and morphine sulphate gr.  $\frac{1}{4}$ , or less, according to weight. This causes a relaxation of an otherwise nervous individual. It makes him rather somnolent and comfortable, while in no way interfering with his co-operation. The ill-effects ascribed to scopolamine are not apparent at that dosage.

*Operation.*—The patient is placed on the operating table in a semi-recumbent position. This is the most favorable position for the patient. It is the natural position of rest. It is also easiest for the surgeon, as he stands in a comfortable, relaxed posture. It is well to have a low stool for changing the operator's elevation when desirable. A few whiffs of 10 per cent. cocaine are sparingly sprayed into the nose while the surgeon is scrubbing. This takes away the painful reaction of the mucous membrane and does much to make the procedure go smoothly. Placing of cotton applicators in an unanesthetized nose is extremely disagreeable, if not painful, to most people. The patient is draped and the instruments are prepared according to modern operating-room standards. Experience, afforded by thousands of cases, has shown that cleansing of the face or interior of the nose by antiseptics is not necessary. However, where conditions make it advisable, the parts to be touched are painted with a solution of iodine which is wiped off with alcohol. Where such precautions are taken, a square of gauze of four to six thicknesses is pasted to the lower edge of a strip of sterile adhesive, about 1 inch wide. This is then pasted across the upper lip and cheeks. It gives a sterile field and allows the patient to breathe comfortably.

A fine cotton applicator, with a small brush of cotton, is moistened in 1 : 1000 adrenaline. This is dipped into powdered cocaine. The excess is flicked off and the septum is carefully painted. Especial attention is given to the position occupied by the sphenopalatine nerve, coursing downward about the line of the upper border of the vomer and the anterior and posterior ethmoid branches high up in the nasal space. A similar applicator, bent slightly, is inserted into the region of the sphenopalatine ganglion. These applicators are left in place only a few seconds. All sensation in the septum is quickly abolished. A 1 per cent. novocaine solution, with 3 or 4 gtts. of 1 : 1000 adrenaline, is now injected with a very fine skin needle at the base of the septum in the vestibule; also with the same fine needle the skin and mucous membrane over the columella and anterior part of the septum are infiltrated. This is necessary where the incision is well forward or where a total luxation of the triangular cartilage makes its entire removal necessary. With a longer needle the mucous membrane is now raised over the region of the sphenopalatine nerve; one or two injections to each side. It is quite possible to operate with the cocaine alone, but the use of a small amount of novocaine, never more than 2 to 4 drams of a 1 per cent. solution, makes the operation absolutely painless, and need cause no anxiety.

As soon as the injection is finished, the incision is made. Any small-bladed, sharp knife will serve for this purpose. Those who are really ambidextrous, but there are few men equally clever with both hands, may make

the incision on either side. It seems simpler usually to make the incision on the side of the convexity. We make the incision with the right hand, on the left side of the patient's septum. The position of the incision and its length and form will vary in different cases. For instance, where the anterior edge of the triangular cartilage lies across the patient's right nostril, a forward placed incision is essential. On the other hand, where the deflections all lie fairly well back the incision may be placed more within the mucous membrane. When operating upon a septum that has an anterior perforation, the incision must be modified by the exigencies of the case. It can usually be made along the posterior edge of the perforation. It is well to cut slightly into the cartilage so as to insure the severance of the entire mucoperichondrium. The incision is not so deep in the lower part because troublesome bleeding is apt to occur there and is better met after the mucous membrane has been started from the cartilage. Precaution must, of course, be exercised to observe the principles of the operation and not cut through the mucous membrane on the opposite side. When making this incision con-

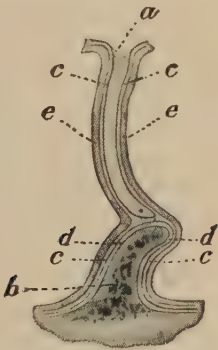


Fig. 53.—Section through the nasal septum: *a*, Quadrilateral cartilage; *b*, vomer; *c*, *c*, agglutination of the perichondrium to the periosteum; *d*, *d*, periosteum reflected over the crest of the vomer (it is not continuous within the perichondrium); *e*, *e*, mucoperichondrium. (Ballenger, "Diseases of the Nose, Throat, and Ear," Lea & Febiger, Philadelphia.)

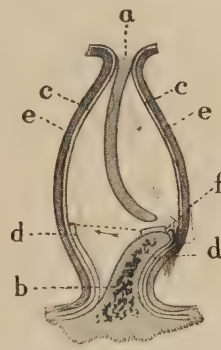


Fig. 54.—*a*, Cartilage; *b*, vomer; *c*, *c*, perichondrium; *d*, *d*, periosteum of the vomer; *e*, *e*, mucous membranes; *f*, two incisions through the periosteum along the crest of the vomer. On the concave side the periosteum over the vomer is elevated. (Ballenger: "Diseases of the Nose, Throat, and Ear," Lea & Febiger, Philadelphia.)

siderable care is necessary in starting to raise the mucous membrane from the cartilage. It must not be split, but must expose the shining cartilage. This is well accomplished by a scraping motion of the elevator. A fairly sharp Freer elevator serves very well here. After the mucous membrane is started, the incision may be carried down to the floor and the posterior edge of the line of the incision raised with the elevator. A duller elevator of the same type may now be used and the mucous membrane quickly raised from the upper part of the septum as far as necessary to expose the deflection. The mucous membrane over this upper part usually strips off easily with a satisfying crepitan sound. Sometimes the mucous membrane may be raised from the entire septum with ease. Usually, however, it is apt to be adherent along the suture lines at the upper edge of the vomer. The fibers from the periosteum pass through the septum to the opposite side along the upper border of the vomer. When such adhesions are reached, it often is possible to start low on the septum and raise the mucous membrane

below the crest well down to the floor of the nose. The adherent part is now carefully detached by cutting through with a sharp elevator or knife, working either from above or below. After the mucous membrane has been satisfactorily raised from the septum on the side of the incision, the cut is carried through the cartilage. Great care must be employed at this juncture to avoid cutting through the mucous membrane of the opposite side. It is well to check up carefully the position of the knife by watching through the opposite nostril. If the knife has gone too far and penetrated the mucous membrane, it may be removed and an incision again be made through the cartilage a few millimeters posteriorly. This is only allowable in case the position of the deflection warrants. This incision may be made in an oblique direction and with care the mucous membrane of the opposite side will not be injured. A fairly sharp elevator now follows the knife and the mucous membrane is raised. It is well to hug the septal cartilage when starting the mucous membrane and raise it only in one place backward for a distance sufficient to make certain that the elevator is next the cartilage. A circular movement then easily detaches the mucous membrane and it is worked off the septum, much as described upon the first side attacked. Where particular difficulties present, it may be advantageous to cut through the cartilage and elevate the mucous membrane for a short distance only on both sides. The stripped cartilage is then removed. Direct view thus obtained of both sides sometimes makes the task easier. One may thus move back gradually, working with both sides in view, taking out the deflection as one advances. It is not a matter of great concern if the mucous membrane be torn on one side or the other as long as the openings do not fall together. In a typical case, after the mucous membrane has been separated from the underlying cartilaginous and bony septum, the cartilage is first removed. The amount removed varies in the individual case and according to the method of the different operators. It is well to have good support at the anterior end and also at the roof. The necessities of the case govern this. All of the vertical plate of cartilage should never be removed. As much should be left in the upper part as consistent with thorough correction of the deflection. There is a tendency for the ridge of the nose to sink below the ends of the nasal bones where too much of the superior part of the cartilage has been removed. While such deformity may follow infection or abscess formation in the operative field, it is otherwise very rarely seen. To remove the cartilage the speculum of whatever type preferred is introduced with one blade upon either side of the cartilage. This exposes it perfectly. Where working further posteriorly, longer bladed specula may be used, for example, the Killian of different lengths. A nick is now made with the scissors and into this is fitted that ingenious invention of Ballenger—the swivel knife. With this as much or as little may be taken as desired. It is advantageous to bite out the higher part with the Jansen-Middleton cutting forceps. With these or other fairly narrow cutting forceps the vertical plate of the ethmoid is cut off from its upper attachment and then removed, either with these or other grasping forceps. The Luc forceps serve very well for removing the well-loosened bony parts. Ordinarily all of the deflection should be removed. It is well to examine the nasal cavity frequently and thus determine whether all obstructing irregularities have been corrected. Someone has said to take away a little more than seems necessary. However, by such inspection a definite measure of the amount

necessary to be removed can be obtained. Care must be given to the ethmoid region. It is here that often the greatest benefit from the operation comes. It is necessary, in some instances, to go quite high to accomplish this satisfactorily.

The cartilage and the upper part of the bony septum are ordinarily removed before attention is given to the nasal spine. Bleeding may occur here, so it is left for the last. This part, when obstructing, must be thoroughly removed or the result will be disappointing. The irregularities at the bottom may often be removed with straight forceps of various types. Where it is impossible to remove them thus, the chisel is called into play. Care must be observed that the lower parts of the mucous membrane are not adherent, as tearing may disturb an otherwise ideal operation. The chisel being applied by the operator at the desired point and in the proper direction to remove as much of the nasal spine as desired, it is tapped with a light mallet by an assistant. The mass, usually in one piece, is removed with small grasping forceps. It is sometimes necessary to cut a few adherent fibers with scissors or knife.

The operation in the majority of cases may be performed as outlined, but a word should be said about the correction of the luxated triangular cartilage, where the anterior edge presents in the vestibule. Some advocate the making of the incision well anteriorly over the protuberance. It seems much simpler to keep the incision near the junction of skin and mucous membrane, possibly somewhat more anteriorly than normal. The mucous membrane can be separated, working anteriorly from the incision and by a prying movement the anterior edge can be brought into the wound and as much as desired be removed. No harm results from the removal of the anterior part of the triangular cartilage. However, it adds to the support of the columella and should be retained where advisable.

From this point a variety of contingencies may modify the technic. Some operators sew up the incision in the mucous membrane. This is easily accomplished by the use of the one-piece needle and handle, or by the use of small curved needles held in small hemostats or needle-holders. It is ordinarily neither necessary nor advisable to sew up the wound. When an incision or tear has occurred through both flaps, where apposition would lead to ultimate perforation, the tear should be carefully repaired when possible.

It is good practice to cut the mucous membrane free well back along the lower attachment on the side of the incision, so that any subsequent bleeding will not be retained and lead to the formation of a hematoma. There may be sufficient tearing present to obviate the necessity of further drainage. Some cases with a roomy nose, a straight falling septum without bleeding, do very well without any packing whatever. Some men never pack the nose at all. This may lead to anxiety until the mucous membrane straightens out, which it fortunately usually does. Some use mechanical splints to hold the flaps of the mucous membrane together. However, in the majority of cases packing is advisable. It is best to protect the nose against the packing, when some form of gauze is used, by a piece of gutta-percha tissue, cut about 3 inches long by  $1\frac{1}{2}$  inches wide at the wider end and one side curved to 1 inch at the narrower end. This is easily introduced with the ordinary bayonet forceps. It is slipped in wet and can easily be fitted against the septum and also curved outward to protect the upper

part and floor and even the turbinates. Into this the  $\frac{3}{8}$ -inch gauze is packed. This amounts to about the same as the cigarette-drain used by many. It seems to have the advantage of being adaptable to the individual cavity and presents the disadvantage of requiring some practice in properly placing it. Both sides are so packed; a bit of adhesive is placed across the end of the nose to prevent the gauze working out.

The patient is taken directly to bed on an ambulance or wheel chair. The less he exerts himself, the less the possibility of bleeding. Most cases do bleed somewhat and sometimes an opiate may be necessary to control it. Usually, however, there is no bleeding.

The most unpleasant part of the whole proceeding is the subsequent stuffiness of the head and the mouth-breathing. Even when no packing is used, the swelling in the nose usually makes it temporarily useless for respiration. The packing is removed always early on the following morning, no matter at what time of the day the operation was done. The protected gauze almost falls out after it has become moistened with secretions. There may be a little bleeding for a few minutes, but usually it amounts to nothing. The patient should be kept at least another day in the hospital where ideal conditions are possible. Nothing further is done to the nose in the way of postoperative treatment. The patient is carefully watched to see that no complications arise. Sometimes a spray of 1 per cent. camphor-menthol or normal salt gives the patient relief.

**Submucous Resection Under General Anesthesia.**—It is sometimes necessary or advisable to operate under general narcosis. Ether is the safest anesthetic and is best preceded by nitrous oxide gas. The ether vapor is conducted by a tube to the patient as in a tonsil operation. The septum is thoroughly swabbed with adrenaline solution 1:1000, also 4 or 5 drops of that solution well diluted in normal salt solution is injected under the mucous membrane as the novocaine is injected, as described under Local Anesthesia. The operation is now carried out in the usual manner. There is apt to be more bleeding than where local anesthetic is used.

**Complications and Sequelæ of Submucous Resection.**—*Hemorrhage.*—Bleeding rarely gives serious trouble. It is seldom necessary to repack, or even to pack, where packing was not used at the time of operation. This subject will be discussed at length under *epistaxis*.

*Hematoma.*—The accumulation of blood between the layers of mucous membrane is always to be watched for and guarded against. It rarely occurs where the blood has been thoroughly cleaned out from between the mucous membrane flaps at the time of operation. The incision of the flap, cutting it free at its lower border as described, usually will prevent clot formation. Where the accumulation is minimal it may be ignored and will be absorbed without thickening. When the amount is such as to presage too much ultimate thickening of the septum, the incision must be opened and the clot curetted or wiped out with gauze strips. The nose may then be repacked if necessary. This necessity is of extremely rare occurrence.

*Acute Sinusitis.*—The obstruction of the nasal chambers, due to packing or swelling, may cause an acute exacerbation of a chronic sinus infection. This occurs very seldom, but where known infection in the sinuses is present, packing of the nose is to be avoided where possible, as described. Where such infection has occurred, drainage should be favored by keeping the nose as free from discharge and swelling as possible. It may be advis-

able to shrink the mucous membrane at intervals with ephedrine or cocaine adrenaline. There is usually a compensatory hyperemia following the use of these drugs, so that their employment depends upon their efficacy in the individual case.

*Chronic Sinus Infection.*—Attention has already been called to the importance of the obstruction in chronic sinus disease. It is often impossible to operate intranasally upon the sinuses because of an obstructing septum. Ordinarily it is best to attend to this septal deflection and then proceed at the same sitting with the sinus operation, where that has been deemed necessary.

It should be borne in mind, however, that a certain percentage of infected sinuses will get well without operation following the submucous resection of the septum because of the better aëration and drainage.

*Acute Follicular Tonsillitis.*—This is apt to occur following any nasal operation, especially at the time of the year when acute throat infections are prevalent. It is wise, where tonsillectomy is required as well as the nasal operation, to remove the tonsils first. Generally speaking, tonsillitis does not enter as a frequent complication.

*Infection or Abscess Formation.*—This shows itself ordinarily a few days after operation by the usual symptoms of acute wound infection. Pain, tenderness, temperature, leukocytosis, swelling, redness—all may or may not be present. Abscess usually follows upon the heels of a hematoma and is recognized by distention, swelling, and edema of the septum. The swelling is more or less symmetrical on the two sides and imparts to the examining probe a feeling of soft, elastic resistance. This is a serious complication because of the close relationship between the venous channels of the nose and the cranial cavity. The possibility of meningeal infection, though remote, is present.

The wound should be opened at once. All the infected material possible should be curetted or wiped out. A strip of gauze or rubber tissue drain should be inserted. The cavity should be under constant supervision and the drain should be changed as frequently as necessary to prevent retention.

Healing usually takes place without ultimate deformity. It is, however, the cause of most of the bad postoperative nasal depressions. The nose is not so strong after a submucous resection. However, this applies more to the movable than the bony part of the nose. The strength of the latter part of the structure is afforded by the arch formed by the nasal processes of the superior maxillary bones in conjunction with the nasal bones. Little support is given by the bony parts of the septum, which in many cases hardly extend forward under the nasal bones. The movable part of the nose is more apt to be caused to sink by a blow, because of the lack of septal support given by the triangular cartilage. It is always advisable to leave as much of the edge of the cartilage as consistent with proper operative result.

#### PERFORATIONS OF THE SEPTUM

**Congenital.**—Few cases are on record.

**Perforation Caused by Trauma.**—This occurs most often in the course of a submucous resection of the septum, as described. This perforation can often be repaired at once. The edge can be caught by a fine needle and the opening closed. Where it is too large for this to be done, the flap may be cut freely far above and slid over the opening as in plastic skin opera-

tions. Where the perforation has persisted after healing of the septum, it usually can be ignored. Sometimes where it is small and occupies a forward position, it may cause annoyance by the whistling of air in breathing.

**Spontaneous Perforation.**—Spontaneous perforation of the septum may take place as the result of what has been called a simple perforating ulceration. This perforation occurs on the septum in Kiesselbach's area and is usually the result of irritation from air currents or dust particles thrown against this area by some septal or nasal peculiarity. A bump of projecting septum at this point certainly encourages this irritation.

The protective faculty of the human organism applies the usual remedies for relief of the irritation. More blood is thrown to the part and the columnar ciliated epithelium loses its moist, velvety character and is replaced by the pavement type of epithelium. This is more dry and brittle and is apt to

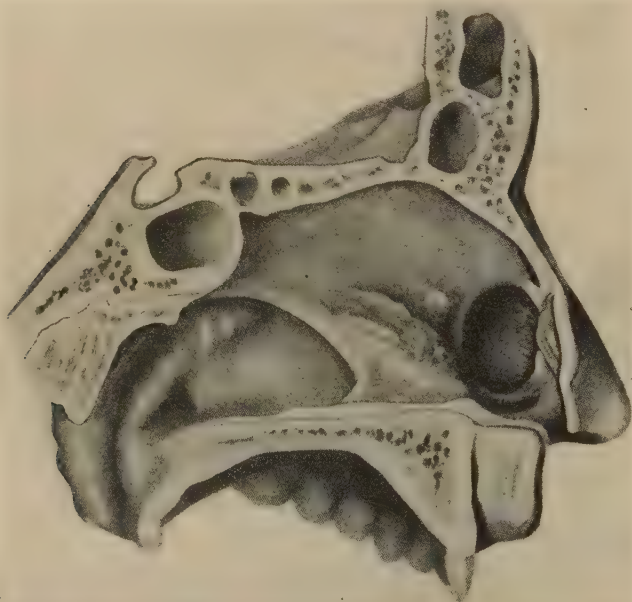


Fig. 55.—Syphilitic perforation of septum. (From Beck, "Applied Pathology in Diseases of the Nose," published by C. V. Mosby Co.)

crack, causing superficial ulcerations. Small crusts of mucus adhere, and the removal of these by the patient, if with the finger-nail, does not soothe the trouble. The vascularity of this already vascular region is increased as inflammation gradually progresses and hemorrhages are frequent. If allowed to progress, the slow, indolent ulceration includes the cartilage and ultimately leads to its perforation. Cartilage must be well nourished by its coverings, as it contains no blood-vessels. After perforation has taken place and has, to a large extent, occupied the area where the vicious circle started, the edges heal and it becomes less annoying. There may still be bleeding and crusting about its edge.

**Syphilitic Perforation of the Septum.**—Syphilitic perforation of the septum was formerly so frequent that every patient with a septal hiatus was the subject of medical suspicion. Modern medicine has changed that con-

dition until now perforation of the septum from that cause is comparatively rarely seen. Such perforation can usually be recognized because of its large size and irregular shape. It is wont to attack the bony part as well as the cartilage.

**Lupus and Tuberculosis.**—These diseases may lead to perforation of the septum. Lupus and tuberculosis are more apt to involve the cartilaginous parts of the septum.

It is hardly possible to make a diagnosis of the cause of these granulomatous types of perforation from the appearance or location alone. General examination of the patient and laboratory methods must be largely relied upon for diagnosis.

**Treatment.**—The simple perforating ulcer, or the antecedent inflammatory area, can sometimes be caused to heal by the regular application by the patient of a soothing ointment. Where this fails and where the condition is at an early stage, it may be healed by raising up the mucous membrane over the area inflamed as in a submucous resection, and inserting a bit of gauze under the mucous membrane flap for twenty-four to forty-eight hours. This causes the mucous membrane to take on new succulence. In some cases, when then allowed to heal by the removal of the gauze, the symptoms disappear. Where irregularities of the septum seem to be responsible for the overlying condition, a submucous resection is advisable. Care must be exercised in the choosing of such cases, as otherwise there will be disappointments. For the treatment of active syphilis, lupus, or tuberculosis the reader is referred to treatises on those subjects.

The perforation of the septum may present an operative problem for the rhinologist. Small perforations, fortunately, are the most annoying to the patient. The large ones give little more than mental disturbances. The small ones can always be converted into large ones where it is inadvisable or impossible to close them. The surgical closure of the perforation may be obtained by one of the various plastic devices for shifting the mucous membrane.

#### EPISTAXIS

Bleeding from the nose may be caused by:

##### 1. Trauma.

2. **Disturbances in Kiesselbach's Area.**—Nearly all spontaneous hemorrhages from the nose arise from bleeding vessels in the anterior lower part of the septum just inside the vestibule, known as Kiesselbach's area. Here the septal arteries abetted by terminal branches from the superior coronary artery form a plexus. These vessels are prone to bleed when the mucous membrane covering them becomes irritated. This process has been described under Simple Perforating Ulcer of the Septum.

##### 3. Malignant Neoplasms.

—These do not often arise from the septum.

4. **Angioma of the Septum.**—This tumor, the size of a pea or larger, has its attachment to the cartilaginous septum. It is usually of a reddish color with smooth, slightly lobulated mucous covering. It varies materially in its histological structure, so that various names are given it according to the predominating elements. It is remarkable for its vascularity and is possibly caused by the irritation to which this part of the septum is subject. It occurs more often on the left side and more frequently in women than in men. It gives rise to nasal obstruction when large enough, and has a tendency to bleed frequently.

It should be removed with a snare or knife and the base cauterized to control the hemorrhage.

**5. General Diseases.**—Bleeding induced by blood or vascular changes is a frequent symptom in the following conditions: Hemophilia, scurvy, arteriosclerosis, kidney disease, the anemias, the infectious diseases, phosphorus poisoning, acute yellow liver atrophy, vicarious menstruation, etc.

**Diagnosis.**—The location of the bleeding vessel or vessels is not always easy to determine.

*Where due to trauma*, operative or otherwise, the bleeding may come from any part injured. It is not then usually practicable to find the bleeding vessel. The hemorrhage, in such instances, is usually controlled by packing.

The author once had the unusual experience of a hemorrhage from an internal maxillary, where it enters the sphenopalatine foramen and gives off the nasal vessels. This hemorrhage followed the insertion of a needle from without, by a general surgeon, for the injection of alcohol into the sphenopalatine ganglion. The bleeding vessel was cauterized from within the nose.

*Spontaneous Bleeding.*—The blood usually comes from Kiesselbach's area in most cases and the bleeding point is easily seen.

**Treatment.**—*Spontaneous Nasal Hemorrhage.*—The bleeding, as already stated under the section on Perforating Ulcer of the Septum, usually comes from Kiesselbach's area. The region may be well forward and low on the septum. In some instances mere pressure with the thumb and fingers upon the collapsible part of the nose may bring pressure against the bleeding point. Otherwise cocaine and adrenaline are used and the bleeding point found. It either shows as a source of the hemorrhage or, when the bleeding stops, the enlarged vessels stand out clearly against the blanched septum. A cautery of some kind, applied under direct vision, usually easily controls the bleeding. Chromic acid is a most useful cauterizing agent for this purpose. It is used in the following manner: A silver probe is slightly heated over the burner and dipped into the chromic crystals. Some of these will adhere, which heated slowly over the flame will melt and form a bead at the end of the probe. This touched to the bleeding vessel, even when the blood is flowing freely, will usually stop the hemorrhage. The excess of chromic acid is washed off with water on a cotton swab. It is rarely necessary or advisable to pack the nose for this type of hemorrhage. Rarely spontaneous recurrent hemorrhage comes from some other part of the nasal cavity than the anterior part of the septum. It is best to refrain from packing in such cases if possible. The nose should be repeatedly searched anteriorly and with the nasopharyngeal mirror for the source of the bleeding.

*Postoperative Hemorrhage.*—This type of hemorrhage or bleeding, whose source cannot be determined, often requires more radical measures to control it. Morphine, where not contraindicated, is always used in troublesome hemorrhage. Where it does not stop the bleeding, it quiets the patient and makes the packing, where that is necessary, less painful. Cocaine does not anesthetize the nose well in the face of profuse hemorrhage and adrenaline also is not efficient. Where the location is not determined and the hemorrhage must be stopped, the bleeding side of the nose should be carefully and firmly packed with gauze, narrow strips  $\frac{1}{4}$  to  $\frac{3}{8}$  inch in width are

best. Where, after packing and possibly repacking more thoroughly, the hemorrhage continues, or where after packing anteriorly the blood runs down the pharynx, it may be necessary to insert also the postnasal plug.

Where, therefore, the hemorrhage warrants its employment the following technic is used: A small, soft-rubber catheter is passed through the nose on the bleeding side and the end grasped by curved artery clamps in the pharynx and brought out through the mouth. A string is tied to the end of the catheter which is withdrawn through the nose, pulling the string with it. About the center of a sterile sea-sponge is tied three strings. This sponge is about 3 inches long by about  $1\frac{1}{2}$  inches thick. It has been previously wet and wrung dry. Two of these strings are now fastened to the string hanging out of the bleeding patient's mouth and the strings are then drawn out through the nose from behind forward. By pulling firmly on these two strings and guiding with a finger in the throat, the sponge is drawn into the nasopharynx. The third string follows the sponge and hangs out of the mouth and is only for convenience in removing the sponge. Where the bleeding is from both sides, two sponges of smaller size may be put into the nasopharynx in this way, or one large one will often suffice for both sides. The strings are separated at the anterior naris and the nose is firmly packed with the narrow gauze strips. The strings are then brought over the packing where it protrudes from the nose and firmly tied. Where more pressure is needed, more packing may be added and the pressure on the strings will bring the nasal packing more snugly against the postnasal plug. Gauze may be used in the nasopharynx instead of sea-sponge if preferred.

Clots sometimes interfere with the efficiency of packing. A clot between the folds of the septum may require removal before the pressure can be brought directly against the bleeding vessel.

Packing should never be left in the nose longer than absolutely necessary. The possible ill-effect upon the ears and the sinuses must be kept in mind. It is usually removed on the morning following its insertion. Circumstances may, of course, modify the rule.

#### HEMATOMA OF THE SEPTUM

Hematoma of the septum is practically always caused by traumatism. It has been described under Submucous Resection of the Septum as a complication to be guarded against. Where it follows a blow on the nose, there is usually a fracture of the septum itself which allows the accumulated blood to communicate from one side to the other. Hematoma of the septum is therefore usually bilateral. After the patient has received the blow, there is usually nosebleed; after this has ceased he complains of stoppage of the nose.

**Diagnosis.**—Inspection shows a double-sided swelling of the septum covered by red and swollen mucous membrane. The tumor is soft to the touch of the probe and movement is sometimes transmitted through the tear in the septum to the opposite side.

**Treatment.**—Inasmuch as infection usually takes place either by way of the blood-stream or a tear in the mucous membrane, it is advisable always to incise and clean out the clot. An early incision on one side may suffice, but it may be necessary to open on both sides.

## ABSCESS OF THE SEPTUM

This usually follows hematoma of the septum as described. It is differentiated from simple hematoma by symptoms of pain, fever, and tenderness. Otherwise the diagnosis is made as in hematoma of the septum. It should be taken seriously and be treated efficiently.

**Treatment.**—The mucous membrane should be incised freely on one or both sides as required. The infected material should be removed as well as possible and a small drain of rubber or gauze should be inserted. This should be frequently changed and continued in place until the symptoms of infection subside.

There may be necrosis of some of the cartilage denuded of the perichondrium. This must be removed.

A characteristic depression of the soft ridge of the nose may rarely follow such an abscess. Ordinarily prompt treatment averts such a contingency. The relationship between the veins of the nose and those within the cranial cavity accentuates the necessity for proper treatment of the abscess.

Dr. Frank E. Blaisdell has written the section on Embryology and wishes due credit to be given Schaeffer, Keibel and Mall, McMurrich, and Heisler.

EDWARD CECIL SEWALL.

## INFLAMMATORY AFFECTIONS OF FRONTAL, ETHMOID, AND SPHENOID SINUSES

**Etiology.**—The inflammatory affections in this region are practically all of bacterial origin. Traumatism, such as foreign bodies, bullet wounds, or fractures, may produce irritation, but unless complicated by a superimposed infection the inflammation is quite transient. The bacteria most commonly found are classified by different investigators in their order of frequency. The most recent classification is given by Babcock<sup>1</sup> from a series of 177 cases, both acute and chronic, as follows:

<i>Acute Cases</i>			
<i>Pneumococcus:</i>			
Type not determined.....	5		
Group II.....	9		
Group III.....	3		
Group IV.....	15	Total	32
<i>Streptococcus:</i>			
Hæmolytic.....	3		
Non-hæmolytic.....	2	Total	5
<i>Staphylococcus:</i>			
Aureus.....	13		
Albus.....	17	Total	30
B. Influenza.....	4		
M. Catarrhalis.....	2		
A. Diphtheroid bacillus.....	2		
B. Coli communis.....	2		
B. Fæcalis alkaligenes.....	3		
B. Aureus.....	3		
B. Proteus.....	1		
M. Tetrageus.....	3		
B. Subtilis.....	1		
No growth.....	1		

*Chronic Cases*

<b>Pneumococcus:</b>			
Type not determined.....	1		
Group II.....	1		
Group IV.....	4	Total	6
<b>Streptococcus:</b>			
Hæmolytic.....	15		
Non-hæmolytic.....	4	Total	19
<b>Staphylococcus:</b>			
Aureus.....	18		
Albus.....	20	Total	38
B. Influenza.....	2		
B. Mucosus capsulatus.....	5		
A. Diphtheroid bacillus.....	4		
B. Coli communis.....	2		
B. Fæcalis alkaligenes.....	3		
B. Aureus.....	3		
B. Proteus.....	1		
M. Tetragenus.....	3		
B. Subtilis.....	1		
No growth.....	1		

The bacillus of Friedländer though not included in the above list is occasionally found either with other organisms or in pure culture.

The normal nasal chambers contain bacteria at all times, but the normal membrane is able to withstand invasion till some influence affects the local resisting agencies or lowers the general resistance of the individual. Likewise, the air continually drawn through the nostrils contains a certain number of organisms, yet they produce no effect till some subsidiary cause becomes operative to lower the individual's resistance. Though nearly all the infections of the sinuses are air-borne an infection may also occur through the circulation as shown by Killian<sup>2</sup> in scarlet fever cases.

The predisposing causes to infection may then be considered as general and local. Of general causes the most commonly mentioned is exposure to cold. Fatigue as a temporary condition from overwork or overplay is a potent factor in lowering individual resistance. General exhaustion brought about by poor food, poor surroundings, and worry also markedly reduce resisting powers. Various toxemias from intestinal absorption or pus foci anywhere in the body, syphilis and tuberculosis, and, in fact, any influence which produces a deterioration of health, render the subject a liable prey to the ever-present, mischief-making bacteria.

The local causes predisposing to infection of this region are: Anything interfering with the free ventilation of the nasal chambers; congenitally narrow nasal passages or passages ill-developed due to persistent obstruction from neglected adenoids in childhood; injuries which have produced deviations and spurs on the septum; enlarged turbinates either from repeated acute nasal infection or from pollen and protein sensitization; infected foci in the neighborhood, such as teeth and tonsils. Foreign bodies and local traumatism may produce changes in the membrane conducive to the initiation of an infection. Dryness of the membrane usually due to living in overheated rooms renders the membrane more vulnerable to infection.

**ACUTE INFECTION OF SINUSES**

**Period of Incubation.**—This varies according to the infecting organism, but is usually two or three days. The pyogenic organisms can produce symptoms very quickly. The writer remembers seeing a scalp wound full

of pus before it was twenty-four hours old. Where nasal infections can be traced to contagion from one person to another the incubation is rarely more than three or four days and sometimes only one day.

**Onset.**—The onset of an acute infection in the sinuses is very rarely different from that described in acute coryza or acute rhinitis, and the differentiation of the two conditions is now largely recognized as a distinction without a difference. The attempt to define exactly that differentiation is more or less of a sophistry, the severe cold being called a sinusitis and the mild one a coryza. The symptoms vary greatly according to the virulence of the infection and the resistance of the individual. Also the amount of discomfort depends considerably on whether the individual has spacious or contracted nasal chambers. Thus, one may often observe that in patients who have had an exenteration of ethmoids and the antra opened, an acute nasal infection gives very little local discomfort. Even though they may have a very profuse discharge such patients are often unaware of the onset because of the absence of the usual symptoms.

The usual symptoms when established are nasal obstruction with sneezing and a profuse watery discharge. A severe reaction in the mucous membrane blocks the sinus opening and headache ensues. If these openings remain patent there may be no headache throughout the course of the disease and the infection will probably be more quickly overcome. The pathological condition in the nasal membrane at this time is dilated vessels in the submucosa with free escape of serum and leukocytes, the serum escaping to the surface between epithelial cells. The irritation of the mucous glands stimulates them to greatly increased activity with a free outpouring of mucus. The phenomenon is most marked in the thick membrane over the turbinates. There is not nearly as much exudate from the membrane within the sinuses, as this membrane contains fewer glands. As the discharge becomes purulent the swollen membrane gradually subsides, the dilated vessels regain their tone, and absorption of the exudate in the submucosa goes on till resolution is complete both in the nasal mucous membrane and the sinuses. The duration of the purulent stage in a typical case will be only about a week, when resolution will begin and the discharge will change to mucopurulent, then to clear mucus, this finally decreasing till reduced to the normal amount—the process entirely subsiding in the second week.

The general symptoms occurring in the course of such an infection are so well known that they need scarcely be mentioned. There is usually some fever, varying from 100° to 102° F., with a pulse-rate of 90 to 100. The amount of prostration when all the sinuses are involved may be quite extreme. Night-sweats may occur and profuse perspiration on exertion. The local symptoms of nasal obstruction with fulness in the head and headache are very distressing and render the victim entirely incapable of any concentration, and in many cases there is interference with the ocular muscle, producing eye-strain to aggravate the headache.

**Untoward Symptoms.**—*High Temperature up to 105° F.*—This denotes a very severe infection and usually signifies a blocked sphenoid or the beginning of some intracranial complication.

*Chills* may occur at the onset or during the course of the disease—most often seen from obstructed drainage in a sphenoid.

*Severe Headache.*—This may occur over the frontal region, over the vertex, or in the occiput. It may be so severe as to require opiates for its

relief. As a rule, after the first two or three days the congestion subsides, free discharge is established, and the pain is relieved. In severe cases, however, the intensity of the pain continues and may last for a week or ten days, and be so severe as to simulate a beginning meningitis, especially when a sphenoid becomes blocked. The pain in these cases is usually occipital and, with such associated symptoms as retraction of the head and vomiting, may simulate a basilar meningitis. This pain may be associated with marked tenderness on pressure just posterior to the mastoid process. Severe pain in the frontal sinus is usually associated with tenderness over the frontal region.

*External Swelling.*—This is most common over the frontal sinus. It is due in most acute cases to a periostitis, and subsides when intranasal drainage is established. In acute exacerbations of an old chronic condition the swelling may be due to actual rupture through the diseased anterior wall or floor of the sinus. This is a more pronounced swelling with great edema of the eyelid and some displacement of the eye outward and forward. The external swelling from anterior ethmoids appears at the inner canthus, and may often be a rupture of a distended cell through the thin lacrimal bone. This may occur in primary acute cases. Rupture of an ethmoid into the orbit may occur in primary acute cases, but is more often due to an acute condition grafted on an old chronic ethmoiditis. This will produce marked displacement of the eye with swelling of both lids and sometimes marked chemosis.

*Dizziness.*—This is not unusual during a severe acute attack, and is possibly due to interference with the proper action of ocular muscles, or there may be some concomitant labyrinth irritation from the same infection or from toxins absorbed from the sinuses.

*Vomiting* may occur in children, but is very rarely seen in adults in an uncomplicated case of sinusitis.

**Complications.**—*Meningitis.*—This is an extremely rare complication of primary acute sinusitis. It occurs in the acute fulminating type associated with acute osteitis in children and may occur from an acute exacerbation of a chronic condition. In these cases, however, there is probably marked bony disease already present.

*Thrombosis of Cavernous Sinus.*—This has been described as following a primary acute sinusitis. I have seen it several times from a secondary acute condition, but have never seen it as a complication of a primary acute condition.

*Retrobulbar Neuritis.*—This is more apt to be a complication of subacute and chronic conditions, but it may first appear during the presence of an acute attack and recover as the acute attack subsides. I can recall 2 cases showing this phenomenon. Neither required an operation and there have been no symptoms since.

*Ocular Palsies.*—These also are more liable to occur in subacute and chronic conditions, but may be initiated with an acute flare-up on top of residual infection.

**Treatment.**—For better classification of treatment it is advisable to consider three stages:

*The Stage of Onset.*—The treatment in this stage should be almost entirely general. The patient should be put to bed at once and given a cathartic, such as calomel or castor oil. This eliminates toxins from the in-

testinal tract, and renders the patient the better able to fight an infection. Something to promote similar free action of the kidneys should also be given, such as plenty of water, or lemonade without whisky. The administration of Dover's powder 3 gr., aspirin 3 gr., phenacetin 3 gr., and camphor  $\frac{1}{2}$  gr. will add greatly to the comfort of the patient and will tend to reduce the congestion of the mucous membrane, thus tending to prevent the vicious cycle of an early obstruction of the sinus outlets.

*During the first forty-eight hours* "acute noses" should be labeled "Hands Off." Most all local treatment during this stage is irritating and harmful except warm soothing inhalations of hot steam to which may be added tincture of benzoin comp., 1 dram to the pint, or spirits of camphor,  $\frac{1}{2}$  dram to the pint. Wool impregnated with camphor and menthol placed in the nostrils gives some comfort and makes the patient feel that he is doing something to help kill the germs. The instillation of warm argyrol has been recommended, but is usually slightly irritating to the membrane. One of the salts of ephedrine dissolved in a bland oil sprayed in the nose will relieve the congestion and promote sinus drainage. This may be repeated every three or four hours.

*The Stage of Purulent Discharge.*—Local treatment of this stage is very important. It not only greatly accelerates recovery, but is almost certain to prevent the case from gradually drifting into a chronic condition. If the nasal chambers are badly blocked a little 4 per cent. cocaine may be applied either with an applicator or spray. Ephedrine hydrochloride or sulphate solution used in the same way will be just as effective and is attended by less subsequent reaction. After waiting a few minutes a warm saline irrigation can be given. This can be carried out very well by means of a suction irrigator, attaching a suction tube in one nostril and an irrigating tube in the other. This clears out all the secretion and the warm saline stimulates the ciliary action of the epithelium. After thoroughly cleansing the nose  $\frac{1}{2}$  dram of 10 per cent. argyrol or neosilvol may be put into the nasal chambers. Probably the best way to administer this is by the postural method. Gravity will carry further into the recesses of the nose and sinuses than a spray or applicator. In cases not treated till relief is sought on account of profuse purulent discharge the same local treatment should be continued till the discharge has ceased.

Surgical measures are rarely indicated in acute conditions of the ethmoid, sphenoid, and frontal. Occasionally, in a nose badly blocked, an excision of the anterior end of the middle turbinate or a limited submucous resection of the septum may be necessary if pain shows no remission at the end of a week. Likewise, a sphenoid may have to be punctured and washed out where pain and toxemia are severe. The possibility of doing this through the natural opening is rather remote in an "acute nose," but it can be punctured with a long antrum trocar, the curved end pointing downward and inward. This is less traumatic than a long attempt to enter the natural opening with a blunt cannula through swollen mucous membrane. Rupture of ethmoid cells either into the orbit or anteriorly under the skin at the inner canthus may call for an external incision to establish drainage, and at the same time a drainage communication should be established into the nose. It is a surgical axiom which holds for anywhere in the body that surgical interference in an acute purulent process should be as conservative as possible, but enough must be done to provide free exit for the pus. The

presence of external swelling over the frontal, even with considerable edema of the eyelid or swelling above the inner canthus, where there is no pus manifest beneath the skin, does not always call for an external incision; in fact, it very rarely is necessary. Intranasal drainage should be promoted in every way. Shrinking up the nose with cocaine or ephedrine solution several times a day and applying suction will help a great deal, and as intranasal drainage becomes established the external swelling will disappear. Many of these cases which I have observed and treated in this way have not had any recurrence of trouble during a period of eight or ten years. I had formerly thought that a radical operation on the sinuses would be necessary later on, but experience has not justified this assumption. I have seen cases which have ruptured into the orbit clear up through intranasal drainage, but I think this condition calls for surgical interference in nearly all cases.

### CHRONIC INFLAMMATION OF ETHMOID, SPHENOID, AND FRONTAL SINUSES

**Causation.**—The same general and local causes are operative in producing a chronic condition which have been enumerated as causative factors in producing acute conditions, and practically all chronic cases have evolved from either a severe acute attack which has not been treated or from frequent acute attacks. It occasionally happens that one finds a chronic process in the sinuses which gives no history of any acute attack to mark especially the onset, just as one may find a chronic infection in the tonsil with no history of an acute tonsillitis. Sinuses can sometimes be infected during a severe attack of tonsillitis where the tonsillar infection produced the dominating symptoms at the onset. Likewise, an antrum infected from a tooth may spread the infection to other sinuses with no very acute symptoms marking the onset.

The greatest number of chronic conditions are found in people with bad anatomical noses. This not only predisposes to acute attacks, but renders the field of infection much less accessible and, therefore, less amenable to treatment. Neglecting to take proper treatment during acute attacks or afterward is responsible for many chronic cases. It is surprising how unconcerned many people are in regard to the persistence of a purulent nasal discharge and almost regard "a little catarrh," as they call it, an inevitable incident of their local climate. Physicians have not in the past sufficiently warned their patients of the evil consequences which may ensue from such carelessness. Chronic sinus conditions can produce as much disability as a limited tuberculous lesion in a lung, and many a person has gone through life as a chronic invalid, labeled a neurasthenic or hypochondriac from an undiscovered pus focus in the nasal sinuses. The incidence of contagion has never been sufficiently emphasized. It is quite common to find the condition in several members of the same family who are so afflicted, and nearly all show the same manifestations. One can easily observe, also, that in a special hospital a great many of the house surgeons and nurses become infected.

**Pathology.**—The pathological changes may be limited to the soft tissues, or both the mucous membrane and the bony walls of the cavities may be affected. In the soft tissue the result of a long-continued irritation is to produce degeneration of the more highly specialized functioning part of the membrane and its replacement by fibrous tissue. If the irritant is

sufficiently low grade the hyperplastic or hypertrophic changes may prevail during the patient's lifetime, but if the irritation is more severe there is more tissue destruction and exudation, producing ulceration and granulation tissue with eventually polypoid degeneration or fibrous and atrophic changes. In time the ciliated epithelium becomes changed to stratified, the fibrous tissue in the submucosa cuts off the blood-supply, and the mucous glands atrophy, causing deficiency of mucus. Hence, the pus secreted is no longer washed away and dries in the form of crusts, producing *ozena*.

**Changes in Bone.**—The results of an inflammatory process in bone are caries necrosis and sclerosis, and these results depend upon the density of the bone and the severity of the inflammation. Thus, in cancellous or cellular bone like the ethmoid, caries and sclerosis are the usual changes, while in dense, compact bone necrosis is more usual. Carious changes in the ethmoids are not uncommon, leading to destruction of the intercellular septa with the formation of granulation tissue and polypi. The frontal and sphenoid walls may also be subject to carious changes or may show necrosis of small areas. All these sinuses may undergo sclerotic changes in their bony walls from a prolonged chronic irritation, the sclerotic change being so marked sometimes as nearly to obliterate the sinus cavities. It may go on as a more or less even thickening of the sinus walls or may occur as uneven masses of ossified granulation tissue producing *exostosis*. Such conditions have been found both in frontals and ethmoids.

Polypi are usually found associated with an ethmoiditis. They are of inflammatory origin rather than a new growth, and are produced by degenerative changes in the mucous membrane—the result of a chronic irritant. They are most frequently associated with a condition of the ethmoidal labyrinth in which the bony walls of the cells are carious. Whether this condition of the bone is a cause or result of the polypi has been much discussed, but the most general belief today is that there is carious bone as a preliminary change to the formation of polypi.

**Symptoms.**—*General.*—There are few ills which produce a state of health called partial disability that render their victim more depressed and miserable. A person half as wretched with tuberculosis would be placed in a sanatorium. Sometimes the depression amounts to real melancholia, but it usually manifests itself in a lack of ambition and power to concentrate. A dull headache or feeling of heaviness is present in most cases part or the whole of the day. Changes in the weather affect the condition, patients being more miserable in high humidity. If there is free discharge the constant use of handkerchiefs is a great annoyance especially to the cleanly sensitive individual. Chronic sinusitis is responsible often for the state of general ill-health called *neurasthenia*, and many suffer from exhaustion and fatigue similar to that found in tuberculous patients.

Fever is more often present than is suspected. Patients when placed on a four-hour temperature chart often show a daily variation in temperature amounting to 1 or 2 degrees. It is usually of a nondescript character depending somewhat on the drainage conditions. In many cases the slight elevation will appear in the morning and be normal or subnormal in the evening.

*Local.*—**Nose:** A chronic discharge either purulent or mucoid is usually present, and there may be considerable obstruction to breathing from congested, swollen turbinates. This is apt to be a rather intermittent obstruc-

tion, usually worse at night. Sneezing is a fairly common symptom, severe paroxysms occurring in the morning. This is most common with involvement of anterior ethmoids especially if there is defective drainage of one or more cells.

**NASOPHARYNX:** Granulations on the pharyngeal wall are usually present due to the irritation of the purulent discharge. These may cause continued sore throat and cough.

**LARYNX:** Irritation of the larynx with chronic laryngitis and irritation of the lingual adenoid region are very common.

**BRONCHI:** An associated bronchitis is often present, but many cases have severe spasmodic cough. This is especially common with cases which have pharyngeal or lingual adenoid irritation from the secretion. Irritation from the sinuses themselves can cause a reflex cough which is often troublesome. Some cases of asthma seem to come under this category. Pneumonia<sup>3</sup> may be secondary to an infection of the nasal sinuses.

**EYES:** Some amount of muscular asthenia is often present, and the pain from a sphenoid is often described as being back of the eyes. Patients frequently complain of soreness of the eyeballs.

**Digestive Disturbance:** Loss of appetite and indigestion, sometimes with nausea and vomiting, may be due to swallowing considerable quantities of pus.

**Headache:** This is one of the most common symptoms of chronic sinusitis and accounts for the major portion of head neuralgias. Its localization is variable. Over the frontal it may be due to infection of the frontal, or pain from the sphenoid may be transmitted to this region through the ophthalmic division of the fifth nerve. Pain over the vertex usually denotes ethmoiditis, but in this region a feeling of fullness or pressure is more often complained of than actual pain. Pain in the occiput, back of the neck, even going down to the shoulder may be associated with disease of the sphenoid. The headache from sinusitis is usually intermittent, very often worse in the morning, being relieved as the patient goes about and the drainage becomes freer. Most sinus headaches are made worse by alcoholic stimulants and the "morning after" usually brings with it a prostrating headache. Some of the cases with severe headache have almost no discharge.

**Diagnosis.**—The diagnosis of chronic infection of the sinuses is often a matter of great difficulty. A goodly number show symptoms which are very apparent. A chronic discharge is always indicative of trouble in the sinuses, and in these cases all that concerns the diagnostician is to determine whether one or two or all of the cavities are involved. There is, unfortunately, a large group which has very few apparent or objective symptoms, which, nevertheless, may be the primary focus to some serious secondary lesion somewhere else in the body. The importance of a thorough history of the case cannot be overestimated. The inquiry should seek the records of past general illnesses, such as influenza, scarlet fever, measles, and pneumonia, and of any obscure illness which was not definitely diagnosed. Repeated colds and frequent headaches at any period of life may have some bearing on the evidence. The most usual history, of course, is that of a severe head cold which did not clear up satisfactorily.

Having elicited all the facts which may possibly have any connection with a sinus infection the next step is the direct examination of the nasal chambers. The anatomy of the nose should be observed first, as it is strong

presumptive evidence that a nose with a marked deviation of the septum and enlarged turbinates, causing poor ventilation and drainage, may be holding an infection. The mucous membrane should be shrunk up with a 5 per cent. solution of cocaine applied on an applicator. A spray may be used for the same purpose, but should be used sparingly and the head held slightly forward after using. On the first examination of a patient a few minutes should be allowed after the first small application of cocaine to make certain that the patient possesses no idiosyncrasy to the drug. If there is no evidence of this, more liberal quantities may be used till the membrane is thoroughly shrunk. If there is no evidence of any secretion a little suction should be applied and the examination repeated. Also it is wise to have the patient incline the head downward for a minute or two and look in the nose again. The pharynx and nasopharynx should be carefully inspected. The presence of granulations on the pharyngeal wall is usually due to an irritating discharge which escapes from the posterior sinuses. The nasopharyngoscope should be passed through each nostril and the posterior portion of the nasal chambers and nasopharynx inspected. Transillumination may be used for the frontal sinus and occasionally may show the size of the sinuses, but I have never found any evidence of pathology obtained in this way to be worth anything. After all this procedure, if no pus has been found in any part of the nose, it is advisable to wash out the nose with a suction irrigation and again apply suction. If this fails to reveal any positive evidence ask the patient to come back in three or four days and go through the same performance again.

If, on the other hand, pus is found free in the nasal chambers, proceed to determine just what sinuses are infected. If pus is found posteriorly above the middle turbinate it must come from the posterior ethmoids or sphenoid. If found in the middle meatus it may come from anterior ethmoids, frontal or maxillary. If now the maxillary is washed out and pus still appears in this region, it must come from anterior ethmoids or frontal. In some cases it may be possible to wash out the frontal, and if pus appears after this it comes from the anterior ethmoids. In the posterior region the Holmes nasopharyngoscope may enable one to determine which sinuses are affected.

Failing to find any pus after repeated examinations, it is not yet possible to give the sinuses a clear bill of health. A thickened polypoid mucous membrane and granulations within the cells can be present in old cases without any secretion, and this condition can act as a focus of absorption. Atrophic changes denote an infection of the sinus which has occurred in early life. The membrane of the nose in these cases will be dry and somewhat atrophic and will usually give a history of having had discharge earlier in life.

The appearance of the nose in hyperplastic sinusitis is usually quite characteristic. There is swollen, congested mucous membrane, rather moist in appearance, and the history of a variable, intermittent obstruction, often with sneezing, will be given. Usually there is some mucoid discharge.

The presence of polypi is considered by many to be certain evidence of an ethmoiditis.

**Roentgen Ray in Diagnosis.**—The Roentgen ray has become an invaluable aid in the diagnosis of obscure sinus infection. Its chief field of

usefulness is in the diagnosis of frontal and ethmoid conditions. The evidence obtained by Roentgen ray of the sphenoid is not so reliable, but it does give a good definition of the anatomy which is a great help in operating. If a large sphenoid sinus is shown on the plate with a deep cavity extending far into the body of the sphenoid bone, with such anatomy the drainage is difficult, and one may be warranted in suspecting that it is infected provided the ethmoids and frontals show distinct signs of infection. There may be a tendency to count too much on Roentgen-ray evidence and not give sufficient time to acquiring clinical data, and it should, therefore, be remembered that it is the proper correlation of all the data and the proper balancing of this evidence which make for accuracy in diagnosis.

**Complications.**—*Intracranial Thrombosis of Cavernous Sinuses.*—This may occur in acute and chronic sinusitis, but is most commonly observed in acute exacerbation of chronic conditions. The initial symptoms are usually an increase in temperature, not usually extreme, but which may go to 104° F. Chills are not common nor are the usual septic phenomena present as in thrombosis of the lateral sinus. There is increasing swelling of the eyelids and some loss of movement in the eyeball. Chemosis in upper and lower lids occurs early. Later on there is paralysis of the eye muscles with exophthalmos and dilatation of superficial veins tributary to the sinus. Death occurs usually within a week or ten days from meningitis. This supervenes before septic phenomena from the broken-down thrombus can occur. The phenomena of cavernous sinus thrombosis are sometimes confused in early diagnosis with an extension of suppuration into the orbit. In the latter case, however, there is not usually chemosis in the lower lid. The ophthalmoscopic examination will be a great help in diagnosis, as in thrombosis there is early engorgement of the retinal veins with failing vision passing on to complete blindness. The thrombosis frequently extends to the other side with the same train of symptoms. Thrombosis of the longitudinal sinus from suppuration in the frontal sinus has been observed by Killian.<sup>4</sup>

*Meningitis.*—This may occur as a complication of infection in either frontal, ethmoid, or sphenoid, but most commonly from the sphenoid. Sir St. Clair Thomson<sup>5</sup> reported a case of sphenoid disease associated with meningitis in which postmortem showed such a causal relation, and gave notes of 40 other cases. Probably in the majority of cases the infection travels through the venous channels, as the lesion found about an area of necrosis is usually a subdural abscess. The terminal stage of a subdural abscess, or a brain abscess which was undiagnosed, is usually a meningitis. It is also a sequel to sinus thrombosis. A condition simulating a basilar meningitis can occur from a severe infection in the sphenoid with blocked drainage, which clears up promptly when drainage is established. A rapidly fatal meningitis occurs in the acute sinus cases where an acute osteitis is the main pathological lesion.

*Subdural or Brain Abscess.*—This may occur by direct extension through the bone and is usually subdural at first, or it may occur within the brain substance from extension by lymphatics, though these cases are comparatively rare. Abscess in the frontal lobe may arise from disease in the ethmoids, but more commonly from necrosis in the posterior wall of the frontal. Necrosis of the posterior wall of the sphenoid with a basilar abscess may occur. I have seen such a condition in the cadaver, but

no clinical history could be obtained. Two cases in the writer's experience, with bony necrosis in the posterior ethmoids and sphenoid, have died suddenly and the rupture of a basilar abscess has been suspected, though with no postmortem obtainable this could not be confirmed. One case of subdural abscess secondary to sphenoid infection was successfully operated on through the posterior sphenoid wall.<sup>6</sup> A case of abscess in the temporo-sphenoidal lobe from a sphenoid infection, and another of a frontal lobe abscess from the ethmoids, both of which were confirmed by postmortem, were reported by Berry.<sup>7</sup>

Subdural abscess is probably more frequent than brain abscess or meningitis, but it is usually not diagnosed until symptoms of brain abscess have become manifest. Thus Gerber<sup>8</sup> in collecting data on intracranial complications of sinusitis found that in 240 such cases there were 65 brain abscesses and 28 subdural. Cases of subdural abscess are more frequently recognized now than formerly, and they are quite frequently found during the course of a radical sinus operation when not previously suspected. A brain abscess and a subdural abscess having no apparent connection may be present. The writer recently saw this coincidence. A frontal lobe abscess had been drained through the frontal bone and later, on opening the sinuses, a large subdural abscess was found over the ethmoid region on the same side. The patient died from multiple brain abscesses.

*Acute and Chronic Otitis Media.*—Chronic purulent infections in the nose are often responsible for an acute otitis media. The chronic catarrhal changes in the tube and middle ear may be associated with the mild hyperplastic type of sinusitis. Very rarely are these changes seen in a purulent type.

*Ocular Complications.*—Many of the inflammatory conditions of the eye are due to a pus focus in the neighboring sinuses, but as these lesions may be produced from a primary focus in other parts we will not deal specially with them.

Rupture of an ethmoidal abscess into the orbit is not uncommon. The condition formerly described as an orbital cellulitis is usually a direct extension from the ethmoids or frontal. An extension of infection causing marked swelling of orbital tissue with exophthalmos may occur from a sinusitis even though no pus can be demonstrated in the orbit at operation.

The proximity of certain nerves to the sphenoid and posterior ethmoids, as they pass through the sphenoidal fissure, renders these nerves liable to damage from infection in the external walls of these sinuses. The nerves most liable to injury are the sensory branches, viz., the lacrimal, nasal, and frontal from the ophthalmic division of the fifth, producing pain in the region of their distribution, or the motor nerves may be involved, producing ocular palsies. The sixth is the most commonly affected, though it may be either the third or fourth, or even all three, with resulting paralysis of the muscles each supplies.

*Involvement of the Optic Nerve (Retrobulbar Neuritis).*—This is a complication easy to understand by simple reference to the anatomical relation between the optic nerve and the paranasal sinuses. The nerve in its bony sheath passes through the upper and outer anterior part of the sphenoid in about 75 per cent. of cases. In many other cases it projects into the cavity of a postethmoid cell. The bony wall covering the nerve is very thin, and there may be absence of bony covering. In 220 sinus dissections by Neivert

dehiscences in bone were found in 12 subjects.<sup>9</sup> It is thus easy to explain how an inflammatory process in these cells can affect the nerve by direct extension. This is probably what occurs, but the immediate relief of nerve inflammation when these cells are opened have led some to believe that the nerve is affected by pressure. In other cases the phenomenon would appear to be due to toxic absorption, as the neuritis has been observed from toxic causes when the focus was not proximal to the nerve. The sinusitis most often associated with these nerve lesions is the hyperplastic type, though they may occur in purulent cases and have been observed in primary acute conditions of the sinuses.

The association of retrobulbar neuritis with disease of the sphenoidal sinus has been recognized for many years. Berger and Tyrman<sup>10</sup> in 1886 reported 26 cases. A case was reported by Fliess<sup>11</sup> in 1895 in which the neuritis was cured by opening the sinus. Subsequently cases were reported by various authors. A. Onodi<sup>12</sup> published some data on the subject and various cases have been reported, but the importance of its early recognition and early operative treatment was not generally recognized. The present generally accepted knowledge of the disease and its treatment both among oculists and rhinologists has only occurred in the last five years. Leon White<sup>13</sup> in various articles has dealt quite exhaustively with the subject, but has recently become convinced that other foci, such as tonsils and teeth, more frequently produce optic neuritis than the sinuses.

Cases of short duration, before the nerve has become seriously damaged, make a recovery which seems almost miraculous when the posterior ethmoids and sphenoid are opened. Normal vision may be restored in twenty-four hours. Cases which have lasted longer recover more slowly, and the longer the duration, the less prospect of recovery. The age and regenerative power of individuals vary greatly, and as a general rule the younger the patient, the greater and quicker will be the manifest recuperative power. The writer has recently had a case of retrobulbar neuritis in a child of thirteen years of more than two months' duration with vision of 20/200 in the left eye which returned to normal three days after operation. The right eye in this case had been affected less severely and had 20/60 vision. This returned to normal the next day. Although the process had lasted some time in this case there was apparently no atrophic change, so recovery was rapid and complete. She gave a history of an attack of influenza two months before the failure of vision was noticed.

The symptoms are failing vision, often with early loss of colors, enlarged blind spot, and contraction of the visual field. Sometimes headache is present and may be localized over the frontal region of the affected side. The nasal symptoms are often very slight. Pus may be found in the posterior sinus region, but more often it is not, and a thickening of the membrane may be all the noticeable change. This hyperplastic change can frequently be shown by the Roentgen rays. The ophthalmoscope will show a neuritis which may show variable atrophic changes depending on the duration. In early cases it may be distinctly noted that the nasal portion of the nerve shows most involvement.

*Osteomyelitis.*—An acute osteitis involving the bony framework of the sinuses and even the cranial bones is often spoken of as a complication of sinusitis. It is rather difficult to establish this relation definitely, as the involvement of the bone may be the primary lesion, and this may be a blood-

borne infection, the same as an osteitis in one of the long bones. McKenzie<sup>14</sup> in 1913 recorded data on 41 cases of osteomyelitis associated with sinus disease. Of these there were 21 spontaneous and 20 postoperative. Lillie,<sup>15</sup> Kernan,<sup>16</sup> and others have reported cases since then, and almost everyone attending a large clinic has seen one or more. It usually affects the frontal bone, and produces swelling and severe pain over the frontal sinus. If the bone is opened in this early stage there may be a seropurulent fluid found between the periosteum and the bone externally and the same type of fluid found in the frontal sinus. Necrosis involving the whole thickness of the bone follows, and this condition may spread through the diploë to the other cranial bones. Fluid forms between dura and the bone, and if this is not quickly localized a meningitis follows. If it becomes localized a subdural abscess forms, communicating through the infected bone with pus beneath the periosteum. This produces a boggy swelling externally which is not abruptly localized—the “Pott’s puffy tumor” of the old surgical text-books. Recovery may take place after a period of months, with the separation and removal of sequestra as they form. If the condition arises as a sequel to operation on the sinuses the prognosis is less favorable. In McKenzie’s<sup>14</sup> records there was 100 per cent. mortality in postoperative cases, but from later records there have been some more favorable results. The writer has seen 3 cases, one spontaneous and 2 postoperative, all of which made complete recoveries. One occurred in the occipital bone after an intranasal ethmoid operation. A trephine opening was made through the bone and a subdural collection of pus drained. Another case, postoperative to a radical frontal, had necrosis of bone in parietal and occipital. All the sequestra finally separated and were removed. The case of spontaneous osteitis had numerous sequestra removed and finally recovered.

The remote effects of a chronic sinusitis which might properly be classed as complications include all that great category of ills, the causation of which is now ascribed to focal infection anywhere. It includes many of the diseases which were formerly attributed to syphilis and, in fact, many of the degenerative results supposed to be peculiar to syphilis can be duplicated by a pus focus with absorption going on over a long period of years. Even the gumma or syphilitic granuloma is occasionally found associated with a pus focus infected with streptococci or staphylococci. The arterial changes in small thin-coated vessels, such as the choroid and retina, with hemorrhages, are often found associated with a pyogenic infection in the nasal sinuses.

The chronic inflammatory processes due to a pus focus may be enumerated: Inflammatory processes in all the tissues of the eye; labyrinthitis; bronchitis and pulmonary abscess; gastric ulcer; cholecystitis and cholangitis; appendicitis; nephritis in its various forms; pyelitis and cystitis; arteritis; myocarditis and endocarditis; simple and malignant arthritis; myositis; bursitis, and neuritis. Whenever and wherever in the body a chronic inflammatory process has been initiated and the origin from a primary focus is suspected, but not obvious, it is well to have a thorough examination of the paranasal sinuses.

**Treatment.**—*Prophylactic.*—The great number of organisms which we have seen to be responsible for infection of the sinuses renders prophylaxis rather difficult. In larger cities most people are exposed to these organisms every day, yet they only become infected when the organisms seem to acquire increased virulence in some way or the resistance of the individual

becomes lowered. There is no doubt that much could be done to prevent multiplication and spread of organisms by educative means. Infections in the head are contagious, and if precautions were exercised to prevent the spread of this contagion by taking proper measures to destroy the infected secretion, as is done in tuberculosis, it would go a long way toward preventing those seasonal epidemics which recur several times a year. The common pocket handkerchief and the infected pocket is still a menace to the public health, and is a very potent means of spreading infection. Both could easily be eliminated without much inconvenience. Before the germ theory of disease was known an old country doctor found that all his obstetric cases became infected and he worried days and nights over his sad results. Finally he decided to throw away his driving gloves, with the result that his misfortune came to an end at once. While sinus infection is not so disastrous as puerperal infection, yet its prevention is a matter well worth more consideration than it has ever received.

The control of contagious secretion from sneezing and coughing is, of course, very difficult, but both can be suppressed to a great extent by a conscious effort, and people should be taught the importance of this. A member of a family affected with sinusitis could easily be taught the same precautions that a tuberculous patient is now taught and thus prevent other members from acquiring the disease.

*General Treatment.*—The general treatment of cases of chronic sinusitis has never received sufficient attention. They should all be placed as far as possible under the best hygienic surroundings and, if possible, should have an out-of-door occupation. General tonic treatment with good food and fresh air—in fact, the same regimen which is prescribed for tuberculosis patients—would be of great benefit.

*Local Treatment.*—The first consideration will be the condition of the drainage and ventilation of the nasal chambers. If the nose is open and the sinus region is accessible one may proceed with the ordinary non-operative method of treatment. If, on the other hand, there is marked nasal obstruction it is imperative to correct this at once. A complete submucous resection of the septum may be all that is necessary, but more often with this either a partial removal or cauterization of the middle turbinate is advisable, the whole object being to get the upper part of the nose properly opened, whether that means much or little in the way of surgery. Having accomplished this, carry on frequent irrigations with warm saline solution. Usually this can be carried out partly by the patient himself, either using the irrigation with suction carried out by the nasal syphon or water-tap apparatus, or simply using the gravity-flow in one side and out the other with the head inclined well forward. After this place the patient in an extreme opisthotonos position and drop 10 per cent. argyrol or neosilvol into the nose. Gravity will carry this to the top of the nose around the openings of the ethmoid cells and even into the frontal sinus. In office treatment instead of applying argyrol in this way a cotton pack can be inserted in the nose and allowed to remain *in situ* for half an hour, after the method of Dowling. This treatment should be persisted in daily for a period of a month or six weeks provided, of course, that there is no evidence of caries or necrosis of bone or marked polypoid degeneration.

A course of treatment will often reduce a profuse purulent discharge to a scanty mucoid one and will render the patient sufficiently comfortable to

accept cheerfully the little remaining inconvenience even though there is not a complete cure. Headache and the feeling of fulness in the head may also entirely disappear under this treatment.

The sphenoid and frontal can sometimes be treated by direct lavage through a cannula. It is very rarely possible to do this successfully through the natural openings. In some cases after the removal of the anterior end of the middle turbinate a frontal can be washed out successfully.

**Operative Treatment.**—The indications for operation in chronic sinusitis are:

1. Sinusitis with profuse purulent discharge which will not clear up on treatment.
2. Sinusitis with polypoid degeneration.
3. Involvement of neighboring structures, ocular nerves, or threatened intracranial complications.
4. Rupture of cells with pus in the orbit or beneath the skin externally.
5. With certain cases of asthma.
6. Where a pus focus in the sinuses is acting as a focal infection producing disease in other parts.
7. Cases with frequent acute exacerbations producing headache and nasal obstruction.
8. Cases with external fistulæ.

1. *Sinusitis with Profuse Purulent Discharge.*—This usually indicates involvement of all the sinuses, and the patient's life is made miserable by the continual necessity of using handkerchiefs. Usually the intranasal operation of draining the antrum, exenteration of ethmoids, and enlarging the opening of the sphenoid will be the one of choice. The frontals, however, may continue to secrete so much that the patient is not sufficiently relieved, in which case an external radical operation may be necessary. The writer has had a case where, after the complete intranasal operation and lavage of the frontal for six months, the patient still had so much annoyance from the secretion that an external radical frontal sinus operation had to be performed on both sides, the final result being quite satisfactory. After an intranasal operation a free purulent discharge from the frontal may continually infect the operative field so that exuberant granulations continue to form and no healing occurs till the frontals have been cured by a radical operation.

2. *Cases with Polypoid Degeneration.*—The intranasal exenteration of ethmoids is usually sufficient. If, however, polypi are present in the frontal they continually invade the nose, and the condition will not subside till a radical operation is performed.

3. *Involvement of Neighboring Structures.*—In cases with nerve involvement the exenteration of ethmoids and free drainage of the sphenoid is usually sufficient. In retrobulbar neuritis cases, as soon as a diagnosis is made, the posterior ethmoids and sphenoid should be opened. A possible exception to this might be made in young subjects where an early diagnosis is made and the lesion seems to be due to a primary acute sinusitis. In threatened intracranial complications operation is not usually successful in averting disaster, but the writer has seen one case in consultation, with well-marked signs of meningitis, with positive findings in the cerebrospinal fluid, recover after a thorough radical operation on the sinuses was performed. It, therefore, seems justifiable to make the attempt to save these patients by an operation.

4. *Rupture of Cells Into the Orbit or Beneath the Skin.*—Cases may recover without operation even with pus in the orbit provided there is free intranasal drainage, but, as a general rule, an external operation will be necessary. If symptoms are acute the operation should be as limited as

possible. If they are gradual in onset and have persisted for some time the complete radical can be performed with safety.

5. *In Certain Cases of Asthma.*—The intranasal operation may relieve or effect a complete cure. In cases due to a sensitization from bacterial proteins it is probable that the only chance for a complete cure would be the elimination of the focus entirely, and this might necessitate a radical operation.

6. *Where Sinuses Are Acting as a Focal Infection.*—These cases usually require the complete elimination of the focus. They will show improvement from a partial removal and then relapse. An intranasal operation should be tried first, but with recurrence of symptoms a more radical procedure will be advisable. They improve and relapse till the focus is entirely removed.

7. *With Frequent Exacerbations.*—Usually the intranasal operation will suffice provided free drainage of the frontal is effected. If, however, there is continued frontal pain the external operation may be the only means of relief.

8. *External Fistulæ.*—These denote diseased bone and will not get well till a radical operation is performed and all diseased bone removed.

**The Intranasal Sinus Operation.**—This is the operation of choice in most cases and the result usually depends on how thoroughly it is done. The paranasal sinuses show great variation in their anatomy and afford one of the most difficult surgical fields in the whole body. Operations in this region should only be attempted by those who have a thorough knowledge of the anatomy and have had experience on cadavers. Various methods of attacking the sinuses have been described, but the method affording the greatest degree of safety is the one described by Dr. Harris P. Mosher. From a long experience in teaching operative surgery to post-graduate students the writer has realized the great danger attending this operation when many of the instruments commonly recommended are put into the hands of the inexperienced. He has devised special instruments for performing the Mosher operation which have added something to the element of safety, and perhaps to the thoroughness of the operation. Every possible means should be exercised to render the procedure less dangerous. A good *x-ray* should be kept in view during the operation. The patient should be in the recumbent position with shoulders and head slightly elevated. This will prevent the unconscious movement of the head backward which is apt to occur if the patient is sitting up. A local anesthetic is preferable, as there is less bleeding and some vision of the field is allowed. Under general anesthesia the operation has to be done almost entirely by the sense of touch and a great deal more experience is necessary. With the patient recumbent it is possible to use a much larger amount of strong cocaine without untoward symptoms. The usual method of cocainizing for a septum operation can be followed up by painting the ethmoid region with a few drops of a saturated solution or by applying "cocaine mud" on an applicator. A submucous operation should be done as a preliminary procedure on all cases. This provides a flexible septum which can be pushed over, making the field far more accessible to direct vision. Besides it makes possible much more effective after-treatment in the way of taking proper care of granulations and preventing adhesions. The submucous may have been done previously, but if not, it can be done as soon as the nose is cocainized for the sinus operation.

The following is the author's method of performing the Moshier operation with slight modification and the use of special instruments:

Before attacking the sinuses make an incision with a small-bladed knife along the whole base of the middle turbinate underneath and external to the body of the bone (Fig. 56). This will leave a clean-cut edge of mucous membrane at the lower margin of the ethmoid area when the operation is

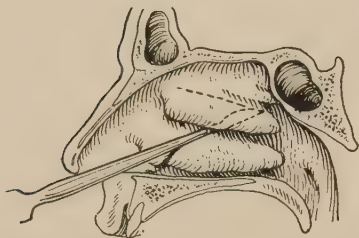


Fig. 56.—Knife cutting the membrane along the base of the middle turbinate.

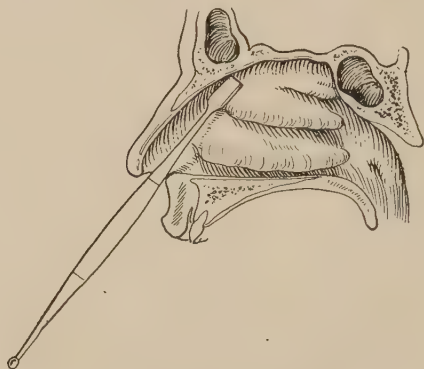


Fig. 57.—Spade curet pressed outward over the cell of the agger nasi.

finished. This will facilitate the spread of epithelium over the granulating surface of the os planum, and it will also prevent loss of portions of mucous membrane over the middle meatus, which may occur when portions of débris are removed with forceps.

The large end of the spade curet is now placed just above the anterior end of the middle turbinate over the cell of the agger nasi (Fig. 57). The upper end of the curet will be just above the level of the inner canthus.

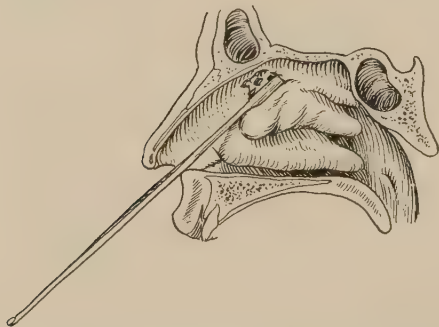


Fig. 58.—Spade curet starting backward pressure breaking down cells and middle turbinate.

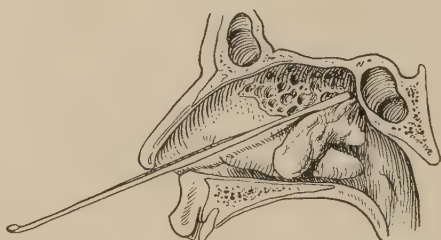


Fig. 59.—Spade curet pressed backward and downward carrying broken cells and middle turbinate before it.

If pressure is now made directly outward the curet will enter the cell. Its broad surface will prevent its being pushed far enough outward to enter the orbit, an accident which can easily happen with a small curet. Having penetrated the agger nasi cell, rotate the curet on its long axis till its concavity faces directly backward (Fig. 58), then push backward through the whole ethmoid labyrinth. When it has traversed all the posterior cells in this direction press it downward. This will carry the whole mass of broken

ethmoid cells down onto the upper surface of the inferior turbinate (Fig. 59). Now insert a blunt grasping forceps, such as Hurd's submucous forceps or Luc's, and with blades well open grasp the whole mass (Fig. 60) and withdraw it. The whole of the turbinate should come away in the mass. If it does not, it may have been pressed into the posterior part of a deep middle meatus and several attempts may be necessary before finding it. It will

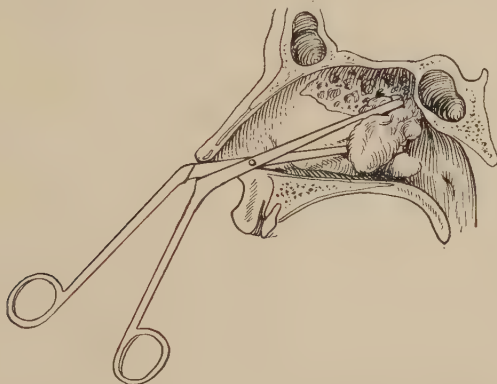


Fig. 60.—Forceps grasping mass of broken cells and middle turbinate.

often be found that the curet in its sweep backward has broken down the anterior wall of the sphenoid and, if it has, so much the better. The large end of the ring curet should now be inserted and gently moved backward and forward over the broken down area, paying special attention to the cells just anterior to the nasofrontal duct (Fig. 61). The small end may be used for these cells, care being taken to exercise pressure in a forward and down-

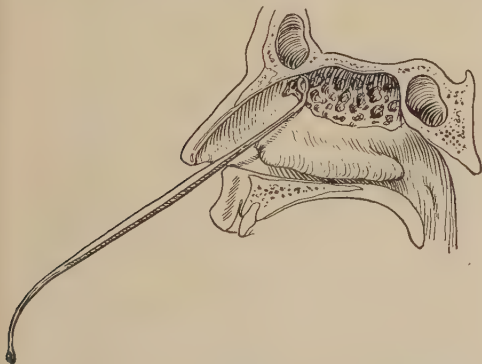


Fig. 61.—Ethmoid curet finishing breaking down of cells and smoothing bony surfaces.

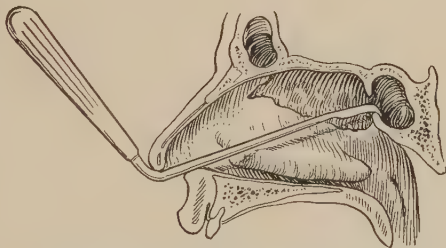


Fig. 62.—Sphenoid curet pulling downward and forward, breaking down anterior sphenoid wall.

ward direction. Considerable pressure can be used with the large ring to break down cells just under the roof plate of the ethmoid, also in the posterior external angle.

To enlarge the sphenoid opening insert the small end of the spade curet along the floor of the nose to the nasopharynx. Withdraw it slowly along the floor of the sphenoid close to the septum till it rests on the anterior wall. The thin portion of this wall will soon be found and the curet can

be pushed through it. By rotating the curet with movements downward and inward the opening can be enlarged enough to insert the hook curet (Fig. 62) or a biting forceps. The biting forceps is a nicer surgical instrument than the curet, but it sometimes makes a clean cut across the sphenopalatine artery and produces a troublesome hemorrhage.

An applicator or probe should now be passed into the frontal sinus, and if any difficulty is experienced it will probably be due to a few anterior cells not having been broken down. When this has been accomplished there is usually no difficulty in entering the frontal. Various instruments have been devised for enlarging the frontal opening, but it is doubtful if their use is advisable, as they can only be used in a forward direction against a hard compact bone and probably accomplish very little. Besides they injure the membrane around the ring-like nasofrontal duct, and the integrity of this is an important factor in maintaining good drainage.

No packing should be put in the nose when the operation is finished. If there is much bleeding a small amount may have to be inserted, but it should be removed as soon as possible. Superficial cotton-plugs placed in each nostril saturated with Dobell's solution one-third strength, and changed every three or four hours, is all the dressing necessary.

*After-treatment.*—This should be as little as possible. If there is considerable secretion the nose should be kept clean with a daily irrigation. Exuberant granulations are apt to form in a moist area like the nose. They can be taken off with a sharp biting forceps or curet. This may have to be done several times before healing is complete. The sphenoid opening is very prone to close up, and this may have to be enlarged as necessity arises.

*Accidents.*—**PENETRATION OF THE ORBIT.**—This is not usually very serious though the rather sudden proptosis from hemorrhage may appear quite alarming. It usually absorbs without suppuration if the sinuses have been thoroughly cleaned out.

**EXPOSURE OF DURA.**—This may occur where there is bony necrosis in the roof of the ethmoid. If the sinuses have been thoroughly cleaned out and dura is not penetrated probably no harm will result. If the dura is penetrated with a flow of cerebrospinal fluid meningitis is very liable to follow, though two or three weeks may elapse before its onset.

**INJURY TO THE OPTIC NERVE CAUSING SUDDEN BLINDNESS.**—This could only occur from using instruments, such as biting forceps, too boldly in the posterior ethmoid angle.

**OSTEITIS.**—Cases of osteitis with necrosis in the cranial bones have occurred after intranasal operations on the sinuses.

*Results.*—The final results are usually satisfactory, but there is a group of cases with persistent osteitis and obstinate headache in which the operation may be very disappointing. Dryness of the nose has been mentioned as a sequel due to removal of the middle turbinate. I have never seen a result of that kind where I thought the removal of the turbinate had anything to do with it. A continued purulent discharge from the frontal, or from an ethmoid not completely exenterated, may induce an atrophic change with its usual symptoms of dryness. The results in cases with secondary foci of infection are very long delayed oftentimes, and not till every cell has been thoroughly drained and pus has entirely disappeared will the secondary condition clear up. Most commonly the defective drainage is found

in the sphenoid and these cases may have to be dealt with in a very radical way. The writer has in 2 cases found it necessary to remove the posterior portion of the nasal septum, removing the lower anterior portion of the sphenoid septum and the floor of both sphenoids. Both cases had a cess-pool deep down in the body of the sphenoid and drainage of this cavity was imperative in each case. The result in one case was excellent, in the other, symptoms still persisted.

**The Radical Operation.**—This operation is done only when the intranasal operation has failed to cure chronic disease in the frontal sinus or in some inaccessible ethmoid cells. In some ways it is the safer method, but on account of the probability of causing some external deformity it is avoided when possible.

The method usually practised is the one devised by Killian with some modification. This leaves the supra-orbital ridge as a bridge of bone, thereby greatly lessening the deformity. Every vestige of diseased membrane must be removed from the sinus walls and the ethmoid cells, the final aim being to obliterate these cavities and destroy them as secreting cavities. Unless this is done the operation will likely be a failure. Many conservative methods have been tried and have failed. Opening externally to enlarge the intranasal drainage opening in chronic disease (the Ogston-Luc operation) is not a successful procedure. The removal of bone from the nasal process and floor of the sinuses which is done in this operation allows the orbital tissue to encroach subsequently on whatever drainage passage there is between the frontal and the nose. It may be adopted, however, as a temporary measure in acute infection.<sup>17</sup> In small sinuses the entire anterior wall of the frontal sinus can be removed and the cavity obliterated without causing serious deformity. This method may be considered a modification of the Kuhnt operation described by Riedel. It is the method most certain to effect a permanent cure, as subsequent trouble with the bony bridge or breaking down of tissue under the bridge may occur after the Killian operation. There is no fixed rule, however, to guide one in deciding which operation to perform. The Roentgen ray will give a fair definition of the sinus, but it is usually wiser to determine the exact size after opening through the bone at the inner portion of the sinus. If the sinus does not extend beyond the middle of the supra-orbital ridge it will cause very little deformity to remove the whole anterior wall. A short portion of the ridge can be left projecting inward as a buttress. Whichever method is adopted for the frontal part of the operation, the complete exenteration of all ethmoid cells must be done, and a large opening made in the anterior sphenoid wall. In most cases it will be found a great advantage to have done as thorough an intranasal operation as possible previous to attempting the radical. This region can then be packed with gauze soaked in adrenaline and a comparatively dry operative field can be obtained for removing any cells which remain and observing exact pathological condition.

**The Radical or Killian Operation.**—Nearly all descriptions of this operation show slight modifications of the original Killian. A good detailed description of the operation is given by Skillern.<sup>18</sup> Many slight changes in procedure are made by other operators. The following method of performing the Killian operation is the one favored by the writer:

If the intranasal operation has been done the nose on that side is packed with a piece of cotton soaked in adrenaline chloride just before the anes-

thetic is started, and a postnasal plug inserted in the nasopharynx. If an intranasal operation has not been done previously the writer prefers to do the intranasal operation after the patient is under the anesthetic, before the external incision is made. The ethmoid region is then packed with gauze soaked in adrenaline. The advantage of this is that by the time the ethmoid region is reached through the external opening the field is dry and the operation can be finished much more quickly and easily and probably with better attention to details. Having done this, an external incision is made through the lower part of the eyebrow, turning downward in a curve at the inner angle, and carried down the side of the nose to the level of the lower limit of the nasal bones (Fig. 63). It is better not to make the incision to the bone for the full length with one sweep, as hemorrhage is quite severe if this is done. The vessels lie deep on the periosteum and they can be picked up more easily if cut one at a time. The soft tissue with the periosteum is separated from upper and inner wall of the orbit, pushing aside the pulley of



Fig. 63.—Initial incision in radical operation and incision over the nose to give wider exposure of sinus.

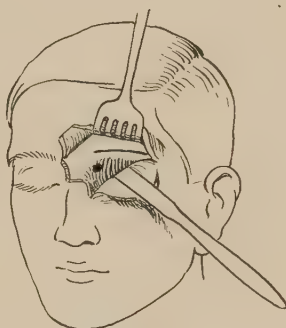


Fig. 64.—Flaps retracted and periosteal incisions shown; point of election for entering the sinus.

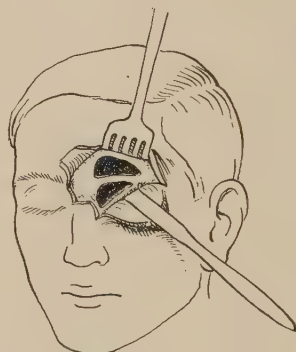


Fig. 65.—Frontal sinus cleaned out, leaving bridge and ethmoid region exposed.

the superior oblique muscle and separating the lacrimal sac gently from the bone with the soft tissue. I now proceed to tie all bleeding vessels, for artery clamps are very much in the way as they lie over the eyeball. If the x-ray has shown a large sinus which extends high up, I make a cross incision straight across the bridge of the nose and then a lateral incision upward on the other side of the nose corresponding to the lateral incision on the side to be operated on (Fig. 63). The whole flap is then elevated upward, leaving the periosteum intact. This supplementary incision if carefully sewed up leaves practically no scar and gives such a good exposure that there is no need of any strong retraction on the soft tissues—a very important matter where one wants to get primary union. With gentle traction on the retractor a good exposure of the anterior wall and floor of the sinus can now be obtained. An incision is made in the periosteum  $\frac{1}{4}$  inch above the supra-orbital ridge (Fig. 64), and the periosteum above this incision elevated as far as necessary. The remaining periosteum is left over the ridge and portion of bone to be left as a bridge. This should be made wide enough to turn over the upper edge of the bony bridge. This sinus is now opened at the inner angle just below the ridge and the whole floor removed. The bone

above the bridge is then chiseled through exposing the whole posterior wall of the sinus. All diseased membrane from every nook and corner should be removed with forceps and gauze, and the condition of the inner plate carefully observed. It is not advisable to curet this bone. The upper part of the nasal process of the superior maxilla should now be removed as far forward as the original incision, and the anterior part of the os planum and upper part of the lacrimal bone removed (Fig. 65). This will give a good opening into the ethmoid region. The remains of ethmoid cells are removed, paying special attention to any prolongation of cells over the orbit. These cells may run back over the roof of the orbit nearly to the optic foramen. The sphenoid opening should now be enlarged till the opening is flush with the floor, if possible. The amount of inner orbital wall (os pla-

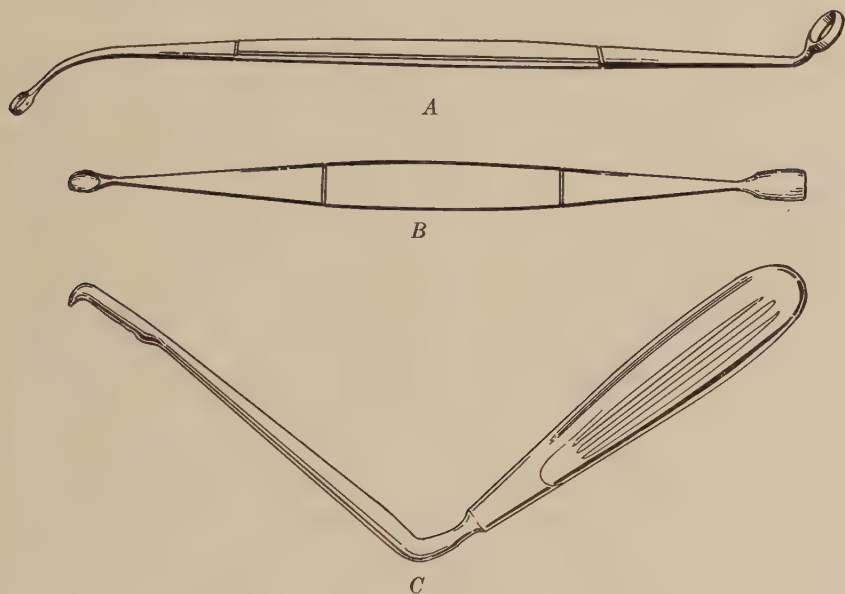


Fig. 66.—Curets for intranasal sinus operation. *A*, Ethmoid curet; *B*, spade curet for starting the ethmoid operation; *C*, sphenoid curet.

num) removed should be as little as possible, for if this is taken down freely the contents of the orbit crowd into the upper part of the nose and adhesions and pockets may form. The external wound is now carefully closed, leaving only a small cigarette-drain at the outer part for forty-eight hours. The inner part of the wound drains into the nose. Pressure pads are placed over the frontal above and below the bridge, and a bandage put on as tightly as can be borne. Cotton soaked in Dobell's solution or alkalol is placed in the nostril and changed every four hours. No packing is inserted in the nose.

If after opening the sinus at the inner angle it is found with a probe that the extension of the cavity is quite limited, I remove the whole anterior wall, leaving a portion of the supra-orbital ridge externally as a buttress, but not a complete bridge. This enables the soft tissue to fill the inner part of the space to be obliterated and, though there is a slight depression from this, the deformity is negligible.

*After-treatment.*—Dressings should be changed every day and pressure maintained for three weeks. The patient should be instructed not to blow the nose. If there is much discharge irrigations of a warm alkaline solution may be advisable; otherwise keep the wet plugs in the nostrils for three or four days and leave the nose alone. If at the operation necrosis of the posterior wall is found with any subdural collection of pus, the wound should not be closed. Iodoform gauze should be inserted and this dressing continued till the wound is thoroughly clean, when a secondary closing of the wound can be done to lessen the deformity.

*Results.*—In cases with no threatened complications at the time of operation results are generally good. Pain is usually entirely relieved and if the operation has been thoroughly done suppuration will cease and any symptoms depending on pus absorption will disappear. A reinfection or lighting up of the old infection may occur even when all cells have been thoroughly treated. This is not to be wondered at when one considers that there is considerable organization of low-grade fibrous tissue afterward which may retain organisms only waiting for a chance to produce a recrudescence when the individual's vitality is lowered. These recurrences may require an external incision and drainage the same as they do in old mastoid wounds. A pocket may be found which necessitates packing the wound for some time before it fills up.

*Untoward Results.*—There is a long list of such happenings many of which had been recorded before the operation was brought to its present state of development. This list includes such fatal complications as meningitis, brain abscess, and osteomyelitis with subsequent meningitis; and of the minor complications, edema of the eyelid, paralysis of the upper lid, hemiceranial anesthesia, supra-orbital neuralgia, falling out of eyebrow or excessive growth of eyebrow, pneumatocele, temporary and permanent diplopia, and blindness on the operated side.<sup>19</sup> A temporary diplopia nearly always occurs, but usually clears up in two to six weeks. The intracranial complications in some cases are due to a latent condition being aroused into activity by the operation.

#### MUCOCELE

This rather unusual phenomenon is characterized by a slowly increasing swelling in the sinuses causing obstruction in the nose or external swelling. It does not present the usual signs of inflammation and is more apt to be mistaken for a new growth. Its most common situation is in the anterior ethmoids and frontal, and it usually produces an external swelling above the inner canthus. If allowed to continue its progress it may grow into the orbit causing great displacement of the eyeball with paralysis of ocular nerves. A few cases have been recorded occurring in the sphenoid and have produced eye symptoms from pressure. The contents of the swelling varies. It is usually a thick, tenacious, mucus-like fluid, but may be either purulent or watery in consistency. It is probably always due to an inflammation in which the infection which initiated the process has been overcome. The cyst-like swelling may increase in size causing erosion of bone till the dura is exposed or the orbital contents are pushed out of place.

The symptoms are usually those of nasal obstruction or referable to the pressure in the orbit. When it produces external swelling it may cause considerable disfigurement. The diagnosis, from suppuration extending through

the bone and causing swelling, is made usually by the absence of any acute symptoms in mucocele and its more gradual onset. It may be very difficult to distinguish it from a new growth and such mistakes have occurred. An exploratory needle puncture will determine the presence of fluid in the mucocele. In some cases Roentgen ray also will often yield valuable evidence toward differentiation.

**Treatment.**—This is always surgical and usually the external frontal route is the method of choice.

E. ROSS FAULKNER.

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### DISEASES OF THE MAXILLARY ANTRUM

**Etiology, Pathology.**—On account of the relation of the lining membrane of the antrum to the nasal cavity, to the other sinuses, and to the teeth, it is more often diseased than any of the other sinuses. Its ostium is so placed as to be easily blocked by acute tissue changes in the middle fossa of the nose, by chronic hyperplastic conditions of the middle turbinal body, or by direct extension of infection from the nasal mucosa. Infection from neighboring sinuses must also be considered. In addition the floor of the sinus is in intimate relation with the teeth which may project into its lumen, especially the second premolar and the first molar. Often, with devitalized teeth, abscess formation takes place without pain, and a dental cause cannot be excluded in maxillary sinusitis unless films of the teeth are taken as a routine. Like other cavities with a lining mucosa it is subject to acute and chronic inflammatory changes, necrosis of its bony walls, cysts, hydrops, and tumors. The inflammatory processes are acute and chronic catarrhal, and acute and chronic purulent. According to some authorities the chronic catarrhal process may be followed by hypertrophic or hyperplastic changes in the lining membrane. As additional etiological causes we have the exanthemata, the infectious diseases—influenza, diphtheria—trauma, and foreign bodies. Syphilis, tuberculosis, and osteomyelitis are seldom, if ever, primary causes of empyema.

#### ACUTE CATARRHAL MAXILLARY SINUSITIS

**Etiology.**—That the acute infections of the maxillary sinuses are only the local manifestations of a general process involving the nasopharynx and other sinuses is well recognized. On account of the relation of its lining membrane to the nose it is probable that no acute process takes place in the nasopharynx without involving the antrum. That we have predisposing causes in marked deviations of the septum, spurs, ridges, and enlarged middle turbinals crowded against the antranasal wall that interfere with the natural drainage and aëration of the antrum is conceded. These are, however, predisposing only, and may exist for years without harm to the patient, the presence of infection in the nasopharynx with impaired ventilation and drainage of the antrum being the active cause of a maxillary sinusitis. That such infection extends to the sinus by way of the lymphatics and blood-vessels of the mucosa is probable, and emphasizes the importance of protecting the antrum by treatment of the nasopharynx until complete resolution takes place in the mucosa following each acute infection, or after operative interference, especially if the drainage through the ostium is at all impaired.

**Pathology.**—In acute catarrhal maxillary sinusitis we have a low-grade infection which involves the surface mucosa. It is characterized by a serous infiltration whereby the membrane is increased in thickness some ten or fifteen times and appears like jelly. The exudate varies in amount and is mostly mucus. These findings have been confirmed by autopsy (Zuckerkindl). Effenorde accepts the classification, and Hirsch only questions whether it is an exudate or serous infiltration.

**Symptoms.**—The disease runs through its various stages of congestion and dryness, which are then followed by a serous or mucoid discharge. During the stage of congestion there is a feeling of stuffiness of the head,

with suffusion of the eyes and a slight rise of one or two degrees in temperature. There is seldom any pain in the teeth or over the canine fossa, although both symptoms may be present. As the infection is of low virulence and the antrum is usually involved in any general nasopharyngitis, there are no characteristic symptoms other than we would expect in an exaggerated cold. Within twenty-four to forty-eight hours there is a profuse, mucoid discharge, with improvement in the subjective symptoms. If the lining membrane of the antrum is much swollen, the discharge may be scanty. With this type of infection often one antrum is involved and then the other. Unless the resistance of the patient is low, or the ostium blocked, the infection gradually undergoes resolution in a week or ten days.

**Diagnosis.**—The diagnosis is made during the course of a routine examination rather than from the subjective symptoms of the patient. Our suspicion is aroused from the persistence of an unusual amount of discharge. Examination shows the tissues in the middle fossa to be swollen, and the mucosa about the ostium may bleed when gently wiped with a cotton applicator. The inferior turbinate is swollen and the nasal mucosa injected. Transillumination is dark over the malar eminence and a roentgenogram shows a thickened lining membrane of the antrum. If the discharge is unilateral, this is another aid in our diagnosis.

**Prognosis.**—The prognosis is good and resolution is the rule. If the middle turbinal is against the antronsal wall, or has been hypertrophied so that the ostium has gradually become obstructed, then a secondary infection may be followed by empyema of the antrum, or the acute catarrhal process may become chronic.

**Treatment.**—During the stage of congestion it is much better to refrain from local treatment and keep the patient quiet in a room with an even temperature day and night, to give a suitable, saline cathartic, and a liquid diet. A hot bath with the ingestion of large quantities of water is also of service. Steam inhalations promote the patient's comfort. Irrigations with watery solutions during the stage of acute turgescence endanger the middle ear and are followed by reaction. They also remove the protection of the mucoid secretion and cause punctate hemorrhages in the mucosa, increase the secretion, and prolong the symptoms.

After the stage of acute congestion is passed we may shrink the engorged mucosa with long thin pieces of cotton wet with a  $\frac{1}{2}$  to 2 per cent. solution of cocaine. (The addition of adrenaline is not of any advantage, as many patients will have vasomotor symptoms lasting two or three weeks following its use.) An inspection of the middle fossa follows. If there is space between the middle turbinal body and antral wall, so that the drainage through the ostium is not wholly obstructed, we content ourselves with the instillation of a solution of albolene to which is added 1 or 2 grains of menthol to the ounce, the inhalation of compound tincture of benzoin, 1 dram to a pint of hot water, oil of pine, or the use of a spray of albolene and menthol or oil of pine. These can be used by the patient. Further depletion can be obtained by the local application of cotton pledgets soaked in argyrol, 10 per cent., and placed gently in the middle meatus, where they are allowed to remain for twenty minutes. The oil inhalations and instillations give comfort to the patient, and small amounts of menthol cause a mucoid discharge that relieves the infiltrated mucosa.

In the later stages irrigations of warm normal salt solution under low pressure can be used to remove thick secretion, but are usually not needed, as the menthol or argyrol promotes a flow of mucus that keeps the nose free. Suction may be tried after shrinking the mucosa, especially if the ostium seems to be blocked. However, it is more important to relieve the swelling and infiltration of the soft tissues about the ostium, so as to promote drainage and ventilation of the antrum and bring about resolution. Moreover, suction, unless carefully used, is apt to be followed by secondary congestion. Its most useful purpose is to remove any plug of mucus that may be obstructing the antral opening. We must remember that the tendency is for the acute process to undergo resolution unless there has been previous thickening of the tissues covering the lateral wall so as to impair drainage through the ostium or interfere with the function of the cilia.

This treatment is usually effective. In obstinate cases it may be necessary to wash out the antrum, after removing the anterior end of the middle turbinal, either through the ostium or after puncture of the antronal wall. On account of the danger of infecting the antrum this should be used as a last resort, and will seldom be necessary.

#### CHRONIC CATARRHAL MAXILLARY SINUSITIS

**Etiology.**—Chronic catarrhal maxillary sinusitis is usually the sequel of an acute catarrhal process in the nasopharynx. As predisposing factors we have to consider the influence of deviations of the septum, ridges, spurs, etc., that have impaired drainage and, because of the uneven nasal respiration, have been followed by a compensatory hypertrophy of the soft tissues, especially around the end of the middle turbinate. This hyperplasia has been increased by repeated acute infections until the mucosa about the ostium has become so thickened as to seriously narrow the normal opening, *impair drainage*, and interfere with the usual protection of the cilia. It has been claimed that occasionally the acute process may undergo resolution in the nasopharynx, leaving the infection still active in the antrum. This, however, must be a rare occurrence, for, with the absorption of the inflammatory products in the nasal mucosa, there is also improved lymphatic and circulatory drainage of the tissues about the ostium and improved ventilation of the antrum. Unless the function of the cilia has been seriously compromised, or the hyperplasia of the mucosa has gone so far that normal drainage is not restored by absorption of the inflammatory products about the ostium, the tendency of the lining membrane of the antrum is to improve with the subsidence of the infection in the nasal mucosa. It is the rule for all mucoid secretions to become thick and viscid as they become scanty, and so to further impair drainage, but absorption takes place without organization as long as the circulation and natural outlet are free.

**Symptoms.**—In chronic catarrhal maxillary sinusitis the prominent symptom is a persistent unilateral or bilateral discharge. This may be diminished in amount owing to infiltration of the mucosa about the ostium and to the thick mucoid character of the discharge. Pain and discomfort are rarely present. On account of the impaired drainage and the absence of symptoms directing attention to the antrum, chronic catarrhal maxil-

lary sinusitis is usually overlooked or neglected until reinfection is followed by empyema.

**Diagnosis.**—The diagnosis is made from the persistence of a characteristic discharge; the positive shadow on transillumination; the accumulation of thick secretion in the morning in the postnasal space; and the changes in the nasal mucosa on the involved side. The roentgenogram shows a thickened membrane.

**Prognosis.**—The prognosis is good.

**Treatment.**—Very few of these cases of chronic catarrhal sinusitis will seek treatment except for an acute nasopharyngitis, and the underlying condition will be overlooked. When the pathology is recognized the indications are to correct abnormalities of the septum that tend to impair drainage and ventilation. The thickened lining membrane can be helped by the use of Dowling's packs. If the middle turbinal is against the outer wall this should be fractured and set toward the middle line. When the end of the middle turbinal is enlarged enough to interfere with drainage through the natural opening it should be amputated. Following these measures the instillation of 10 or 15 drops of albolene, to which has been added 1 grain of menthol to the ounce with a medicine-dropper, will conduce to the patient's comfort. The promiscuous use of a spray of a watery solution should be condemned.

#### ACUTE EMPYEMA OF THE MAXILLARY SINUS

**Pathology.**—The pathological changes in the antrum as the result of an acute purulent process primarily affect the mucosa which becomes swollen and edematous with localized hemorrhages. A purulent secretion covers its surface. In the severe forms ulceration of the lining membrane occurs which may involve the bony walls. The usual termination is in resolution of the inflammatory products, and partial or complete restoration of the mucosa.

**Etiology.**—A history usually shows that an acute empyema arises from some dental cause, or it is a complication of some acute infection, or occasionally it results from secondary infection of an acute or chronic catarrhal process.

**Symptoms.**—In an acute empyema of the maxillary sinus the nose is obstructed, the whole head feels full, and there is a localized or general headache depending in its severity upon the amount of drainage present. In a closed empyema the retention of the secretions, with increased absorption and pressure, makes the symptoms more pronounced, and the pain may radiate to the side of the head or over the eye. There is pain and soreness over the antrum and often pain in the teeth. The pain may be neuralgic in character and is usually intermittent whenever there is any discharge. A persistent cough, that stops almost at once on washing out the antrum, has been observed. The acute secretion may cause a dermatitis about the vestibule of the nose and upper lip. The sense of smell is present unless there is marked swelling of the mucosa in the middle fossa or an associated ethmoiditis. A fetid odor is usually associated with dental necrosis, but not invariably. The eyes are heavy and ache when used. The pharynx is irritated from the discharge, and the digestion may be disturbed when this is swallowed. There may have been a chill and some increase in temperature, although these are not prominent symptoms.

There is no external swelling over the antrum beyond a slight puffiness below the eye, and no external redness. The toxemia is marked and the patient suffers from malaise, mental lassitude, or depression. Sweating or alternate flashes of heat and cold may be present. Many cases occur in which the drainage through the ostium remains fairly free and in which there are but few symptoms beyond those seen in an acute nasopharyngitis. In the majority of cases the discharge of pus appears in forty-eight hours, and the subsequent course is subacute except for some one prominent symptom like pain.

**Diagnosis.**—At the time the examination is made the objective findings vary greatly. There may be only a history of acute infection followed by a persistent purulent unilateral discharge. The inferior turbinate is swollen; the mucosa more injected on the affected side, and there is pus in the middle meatus and epipharynx. On pressure there is also tenderness over the canine fossa. Transillumination shows a shadow, and a roentgenogram shows a thickened membrane. Roentgenograms should be taken in all cases, including all the teeth on the affected side, as an acute empyema will become chronic if a dental cause is overlooked. It is not enough that the teeth have been recently examined by a dentist or that there may not have been pain in the teeth. Devitalized teeth have no pain. The etiology of many cases will be overlooked unless the dental films are taken by a dental surgeon who knows dental pathology.

On the other hand, the nasal mucosa may be red, and the soft tissues infiltrated, with but little if any discharge; or the vestibule may be obstructed by a thick, purulent secretion. If there is a dental cause the discharge is apt to be fetid. On shrinking the tissues with a 1 to 2 per cent. cocaine solution and freeing the nostrils of secretion, pus may again appear in the middle fossa when the head is held to the other side. If these measures are not successful, suction should be employed. In cases of bilateral empyema, transillumination is positive on both sides. That is, both sides may look alike, but the normal brightness beneath the orbit and the pupil-reflex are absent. In no case is it necessary to puncture and wash out the antrum to make a diagnosis of acute empyema of the antrum.

**Prognosis.**—The prognosis is good if associated infections in the other sinuses and teeth are carefully treated. If the drainage through the ostium is but little obstructed many cases undergo resolution, leaving a thickened lining membrane of the antrum. Neglected cases may become chronic.

**Treatment.**—This is an active infection in which the patient is toxic, with danger of some remote process like a chronic nephritis starting up, and, as soon as a diagnosis is made, the empyema should be treated like an inclosed abscess in any other part of the body by immediate and adequate drainage. A sizable opening in the antranasal wall provides for easy approach in the after-treatment. When the opening is small the swollen, lining membrane closes it almost at once, or the membrane may even be pushed ahead of the trocar.

Under gas-oxygen anesthesia introduce the trocar beneath the inferior turbinate, with the point directed upward close to the attachment of the turbinate. With a little pressure the antrum is easily entered if the opening is made in the antranasal wall about an inch from the end of the nose. If a Knowles trocar with a serrated face is used, enough of the nasal wall can be broken down to introduce a bone forceps of the Grunwald

type, or a Myles back-cutting forceps. With these enough bone is removed to give permanent drainage until the purulent process ceases. It is not necessary to sacrifice any of the inferior turbinate unless it is enlarged so that it rests on the floor of the nose, or has an enlarged anterior end. Under these circumstances it is better practice to remove the middle third of the inferior turbinate and enter the antrum through this opening with a Knowles trocar, or a chisel, or hand-drill of the Tilly type, as removal of the anterior end of the inferior turbinate, when a large opening has been made in the antranasal wall, is apt to be followed by the discharge of a large mass of secretion at unexpected times. If there is a dental cause, the diseased tooth should be extracted, and the necrosed bone in the floor of the antrum curetted through the tooth socket. The after-treatment consists in irrigations with warm normal salt solution. As long as the antrum is draining freely, after the secretion has been once thoroughly cleared out, this should only be done often enough to prevent retention. As the artificial opening in the lateral nasal wall is apt to close in a few weeks, it is important to reduce the swelling of the infiltrated tissues about



Fig. 67.—Knowles' antrum trocar.

the ostium by nasal treatment as advised in the case of chronic catarrhal maxillary sinusitis. Even where a purulent process becomes dry the changes in the lining membrane have advanced so far in many cases that the infected sinus is only quiescent and will be subject later to acute exacerbations indefinitely.

#### CHRONIC MAXILLARY SINUSITIS

**Pathology.**—In chronic maxillary sinusitis, as a result of the chronic inflammation, the mucosa becomes thickened, with an increase of the connective-tissue infiltration and the formation of pus or a mucopurulent secretion. In the more virulent infections the mucosa may show areas of ulceration, or the terminal process may result in a diffuse fibrosis. In these cases the discharge is serous, or gelatinous masses may appear after irrigation. The chronic inflammation of the mucosa, with its accompanying edema and irritation, may be followed by the formation of cysts, polypi, or hydrops, or involve the periosteal layer with the development of osteomata. Occasionally pus is found between the mucoperiosteum and the bony wall, or an osteitis is present.

**Symptoms.**—Clinically, the cases can be divided into three classes: *First*, those showing a thickened membrane and free pus. *Second*, those showing a thickened membrane with no pus, and *third*, those in which the connective tissue has undergone a diffuse fibrosis with infiltration.

**Chronic Maxillary Sinusitis with a Thickened Lining Membrane and Pus.**—**Pathology.**—Microscopically, the mucous membrane is generally thickened throughout as the result of edema of the deeper structures. "Adherent to the surface there are remnants of a purulent exudate. The epithelium is generally retained, but in places it may show considerable desquamation of the superficial strata and in some areas show more or

less reduction in the height of the columnar cells. When the membrane is entire the cilia are preserved, while in the deeper areas the cilia are missing. There are only occasional goblet-cells. There is slight infiltration of the epithelial layer with leukocytes, and the superficial part of the subepithelial connective tissue contains a considerable number of young connective-tissue cells, leukocytes, lymphocytes, and plasma-cells. In some of these plasma-cells two or three nuclei are not infrequent, making them practically resemble small giant-cells. The deeper portions of the section are more edematous, loose in texture, and contain more cellular elements. The capillaries show a moderate swelling of the endothelium. There is a fairly well-marked general hyperemia. The mucous glands are few in number and small. They are often apparently normal. In short,

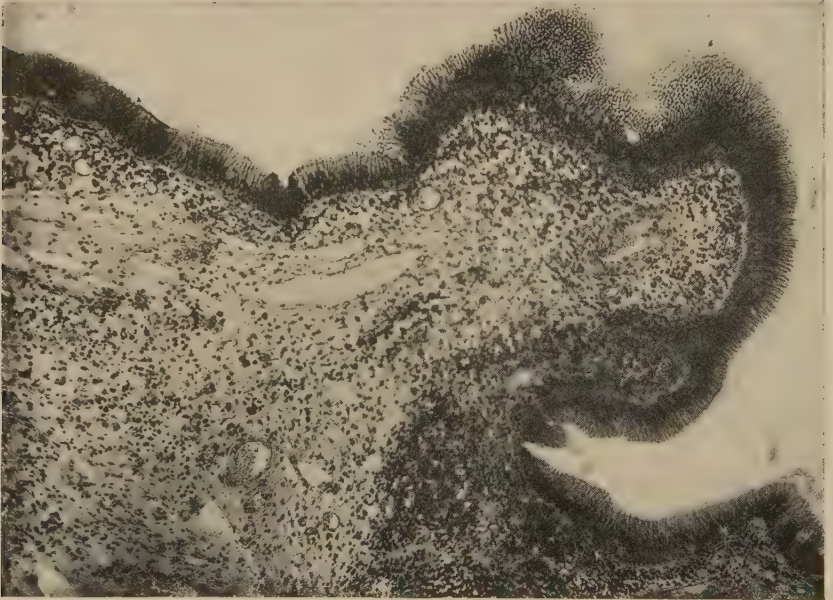


Fig. 68.—There is some desquamation of the epithelium in places, although, on the whole, it is intact and ciliae are present. The round-cell infiltration is marked in the superficial stratum of the tunica propria. These round cells are many small lymphocytes, a few plasma-cells, and some polymorphonuclears. There is a slight amount of edema beneath the epithelium and the blood-vessels show thickening with some perivascular infiltration.

this represents the late stage of a chronic inflammatory process with edema in the deeper layers and infiltration of the subepithelial layers. In the latter tissues there will be an increase of the small blood-vessels" (Tunis).

*Symptoms.*—With a thickened membrane and pus present, in the chronic form of maxillary sinusitis, there may be but few symptoms beyond the purulent nasal discharge and the pharyngeal irritation. With an exacerbation of the chronic process there is discomfort over the affected antrum, or even pain with an increase in the unilateral discharge, although marked pain is rare. The affected side of the head may ache or supra-orbital neuralgia may be present. In exceptional cases the supra-orbital pain may have existed for years. In the afternoon there may or may not be an increase in temperature of one or two degrees. The character of the

discharge varies from a fetid secretion, due usually to dental necrosis, to a mucopurulent or purulent type. If there has been a purulent discharge for some time, there will be dryness of the mucous membrane over which it passes. Such patients will often complain most of their throats, and the difficulty in keeping them free from thick secretion or crusts. Often there is a persistent cough on lying down and marked pharyngitis. The sense of smell, in the chronic cases of maxillary sinusitis, is not often disturbed unless there is an associated ethmoiditis or marked swelling of the middle turbinal that shuts off the upper vestibule of the nose. On account of the presence of pus the relation of systemic infections to the antral pathology is usually recognized early in the disease.

*Diagnosis.*—The diagnosis of chronic maxillary sinusitis with pus is, as a rule, not difficult to make. There is usually a history of thick secretion in the postnasal space, especially in the morning, and difficulty in removing it. The pharynx, from the discharge of pus and the constant hawking to clear it, is irritated. There may be a general or lateral pharyngitis present. Inspection by posterior rhinoscopy reveals thick secretion, or crusts, which may be confined to one side. The posterior ends of the turbinates may be swollen or show hyperplasia. Whenever there is a purulent discharge for a long time there is a dry and glazed appearance of the mucosa over which it passes. If but one antrum is involved, anterior rhinoscopy is apt to show a difference between the two sides in the color of the mucous membrane of the nares, except in elderly people, where the mucosa may be pale on both sides. The septum is usually deviated to the side affected; the middle turbinate may be close to the antronal wall; there is evidence of impaired drainage; and a chronic hyperplastic ethmoiditis is often present. So, also, crusts may be present or absent. Tenderness over the canine fossa and discomfort are not usual, except during acute exacerbations of the chronic process. Transillumination shows a shadow over the malar eminence and the roentgenogram will show a thickened membrane with free pus present. Diagnostic puncture gives us no additional information and is often misleading.

*Prognosis.*—If the pathology is confined to the lining membrane of the antrum, the improvement of drainage through the ostium by treatment of the nasopharynx (see Chronic Catarrhal Maxillary Sinusitis), together with a wide opening in the antronal wall, and the use of warm, saline irrigations, may be followed by a subsidence of the symptoms, resolution of the inflammatory products, and a cure, but this is rare.

*Treatment.*—In considering the treatment of chronic maxillary sinusitis we know, if the deeper structures have been involved, that there is but one way of effecting a cure, and that is by a radical antrum operation. Palliative measures are only justified for a short time, as there is danger of the other sinuses or some remote organ becoming involved.

**Chronic Maxillary Sinusitis with a Thickened Lining Membrane and No Pus.**—*Symptoms.*—When a thickened membrane is present in the maxillary sinus without the presence of pus, the symptoms are due to acute exacerbations which simulate a nasopharyngitis, but are apt to be more prolonged. Sometimes there is an uncomfortable feeling on the affected side, but the rule is that all subjective symptoms are absent. In the early stages there will be a history of a unilateral and purulent discharge which has become mucoid, and the nasal mucosa will be injected

on the affected side. There may be a normal temperature or it may be elevated one or two degrees. There may or may not be tenderness over the canine fossa on pressure. In the same way pain over the eye or side of the head may or may not be a prominent symptom. There is usually sensitiveness to palpation of the gland under the angle of the jaw. The roentgenogram shows a thickened membrane. On account of the absence of subjective symptoms and of free pus in the antral cavity, its relation to systemic complications is often overlooked. It is in these cases that a careful history, told in the patient's own way, will show over a long term of years that his so-called colds have been followed by bronchitis, myositis, arthritis, etc. This is important, for a blood-stream infection having already occurred, acute exacerbations of the chronic process in the antrum have been fol-

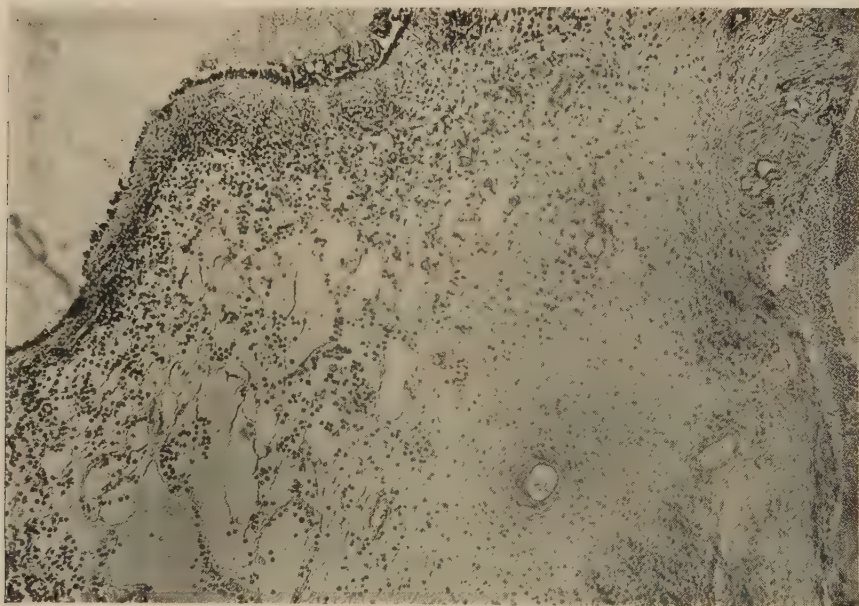


Fig. 69.—The epithelium shows thinning with desquamation. Ciliae are present. There is subepithelial infiltration of small round cells. There is some edema in the superficial portion of the tunica propria, and perivascular thickening with some round-cell infiltration. The glands are slightly active.

lowed by myositis, erysipelas, arthritis, endocarditis, glomerulonephritis, serous labyrinthitis, involvement of the gall-bladder, appendicitis, etc. These complications are more common in this form of sinusitis than when an empyema is present. If, following an acute exacerbation, there is a difference in pressure over the canine fossa some three or four weeks later, it is probable that a more active process is present in the mucoperiosteum, and such cases are almost sure to have serious complications whenever the patient's resistance is lowered, if such a history has preceded it. While a thickened membrane may exist in the antrum for years without symptoms, except those of repeated colds, if there is a history showing systemic infection, such pathology in the lining membrane may be followed by the most serious complications, that eventually may lead to chronic invalidism or death.

*Diagnosis.*—The diagnosis is based on the difference in the nasal mucosa on the affected side, with palpable glands and hyperplasia more marked on the same side of the neck. There is sensitiveness to pressure over the canine fossa during the acute phase. What is fully as important is a history not of the antrum but of the patient's life since childhood, showing the systemic infections following repeated attacks of nasopharyngitis. A roentgenogram shows a thickened membrane. What is often of more help is transillumination through the hard palate, by placing the light behind the rim of the orbit, as recommended by Briggs. Even in patients that have become partial invalids by reason of repeated infections with their

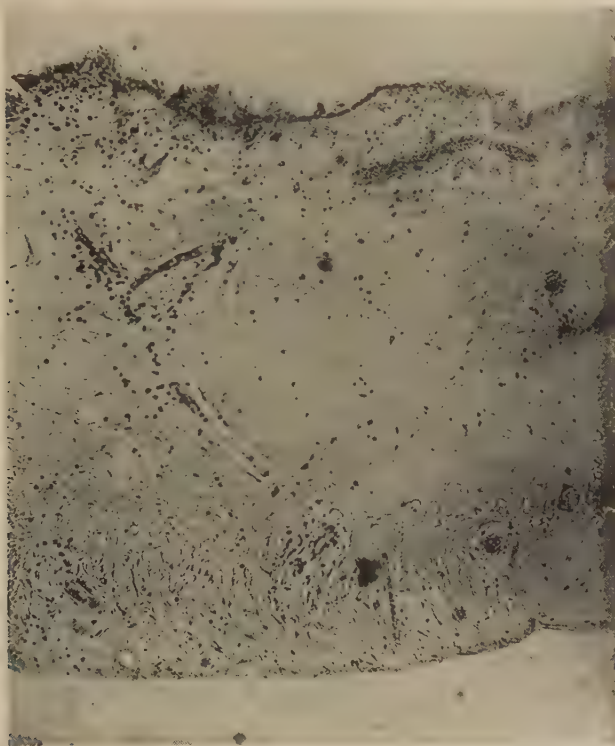


Fig. 70.—The epithelium is thinned. Round cells are slightly infiltrated through the entire section. There is perivascular thickening with tremendous increase of fibrous tissue. The glands are not active. Beneath the epithelium there is a slight amount of edema.

resulting complications, we will find on inspection, after an opening through the canine fossa, a perfectly smooth lining membrane of the antrum. If we take a probe and touch the surface the feeling is velvety and not hard, as we get in probing normal mucoperiosteum. There is also a difference in color—the surface is pink, as distinguished from the normal white lining membrane. The roentgenogram, clinical history, and examination should give us confirmatory evidence in coming to a positive diagnosis.

**Chronic Maxillary Sinusitis with Fibrosis and Infiltration.**—The next step in a chronic maxillary sinusitis, if allowed to go untreated, is that the antral connective tissue may undergo fibrosis with infiltration, with a symp-

tomatology that is characteristic but different from that accompanying a purulent discharge, in which a thickened lining membrane is found. The active symptoms are intermittent, and the disease is latent, continuing so indefinitely. Theoretically, the end-result of a long-standing chronic purulent process should represent a run-out infection. Clinically, a maxillary sinusitis with fibrosis and infiltration is associated with the worst types of systemic disease and the patients often become semi-invalids.

*Etiology.*—Diffuse fibrosis of the lining membrane of the antrum is the end-result of a long-continued, chronic, suppurative process. When a low-grade infection of the mucous membrane occurs, the tissue reaction is one of hyperplasia both of the mucous membrane and later of the connective tissue. A more active infection like that present in an empyema is followed by edema and irritation, with pronounced swelling of the mucous membrane and, in a certain number of cases, the mucous ducts become occluded so that the antral cavity may contain from one to a large number of cysts. Whether or not these cysts are always preceded by supuration is a disputed question, and there is much uncertainty as to the different steps which take place in their development. Clinically, they are found as a large mass in the floor of the antrum, or opposite the middle turbinal body, or as one or two large cysts. Less frequently any number of cysts may be found, either of large or small size, with the antral cavity full of serum or containing a gelatinous mass.

*Symptoms.*—These patients are usually free from symptoms until they have an acute exacerbation, which they consider a cold, and to which they are susceptible. The discharge, being serous, is not noticed. The recurrence of these symptoms may continue for years or, in exceptional cases, the acute phase may be accompanied by albumin in the urine and symptoms of general toxemia, as in the cases with a thickened membrane. Usually there is no pain over the affected antrum. General bad feelings in the face and vertex are often present, which the patient finds hard to describe. These cases are susceptible to changes in temperature or anything that irritates the mucous membrane. Trifacial neuralgia is not uncommon. While it is true that in any long-standing infection eye symptoms may be prominent from congestion of the mucous membrane or through the circulation, so in maxillary sinusitis with fibrosis we are especially apt to have a conjunctivitis, uveitis, opacities in the vitreous, etc. Vasomotor symptoms are often present with exacerbations of the local chronic condition, but between times they consider themselves free from any infection.

*Diagnosis.*—If we see the patient during an acute exacerbation there will be evidence of a low-grade infection of the nasal mucosa *on the affected side* with beginning atrophy. The discharge is unilateral and serous. The ostium is larger than normal from atrophy of the surrounding soft tissues. There is usually a lateral pharyngitis. During the acute phase, but not at any other time, there is soreness on pressure over the canine fossa, although there are exceptions to this rule, and the gland under the angle of the jaw is sensitive to pressure and may show hyperplasia. On washing out the antrum a gelatinous mass is often found, or only a serous fluid is present, or the lavage may be negative. Transillumination of the diseased antrum is more clear than normal. The roentgenogram shows a thinning of the mucoperiosteum and sometimes the presence of cysts, or a rarefying osteitis, or the Roentgen-ray report may be negative or state that there

is thickening on the normal side, from the contrast. Clinically, it is unusual to find the bone involved. The general history of the patient is as important here as in those cases with a thickened lining membrane in showing its relation to some remote complication.

*Prognosis.*—The prognosis is good if followed by proper surgical procedures.

*Treatment.*—No treatment is effective except a radical antrum operation. The entire lining membrane should be removed, otherwise it is not possible to prevent acute exacerbations in the future, as the mucoperiosteum is permanently damaged and often pus is found between the lining membrane and the bony wall. (Note: It is important to know what happens following the removal of the entire lining membrane. Knowlton, working in Mosher's laboratory, carried out a series of experiments on dogs. The first dog was killed at the end of two months and the cavity found covered with young fibrous tissue. The second dog was killed at the end of four months and the cavity found lined with fibrous tissue and covered with ciliated epithelium. A number of goblet-cells were present, but no glands. In the third dog, after five months, the glands were beginning to appear.)

*Operative Findings.*—On inspection, after an opening through the canine fossa, the mucoperiosteal lining may look smooth, or a cystic mass is found in the floor of the antrum, or opposite the middle turbinal body, or the cavity may be filled with serum. The whole mucoperiosteum is undergoing fibrosis, is thin, and shows chronic infection. Sometimes the whole cavity is filled with mucous cysts. While most cysts contain clear serum, occasionally one will contain mucopus tinged with blood. Cysts may be of the mucoid type, or contain a large amount of fibrous tissue, or become calcified.

#### MAXILLARY SINUSITIS IN CHILDREN

*Pathology.*—Is the same as in adults (Dean).

*Acute.*—Many observers have pointed out the relative frequency of acute purulent processes in the antrum in childhood. Many of these cases were discovered in a routine Roentgen-ray examination in an attempt to learn the cause of a systemic infection which was manifest as an arthritis, bronchitis, pyelitis, etc. Improved methods of diagnosis show that the relative frequency of maxillary sinusitis in childhood is about the same as in adults. In 234 routine examinations for diseased tonsils and adenoids in children thirteen years of age and under, 15 per cent. showed a chronic empyema of one or both antra (Dean). None had a positive Wassermann. These cases were also suggestive that the rapid recurrence of tonsil and adenoid tissue after removal might be due to sinusitis.

*Diagnosis.*—See diagnosis in adults. (In children the roentgenogram, to determine the size and relation of the antrum to the floor of the nares, is of great surgical importance.)

*Chronic.*—In a routine sinus examination of all children with a postaural abscess during one service at the Massachusetts Eye and Ear Infirmary, 15 per cent. showed Roentgen-ray pathology in one antrum (Emerson). In chronic maxillary sinusitis in children many cases have vasomotor symptoms and the Roentgen-ray findings show a thickened membrane usually without the presence of pus. Other cases may show the same symptoms or have the same systemic infections as are found in adults.

*Treatment.*—In acute cases removal of the tonsils and adenoids, with treatment of the nasopharynx to relieve the congestion and swelling about the ostium, is often successful. When this fails, puncture of the antro-nasal wall and washing out of the antrum are indicated. In chronic cases the technic advised by Dean gives the best approach to the sinus. The patient lies on the back and the pharynx is kept clean with the suction apparatus. Using a pair of blunt Knight's forceps the whole of the inferior turbinate is rotated outward and upward, the antro-nasal opening is then made, and any hypertrophied tissue removed or carious areas curetted. The inferior turbinate is then replaced in its original position, and the meatal opening always closes, leaving a normal-appearing nose. The after-treatment is the same as in adults. Sunshine and out-of-door life are very important. Some cases even after the cessation of discharge will again have profuse secretion on being confined indoors, which stops only when they can be in the sunshine again. Cod-liver oil is of distinct benefit.

#### ALVEOLAR OPERATION

The operation through the alveolar process was formerly quite generally employed. Its value is limited, however, to recent cases in which the antrum is infected as the result of a diseased tooth.

*Symptoms.*—Following pain in an involved tooth and in the antrum there suddenly appears a unilateral nasal discharge on the affected side or, if the tooth is devitalized, there may be no dental symptoms. A fetid odor may or may not be present.

*Diagnosis.*—The diagnosis is made in the same way as in the case of purulent maxillary sinusitis. If there has been no pain in the teeth the possibility of a dental cause should not be overlooked, even in those cases with obvious nasal pathology. In fact, films of the teeth should be taken as a part of the routine examination, for there is always caries in the antral floor in cases secondary to a diseased tooth.

*Treatment.*—If the roentgenogram shows a diseased tooth, this should be extracted at once, and the necrosed bone about its apex, involving the floor of the antrum, curetted, leaving a free opening for irrigation. If the discharge does not change in character, and cease, before the opening closes, we should not endeavor to reopen it, or introduce an obturator, but do a Caldwell-Luc or local Skillern operation at once. Only those cases in which the diagnosis is made early, or in which there is but little involvement of the floor of the sinus, will respond to treatment through the alveolar process. Even in these cases a counteropening in the antro-nasal wall, with removal of enough bone to prevent early closing, is the only safe procedure.

#### SKILLERN'S PRETURBINAL OPERATION

The Skillern preturbinal operation on the maxillary sinus was designed for those operators who prefer to approach the antrum from the nares and under local anesthesia. It is a modification of the Denker operation. This technic, as given by the author, embodies all the advantages introduced by Canfield, Ballenger, and other operators, and is less difficult:

1. Clean the nose with a warm saline.
2. Anesthetize the anterior attachment of the inferior turbinate above, below, and in front with a 20 per cent. solution of cocaine to which one-fifth its volume of adrenaline chloride has been added.

3. When anesthesia is complete, a solution of 1 per cent. novocaine to adrenaline chloride 1 : 10,000, and normal salt 98 per cent., is injected beneath the mucosa on the nasal side of the pyriform aperture, and subperiosteally on the facial side of the same structure.

4. A perpendicular incision is made slightly in front of and above the anterior end of the inferior turbinate, extending well down into the floor of the nose. This incision should sever all tissue down to the bone. A second incision is made directly back of this, meeting the first one above and below, so as to excise a spindle-shaped piece of mucous membrane.

5. Control hemorrhage with adrenaline tampons, and elevate the periosteum from the crista pyriformis, both externally, toward the canine fossa, and internally, toward the inferior turbinate, until a sufficient portion of the bone is exposed.

6. The antrum is now attacked with a chisel having a concave surface, applying it to the crista pyriformis, first above, and then below, removing the loosened bone with a pair of strong forceps. The opening is enlarged with an electric trephine, giving a smooth round opening.

7. The opening can be enlarged to any size with a curved frontal sinus rasp.

8. Flush out the sinus and, after drying, pack with a thin strip of gauze saturated in cocaine-adrenaline solution; allow it to remain five minutes.

9. Introduce an ordinary large rubber ear speculum into the opening, and inspect the antrum for polypoid degenerated mucosa, areas of granulation tissue, necrotic areas, etc. The nasopharyngoscope is used for inspection of the roof-lacrimal region and ostium.

10. With a curet remove all diseased and degenerated mucosa, inspecting the floor, the postero-inferior and antero-superior angles.

11. Irrigate and pack loosely with iodoform tape.

**After-treatment.**—Remove the gauze in forty-eight to seventy-two hours. If there is no secretion the gauze can be left six or seven days. On removal, irrigate and repack with iodoform gauze. Continue the treatment every second day for ten days to two weeks, when packing can be discontinued. Irrigation and insufflation can be continued at increasing intervals for about four weeks. The ordinary case can then be discharged, cured. The tendency of the artificial opening to close may necessitate curetting the edges of the opening or the use of caustics. After healing, the opening will gradually close.

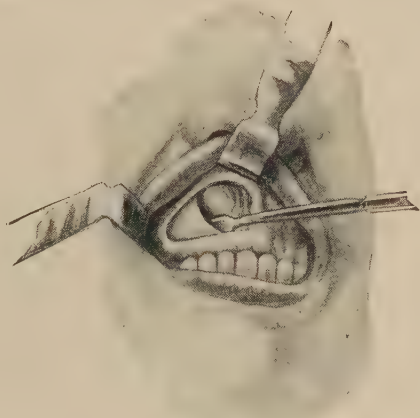


Fig. 71.—Denker's operation. Elevating mucosa and periosteum from nasal aspect of pyriform aperture. (Skillern, *Accessory Sinuses of the Nose*, J. B. Lippincott Co., Publishers.)

### THE RADICAL MAXILLARY SINUS OPERATION

(Caldwell-Luc)

**Indications.**—A radical operation on the antrum is indicated when there has been long-continued suppuration, with the deeper structures involved, or fibrosis, or when caries of the bony wall is present.

**Anesthetic.**—The anesthetic may be either local or general. The gas-oxygen-ether sequence minimizes the amount of ether used, and is preferred to local anesthesia by most operators.

**Preparation.**—A roentgenogram showing the size of the cavity and probable pathology, as well as films of the teeth, should be at hand and consulted before operating. Infected teeth should be extracted and the floor of the antrum curetted either before or at the time of the operation. Any associated sinus disease must be included in the operation. The gums and buccal mucous membrane should be cleansed, the skin about the face and nose scrubbed with soap and water, and followed by 70 per cent. alcohol.

**Operation.**—First, place a postnasal tampon in the epipharynx. After inserting the mouth-gag on the side opposite that to be operated, a long strip of gauze is placed between the teeth and cheek to prevent blood from draining into the pharynx. The lip and angle of the mouth are raised with retractors. An incision in the gingivolabial recess,  $\frac{3}{4}$  inch

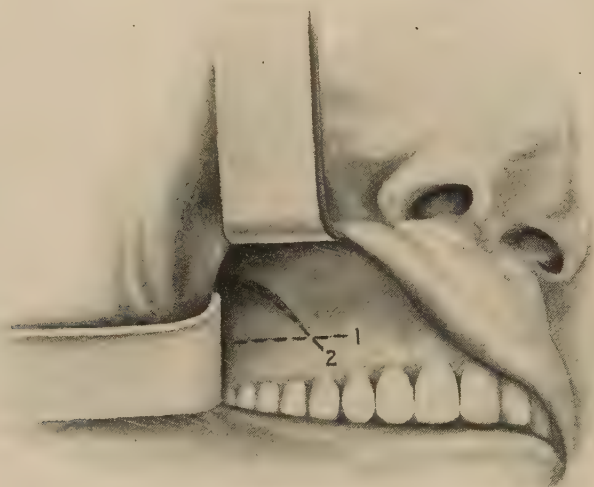


Fig. 72.—The mouth in position for the Caldwell-Luc operation: 1, Parallel line shows position and length of the incision which is carried inward only to the second bicuspid tooth to avoid the anterior dental nerves; 2, oblique Carmody incision for the same purpose. (Modified from Skillern, *Accessory Sinuses of the Nose*, J. B. Lippincott Co., Publishers.)

long, is made; but this should not extend farther forward than the second bicuspid tooth on account of the danger of injury to the anterior dental nerve supply. The periosteum is elevated, care being taken to avoid the infra-orbital nerve. With a chisel and mallet an opening over the canine fossa is made in the bone, and enlarged to about 2 by 4 cm. with a Kerison or other bone-cutting forceps. If pus is present this is removed with strips of gauze, and the cavity is thoroughly inspected. There is usually considerable bleeding, requiring the gauze wiping to be repeated several times, or an adrenalin pack to be used, in order to inspect the whole field. Cysts usually collapse with the gauze wiping.

The canine fossa opening should be large enough to make every part of the antrum accessible, including the anterosuperior angle. When the antrum has been the seat of a long-standing infection the lining membrane is

loose, especially in the floor, and can now be elevated by starting at the anterior wall, and after reaching the floor of the antrum a side-to-side motion is made until a large flap is free. This is grasped with the nasal forceps, and with slight traction and a curved elevator underneath much of the muco-periosteal lining can be removed intact.

We next make a counteropening in the antranasal wall beneath the inferior turbinate with a chisel. This is used through the canine fossa opening, and the bone is removed with Kerrison bone forceps down to the level of the floor of the nose. This opening should only be large enough to take care of the exudate during the process of healing, as we now know that the entire lining membrane is regenerated and the object is to leave the antral cavity as near normal as possible. No trauma has been made of the nasal mucosa, and no part of the inferior turbinate removed.

It is important now to pass a probe into the nose and beneath the inferior turbinate and see if the thickened membrane of the antranasal wall

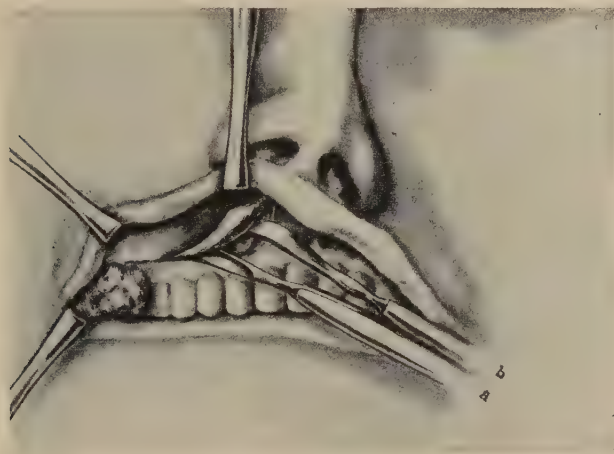


Fig. 73.—Exposure of the maxillary antrum through the outer antral wall by the Caldwell-Luc method: *a*, The line of incision of the mucosa and periosteum; *b*, elevation of the periosteum with periosteal elevator. (Bickham, Operative Surgery.)

has been removed, corresponding to the size of the opening which is to be permanent, so that it will not act as a valve.

In those cases in which the operator desires to reflect a flap of mucous membrane from the nasal cavity into the floor of the antrum, an incision is made in the membranous antranasal wall, corresponding to the opening made by the removal of the bone, and this flap is pushed to one side until the operation is completed. (The advantage of a flap is more theoretical than real. The disadvantage is that the thickened mucosa of the antro-nasal wall is reflected back one-half of the distance across the floor of the antrum interfering with drainage. The retained secretion bathes a chronically infected flap in a cavity that one is trying to free from infection.) With the counteropening in the antranasal wall extending well back it is unnecessary to remove the anterior end of the inferior turbinate, unless there have been hyperplastic changes in that body that have produced marked thickening, so that the turbinate rests on the floor of the nose. All rough edges of the opening should be made smooth to facilitate the removal of packing and provide free drainage.

**Dressing.**—A strip of gauze long enough to fill the antrum is thoroughly impregnated with vaseline, and one end carried through the canine fossa opening and out through the nose. The remaining gauze is then fed into the antrum. Two or three catgut sutures will then bring the wound together over the canine fossa opening. The gauze packing is left from twenty-four hours to three or four days, and acts to control hemorrhage and to set up a reaction that promotes healing. If the lining membrane has been removed the suppuration time will be shortened, one-third of the cavity obliterated, and the whole surface much more accessible to topical application. A firm compression pad is put on over the anterior wall and secured with a Bender bandage. This should be readjusted after recovery from the anesthetic. This helps to relieve pain and prevents swelling and discoloration beneath the eye if the pressure is firm above the malar bone.

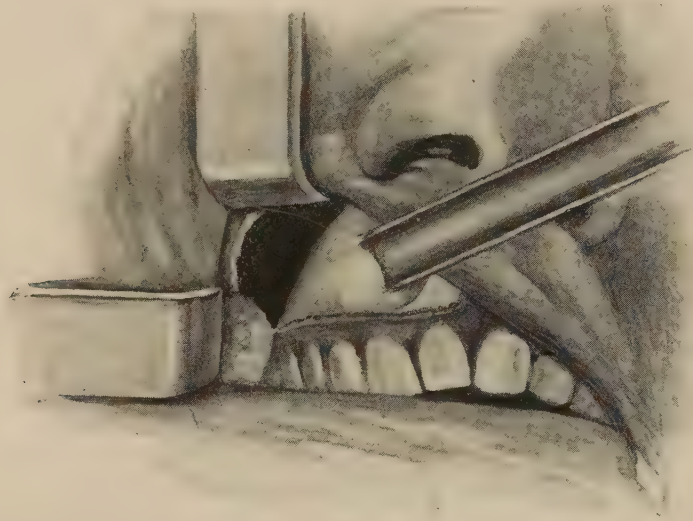


Fig. 74.—Caldwell-Luc operation. The mucosa of the right maxillary sinus removed, exposing the bare bone of the lateral nasal wall. The chisel in position to remove the bony wall without injuring the nasal mucosa. (Skillern, *Accessory Sinuses of the Nose*, J. B. Lippincott Co., Publishers.)

**After-treatment.**—The patient is put to bed and, if an enema preceded the ether anesthesia, no orders will be necessary for the night except morphine subcutaneously,  $\frac{1}{6}$  grain, if necessary. Most patients vomit once on recovery from an anesthetic, so that the stomach is emptied of blood. The after-care should be directed to the comfort of the patient. No direct treatment of the antrum is necessary. Frequent washing opens up a large surface for absorption, promotes flabby granulations, prolongs the discharge, and interrupts the healing process. If drainage is free it is unnecessary unless caries is present, except as indications arise, such as pain, temperature, or offensive discharge. The cavity can be inspected with the nasopharyngoscope, and any partitions, or exuberant granulations, treated. If the cavity is not disturbed by irrigations there will be but little tendency for these to form. Albolene with menthol, 2 to 4 grains to the ounce, should be used in the nose every four hours to protect the nasal mucosa, promote the flow of mucus, and keep the drainage free. The anterior vestibule

should be protected with sterile vaseline to prevent hair-follicle infection and gumming of the discharge at the entrance to the nose. The sinus can be gently wiped with a cotton-tipped applicator dipped in 10 to 20 per cent. argyrol if bands tend to form.

**Prognosis.**—If the surgeon has carefully removed all associated disease in the alveolar process, ethmoid labyrinth, and frontal sinus, the prognosis is very good, and recovery usually rapid and uneventful.

**After-effects of the Radical Maxillary Sinus Operation.**—Anesthesia of the cheek will occur if the initial incision is extended too far toward the middle line, injuring the anterior dental nerve-supply. Infra-orbital neuralgia results if the periosteum is elevated too high and the infra-orbital nerve injured. Permanent fistulæ sometimes follow when the wound is not carefully approximated, but this should be a rare accident. The patient should report for treatment not only until the discharge stops, but until all infection clears up. This has been known to persist from two months to two years, as shown by the return of systemic symptoms with surface irritation and no perceptible discharge. Irrigation makes the patient worse by increasing the congestion, and the cavity should be wiped out with a cotton-tipped bent applicator dipped in a saturated boric and alcohol solution, 10 to 20 per cent. argyrol, or varying strengths of silver nitrate.

#### CYSTS—DENTIGEROUS, PERIODONTAL

**Etiology, Pathology.**—*Dentigerous Cysts.*—These may be due in the first place to unerupted teeth; or in the second place to disturbed development during the embryonal stage. In these cases fully developed teeth may be found free in the cyst cavity.

*Periodontal Cysts.*—These cysts are due to infection about the root of a tooth, and arise from inflammatory changes in its lining membrane. As the result of necrosis at the apex of a tooth a small cyst forms, which subsequently becomes infected. If the tooth is extracted before distention of the cyst takes place the cyst is often brought away intact. Retention of the inflammatory products results in dilatation and pressure-absorption of the surrounding bone. As the lining membrane continues to secrete in a closed cavity the cyst grows in size until it projects into the antrum, pushes the lateral wall of the nose to one side, or ruptures into the antrum, nose, or mouth. The contents of the cyst may be of a straw or brownish colored fluid, containing cholesterine, blood, or pus.

**Symptoms.**—In the early stages all symptoms may be absent or there may be local soreness. As the cyst increases in size and bony absorption takes place, its osseous covering may become very thin or entirely absorbed. Even at this stage the growth has been so insidious that there may be but little pain. Extreme distention of the walls of the cyst is followed by rupture into the antrum, nose, or mouth and, if pus is present, the discharge is often very foul and profuse. If the discharge is into the antrum all the symptoms of purulent maxillary sinusitis are present.

**Diagnosis.**—In the absence of any external swelling, when pus is present in the nose, the diagnosis lies between suppuration of dental origin or primary purulent maxillary sinusitis. The offensive odor points to dental necrosis. This is further confirmed by films of the teeth, which show a bone abscess. Transillumination is negative. If rupture has not taken place a roentgenogram may show a distinctly round tumor in the antrum, or a bony

swelling of the antral wall may suggest a cyst. This may be confirmed by a feeling of crepitation on palpation. It may be noticed that the lateral wall of the nose is pushed inward or the roof of the mouth shows a swelling. If the cyst has ruptured into the mouth no communication will be found with the nose. If films of the teeth are taken as a routine in all nasal suppurations, the diagnosis cannot be wrong.

**Treatment.**—While small cysts of the alveolar process, which have not ruptured, may be cured by extraction of the offending tooth and removal of the cyst wall, most cases will require a radical operation through the canine fossa with obliteration of the cyst cavity. At the same time a counteropening should be made in the antranasal wall.

#### ASPERGILLUS OF THE MAXILLARY SINUS

**Occurrence.**—Aspergillus of the maxillary sinus is very rare. Skillern reports one case, and found four others in the literature.

**Symptoms.**—The symptoms are vague, consisting of a unilateral, non-fetid discharge. There is a feeling of fulness over the antrum and indefinite head pains. There is no postnasal discharge. Skillern found a slightly congested lateral wall, with the rest of the nasal cavity normal. A needle puncture of the maxillary sinus offered considerable resistance to the introduction of a normal saline solution. Repeated washings were followed by a slightly turbid fluid. Subsequent washings brought away inspissated masses resembling cottage cheese. The lavage was continued until the return flow was clear.

**Histological Findings.**—The histological findings showed a close mycelial network, with occasional conidiophores, surmounted by a face-like arrangement of conidiophores. The hyphæ took a faint pink stain, but the fructifying bodies were yellowish in color, apparently resisting the penetration of the dye.

**Diagnosis.**—The diagnosis was aspergillus, probably of the species *Aspergillus fumigatus*.

#### SKIAGRAPHS

Skiagraphs are very reliable in diseases of the antrum, when pus is present, or when there is a thickened mucous membrane. We should exercise care, however, in having the light just strong enough to bring out the shadow, and not shine brightly through the anterior wall. The thickness of the wall varies greatly, even in adults, but a 2 candle-power light is strong

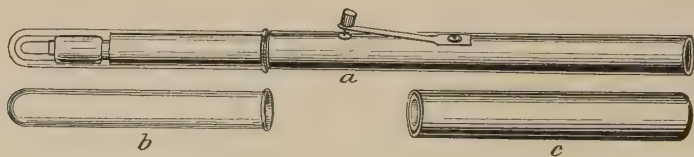


Fig. 75.—Coakley's transilluminator: *a*, Apparatus assembled for transillumination of the antrum; *b*, glass hood for use in transillumination of the antrum; *c*, hood for use in transillumination of the frontal sinus. (Morrow.)

enough for the average case. When fibrosis has taken place in the lining mucous membrane of the sinus, the whole side of the face is too bright and the normal outline is lost. This may occur even when the cavity is full of cysts or when the sinus wall has been made thin by a mucocele. Nor-



Fig. 76.—Transillumination of the maxillary antra (antra of Highmore). *Right* side healthy, as shown by bright illumination underneath the orbit, and through the pupil. *Left* side diseased. (Phillips "Diseases of the Ear, Nose, and Throat," F. A. Davis Co., Publishers.)



mally it should be light about the angle of the nose, then show a shadow over the malar eminence, and then a bright area under the eye, with the pupil-reflex present. When pathological changes have taken place and the lining membrane is thickened, the diseased side shows a shadow, except at the angle of the nose and the eye-reflex is lost. Placing a small light behind the rim of the orbit, and looking at the hard palate through the open mouth give us valuable information in regard to the floor of the antrum (Briggs). A shadow may be present in a quiescent process when there is only a thickened membrane.

Transillumination should be carried out in a very dark room. Having first ascertained that the patient does not wear a denture, the Coakley light is placed in the mouth and the lips closed. Having a suitable rheostat the light is turned on very gradually and the two sides compared. Like most accessory aids, however, skiagraphy should be used to confirm, and not to make, a diagnosis. There may be a normal difference in the anatomical thickness of the bones of the antra. In other words, positive findings are a distinct help, while negative findings may have to be ignored.

#### ROENTGEN RAY

The roentgenogram, while not absolutely necessary, is another aid in diagnosis. It shows the condition of the lining membrane of the antrum, whether or not it is thickened, undergoing fibrosis with infiltration, or pus is present. With it we differentiate between pus and thickened membrane, and between pathology and an abnormally small antrum. In cystic conditions the diminished density of the bone is apparent, as well as the increase in density in cases of empyema. The findings are more dependent upon the thickness of the membrane than upon the presence of pus, and may, therefore, be deceptive when the antrum is acting as a reservoir or when the membrane is not appreciably thickened. In addition to these findings we can determine the presence of partitions, a mucocele, or tumor, or a rarefying osteitis in the bony wall. If we look upon the roentgenogram as an aid in diagnosis, to be considered with our other findings, and not as an indication for operation, it is a valuable adjunct. It is distinctly of more help when one side is involved. In chronic cases the roentgenogram may be negative when transillumination leads us to suspect involvement of the antrum. A good observer, taking the findings as a whole, will be able to interpret this evidence correctly.

It is of distinct help to have pictures taken in two positions, one in the anteroposterior, and one in the Water position, in which the head is tilted back to cut out the shadow of the bones at the base of the skull and the petrous portion of the temporal bone. Of the two, the Water position is the more helpful, as it will often show pathology in the antral floor when the anteroposterior picture is negative. A roentgenogram is indispensable in determining the size and contour of the antrum before operating. It is particularly important for showing the relation of the antrum to the floor of the nose in the case of children, as the alveolar process of the maxilla is not fully pneumatized by the recess of the maxillary sinus until the time when the permanent teeth have erupted (Shaeffer).

### TUBERCULOSIS OF THE MAXILLARY ANTRUM

Tuberculosis as a primary cause of maxillary empyema is very rare. Examination of the literature shows very few cases, if any, that are not open to doubt as to the diagnosis. The fact that, clinically, so few cases are found, and also the additional fact that patients dying of tuberculosis do not show an unusual percentage of cases of empyema of the antrum, further confirm our belief that the antrum is invaded from contiguous adjacent tissue, or is secondary to a bone lesion. The antrum may first be filled with granulations, which subsequently break down.

**Diagnosis**—The tuberculous nature of the empyema can only be determined from a microscopic examination of the curetted tissue, taken from the lining membrane, in which giant-cells are found, with evidence of degenerative changes in the membrane. This is further confirmed by finding tubercle bacilli having the typical morphology and taking the characteristic stain.

**Treatment**—The treatment is the same as for non-tuberculous empyema.

### SYPHILIS OF THE MAXILLARY ANTRUM

Syphilis, like tuberculosis, is rarely primary in the antrum. As is well known, syphilis has a predilection for the intermaxillary bone and for the lamina papyracea of the ethmoid. Its origin is generally secondary to a bone lesion, usually, in these regions.

**Diagnosis.**—The diagnosis is made from the Wassermann reaction, and from the patient's response to specific treatment.

### OSTEOMYELITIS OF THE MAXILLARY BONE

Osteomyelitis of the maxillary bone is due to pyogenic organisms that are secondary, as a rule, to dental necrosis. The antral invasion in osteomyelitis, therefore, is a secondary process, as is syphilis and tuberculosis.

FRANCIS P. EMERSON.

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### SINUSITIS IN CHILDREN

The age of childhood is considered to be from birth to about twelve years—age of puberty.

In considering the paranasal sinuses of this period of life the variation of the sinuses as to existence, development, and position divides the period about in the middle.

At birth the ethmoid cells are the only sinuses always present and occupying their permanent position and relations to other fixed parts of the head, *e. g.*, the brain and eye; the frontal sinus is potentially present in the anterior part of the ethmoidal tract; the antrum is present on the nasal side of the orbit; the sphenoidal sinus may or may not be present in recognizable size. At the middle of the childhood period the frontal sinus has progressed sufficiently to be partially in the vertical portion of the frontal bone. The antrum as the child grows older develops laterally outward behind the tooth buds of the permanent teeth, and as they descend it develops anteriorly to occupy the space formally occupied by them. It is

settled as to position during the latter part of the childhood period. The sphenoidal sinus if not present at birth, develops sufficiently rapidly to be recognized as a sinus by the end of the first or early in the second year.

The student should know that the above conclusions as a rule are true, but not invariably so, as the development of the sinuses in some heads may

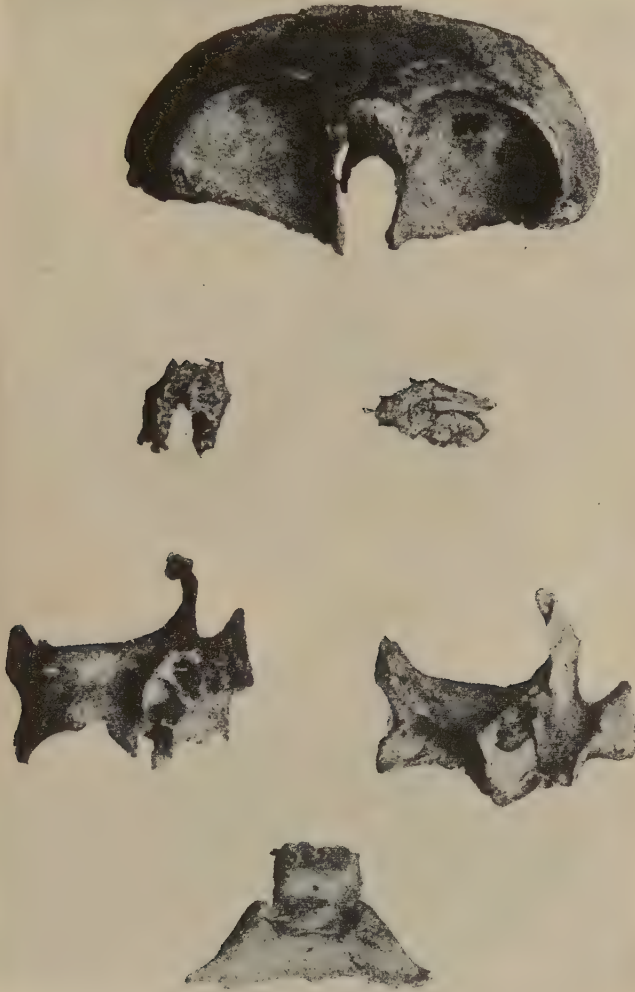


Fig. 77.—Sinus-bearing bones of a seven-month fetus. At the top is the frontal bone with the nasal bones in articulation, below and to the left is the ethmoid body or bone, and to the right is the lateral mass of the ethmoid of another seven-month fetus, the mass is wrong side up. On the third line are the two maxillary bones, the one on the left showing the large size of the tooth sockets, the one on the right showing the nasal aspect of the bone. The antrum is seen just back of the nasal process of the maxillary bone on the ethmoid side of the orbit. At the bottom is the sphenoid bone minus the greater wings.

be quite precocious, whereas in others the development may be tardy. No attempt to discuss the embryology of the sinuses is here made.

About 1900 I was much perplexed on account of several children who were brought to my clinic suffering from undoubted sinus disease, empyemic

*F. b.**E. c.**M. a.**S. s.*

Fig. 78.—*F. b.*, frontal bone. *E. c.*, ethmoid cells. *M. a.*, maxillary antrum. *S. s.*, sphenoidal sinus. External wall of the right nasal fossa of a child one year of age, all turbinated bones have been removed and the opening into the antrum enlarged. Note size of ethmoid cells, antrum, and sphenoid sinus.

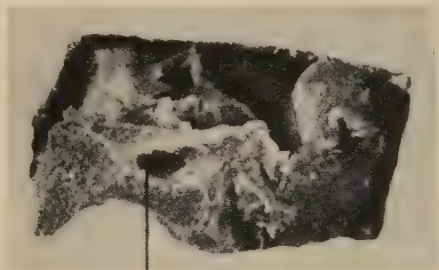
*S. s.*

Fig. 79.—*S. s.*, sphenoidal sinus. The sphenoidal sinus of a child one year of age, coronal section having been made just in front of that sinus.

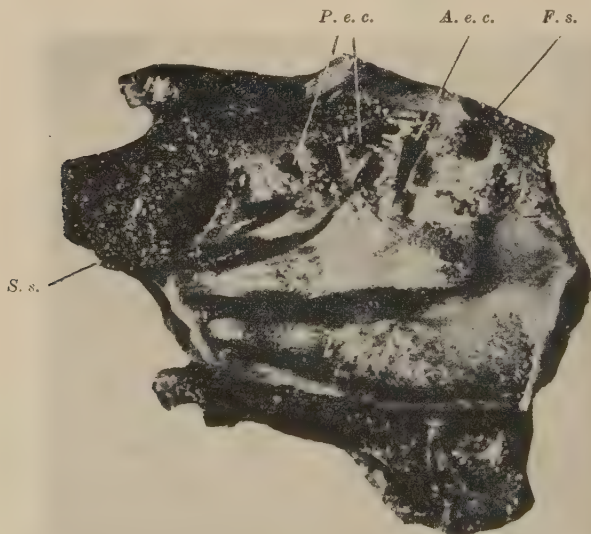
*P. e. c.**A. e. c.**F. s.**S. s.*

Fig. 80.—*F. s.*, frontal sinus. *A. e. c.*, anterior ethmoidal cells. *S. s.*, sphenoidal sinus. *P. e. c.*, posterior ethmoidal cells. The external wall of the left nasal fossa of a child three years of age. Ethmoid cells have been uncapped, the middle and inferior turbinals are in position. Frontal sinus appears at the upper right-hand corner. Note size of sphenoidal sinus.

in character, and evidently needing operation for relief. My perplexity arose from the fact that there was practically no literature on the diseases of the sinuses in children, as well as the vague and divergent statements of

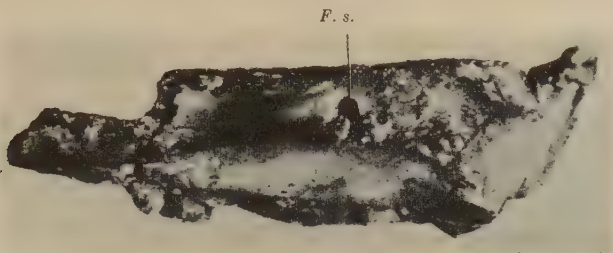


Fig. 81.—*F. s.*, frontal sinus. Horizontal section through the frontal bone of the same head as shown in Fig. 80, showing the frontal sinus extending into the vertical portion of the frontal bone



Fig. 82.—*S. s.*, sphenoidal sinus. The same head as shown in Figs. 80 and 81.

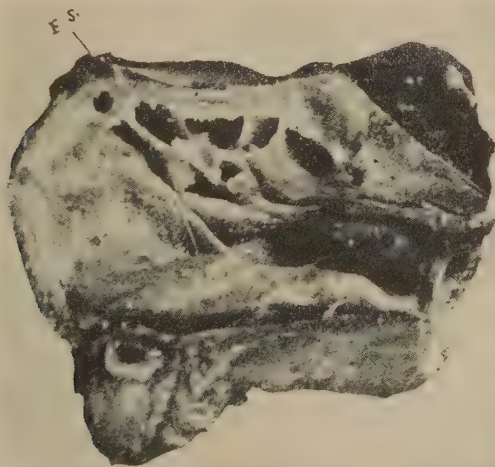


Fig. 83.—*F. s.*, frontal sinus. Head of a three-year-old child, the middle and superior turbinates have been removed, all the sinuses are present. Note the opened bulla and hiatus semilunaris.

anatomists as to the existence, time of appearance, and development of these cavities. I determined to get my lesson from Nature's Hand-book—the dead child's head.

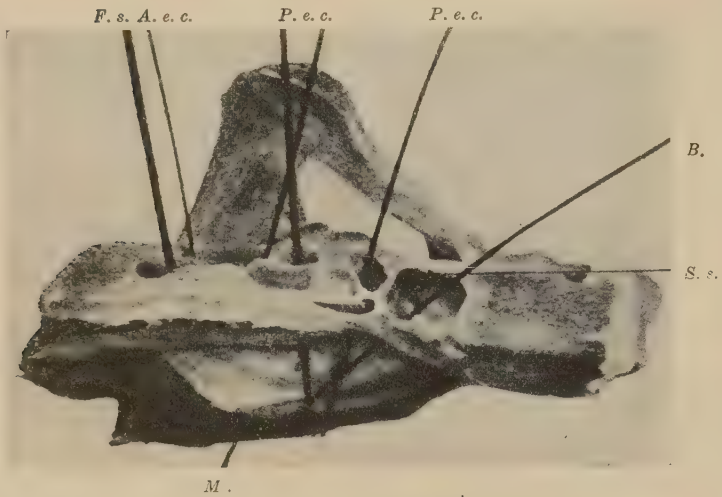


Fig. 84.—*F. s.*, frontal sinus. *A. e. c.*, anterior ethmoidal cells. *P. e. c.*, posterior ethmoidal cells. *S. s.*, sphenoidal sinus. *Mt.*, middle turbinated bone. *B.*, bristle from *S. s.* to middle meatus of nose. The external wall of the right nasal fossa of a child three years and eight months of age. Horizontal section has been made through the ethmoid region in order to uncup these cells and the sphenoid sinus, the bone is tilted so as to show the ethmoid cells from the top as well as the nasal fossa. Bristles passed through the frontal sinus and anterior cells emerge in the nasal fossa external to the middle turbinated bone, while those passed through the posterior ethmoidal cells and the sphenoid sinus emerge on the median side of the middle turbinated, or between it and the septum. Pus from these various cavities enters the nasal fossa by the same routes.

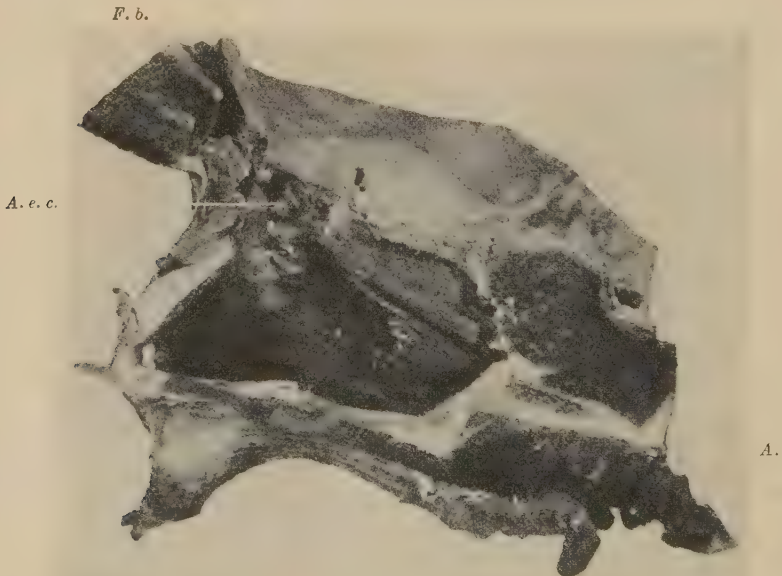


Fig. 85.—*F. b.*, frontal bone. *A.*, adenoid mass. *A. e. c.*, anterior ethmoidal cells. The right nasal fossa through the fenestrated septum, the anterior half of the middle turbinated bone has been removed, a coronal section has been made through the vertical portion of the frontal bone and the flap turned outward. There is no frontal sinus, the ethmoidal cells are poorly developed. Note the large mass of adenoids. The specimen is from a child seven years of age.

About twenty heads, some duplicates as to age, were studied, and from such study the foregoing conclusions were drawn and published in 1904.

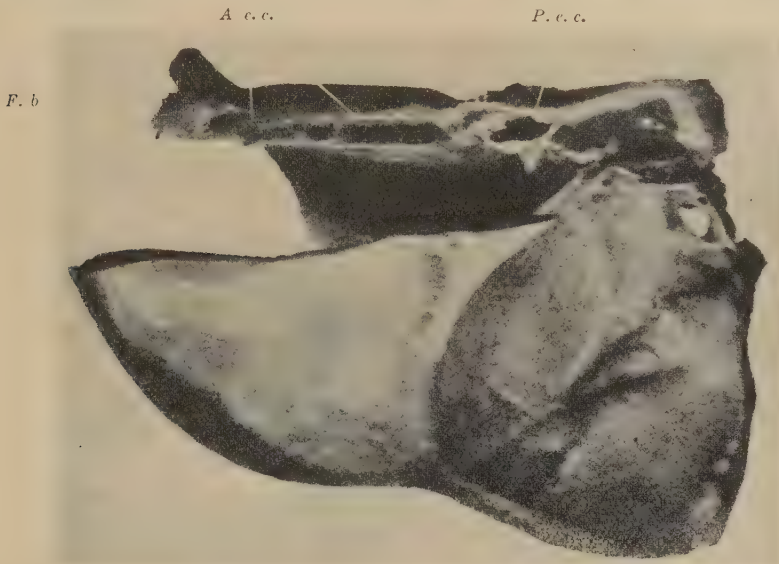


Fig. 86.—*F. b.*, frontal bone. *A. e. c.*, anterior ethmoid cell. *P. e. c.*, posterior ethmoid cell. Horizontal section through the ethmoid region of the left side of the same head as shown in Fig. 85.

This was the first article published on the subject and I take pleasure in presenting the photographic records of the work.

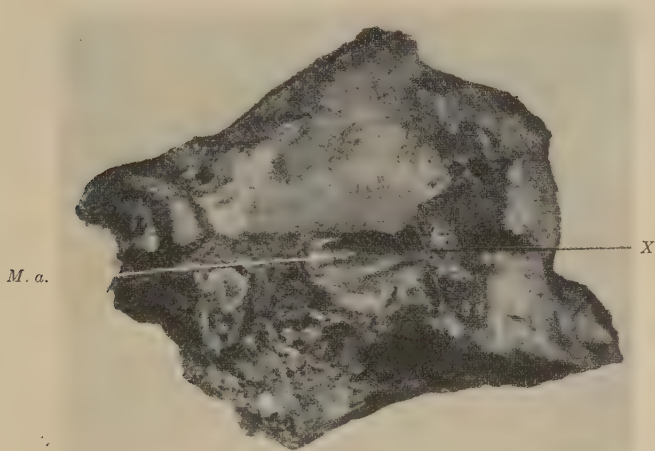


Fig. 87.—*M. a.*, maxillary antrum. The antrum of a child three years of age, the external wall of the cavity having been removed. *X* indicates the top of the tooth socket of the second or permanent tooth. The picture shows the impossibility of entering the antrum through the canine fossa without the destruction of one or two of the permanent teeth.

The classification of sinus disease, its etiology, pathology, symptoms, diagnosis, treatment, and so forth, has been discussed in the section headed General Considerations of Accessory Sinus Disease. In this section com-

ment will be made under the above headings only when it seems to be pertinent on account of the fact that we are discussing sinusitis in children.

**Etiology and Pathology.**—Children frequently have adenoids. Adenoids give rise to obstructed breathing and conduce to retention of nasal secretions which by contact with nasal tissues give rise to irritations and inflammatory conditions known as colds, rhinitis, or coryza. These inflammatory changes in the lining membrane of the nose very frequently extend by continuity into the accessory sinuses and thereby give rise to the simplest form of sinusitis—the catarrhal. Adenoids further deprive the child of nature's forces for nasal and sinus cleaning, *i. e.*, nose blowing, sniffing, and hawking.

The pathological changes taking place in the lining membrane during a catarrhal attack are to an extent permanent, and if often repeated the mem-

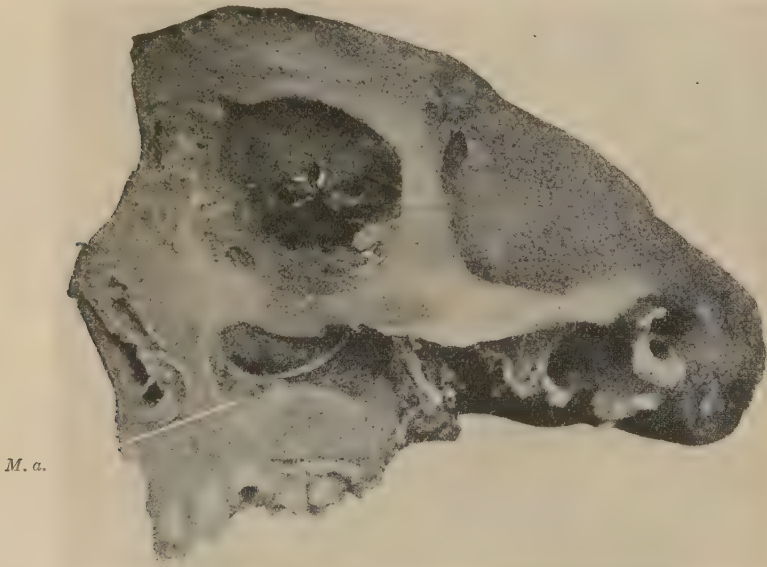


Fig. 88.—*M. a.*, maxillary antrum. The external surface of the superior maxillary bone of a child nine years of age. The external wall of the antrum has been removed, leaving the mucous membrane lining *in situ*. This illustrates how much greater space is had for operating on the maxillary antrum through the canine fossa after the descent of the second or permanent teeth.

brane is liable to become quite pathological. The changes are those of sclerosis. Marked changes in the circulation take place, as a result of which the membrane may become either hyperplastic or atrophic according as to whether the venous or arterial circulation is most affected. In any diseased membrane there may be both atrophic and hyperplastic areas. At any stage of these inflammatory changes infection from the activity of any of the pus-producing organisms may take place when the severity of symptoms and the character of the discharge will depend upon the virulence of the infecting organisms and the resistance of the child. An element of much importance in any particular case will be the patulency of the outlet or ostium of the affected cell or sinus.

If the ostium be small or become closed on account of the swollen lining membrane there occurs a closed empyema which may often result in severe symptoms on account of pressure as well as rupture of cell or sinus walls.

Children are frequently, in fact quite universally, sufferers from the various exanthemata and diphtheria. The sinuses are frequently coincidentally diseased.

**Symptoms and Signs.**—At such ages as children are able to describe their symptoms, symptoms are quite the same as those of the adult, but in younger children we must look for signs rather than symptoms. The signs of the catarrhal type of sinusitis are the open-mouthed breathing, the discharging nose, alar eczema, and cough. There may be an asthma. In the empyemic form of the disease the nasal discharge is more purulent in character; there may be general swelling about the eye, or protrusion of that

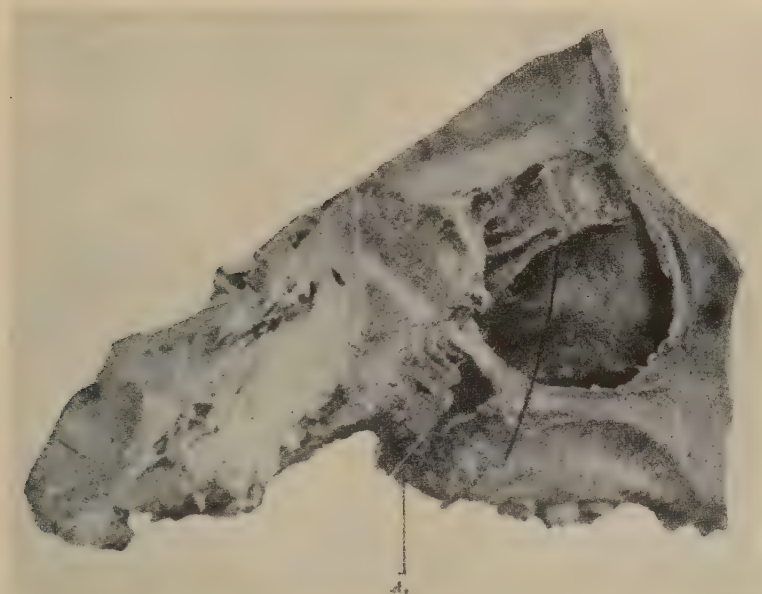


Fig. 89.—A., adenoid mass. The external wall of the left nasal fossa through the fenestrated septum of a child nine years of age. The middle turbinated bone and the anterior half of the superior turbinated bone have been removed. The bristle seen passes through the infundibulum into an anterior ethmoid cell. There is no frontal sinus. Note the enormous adenoid mass in the vault of the nasopharynx; all the sinuses in this child were poorly developed. In fact, from the heads examined I concluded that the sinuses were poorly developed in children who suffered from obstructed nasal respiration. Any and all of the sinuses shown are liable to disease. They are also amenable to treatment.

organ, or evidence of rupture through the frontal or ethmoidal wall into the orbit. Swelling about the eye involves more particularly the upper lid which aids in differentiating sinus disease from dacryocystitis which is accompanied by swelling of the lower lid; conjunctivitis, keratitis, and affections of the optic nerve are other symptoms of paranasal sinus disease not always recognized as such.

Children swallow nasal and sinus discharges giving rise to gastro-intestinal disturbances and pharyngitis. I have seen every one of these evi-

dences of the disease in young children. There may be instead a general swelling of one or both sides of the face in cases in which the disease has not been confined to the sinuses proper, but has attacked the bony walls of the diseased cells or cavities and has extended as an osteomyelitis to the neighboring bones, especially to the maxilla. This form of disease generally points just under the eye. My feeling is that these cases most frequently have their origin in one or more of the tooth sockets, which may be the result of either direct infection or trauma.

A child suffering from empyema of one or more sinuses runs in the acute stage of considerable fever not infrequently of a septic character.

**Diagnosis.**—The diagnosis of sinus disease in children should be a fairly easy matter to him who has well fixed in his mind the above symptoms and signs of the disease. The Roentgen-ray picture is of great help, and, when confirming diagnosis made from clinical observation, is of much comfort. Tooth-buds are not entirely opaque to the Roentgen ray. Because the bones of children are thin and the lining membrane of the sinuses is not thick from former disease the Roentgen-ray picture of the child's head is more dependable than that of the adult. Other valuable aids in diagnosis are suction, transillumination, and the pharyngoscope.

In doubtful cases and especially in those cases in which disease in other parts of the body points to the sinuses as a possible source of focal infection, the method of Dean, applicable especially to disease of the antrum, is the most satisfactory. Through a sterile cannula introduced into the antrum he inserts a fine needle connected with a small syringe, containing a sterile salt solution. The salt solution is carefully introduced into the antrum and withdrawn into the syringe. The withdrawn fluid is now subjected to bacterial study which is of great help in and may determine diagnosis.

This excellent procedure demands a high degree of technical skill as well as good hospital and laboratory facilities.

A child suffering from a chronic purulent sinusitis may suffer from frequent exacerbations. Parents complain of the child not thriving, of constantly taking repeated colds from which there is a considerable run of septic temperature. The child is irritable and cross, and if old enough complains of headache. There may be nausea and vomiting. In several cases I have seen severe attacks of convulsions. These cases have not infrequently been diagnosed as malaria, recurring influenza, pneumonia, or brain tumor.

**Complications and Sequelæ.**—L. W. Dean of Iowa City has demonstrated beyond question that a diseased sinus may be the focus of infection giving rise to various orthopedic diseases as well as other disorders arising from focal infection and, what is quite as important, he has shown that the successful treatment of the diseased sinuses is followed by the relief of those disorders.

Meningitis or brain abscess may be a serious complication, as may also optic neuritis. Malposition of the eye may result from the necrosis of the orbital wall of either the frontal sinus or an ethmoidal cell. Serious eye troubles may arise from the phlebitic condition of the ethmoidal and ophthalmic veins. The ethmoidal veins empty into the ophthalmic, which empties into the cavernous sinus.

Figure 90 shows such a case in a child one and one-half years of age. Figure 91 shows the Roentgen-ray picture of the child's head showing in-

volvement of all the sinuses, but the greater involvement on the left side. The child died. An autopsy was done and such pathological findings as are pertinent will be shown after a study of the following diagram. Figure



Fig. 90.

92 is a diagrammatical sketch illustrating the relationship of the ethmoidal and ocular circulation. Spaces bounded by dark lines represent the various sinuses, the large shaded lines the ophthalmic veins expanding into the

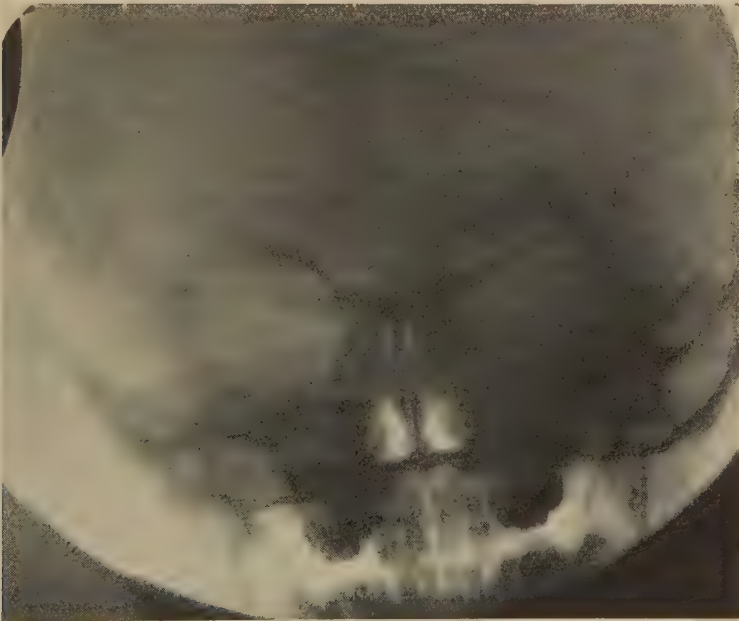


Fig. 91.

cavernous sinus. In the ethmoidal region the small ethmoidal veins may be seen as tributaries to the ophthalmic vein.

Figure 93 shows a microscopic section through the ethmoidal region. The broad white streak through the picture is a section of an ethmoidal

cell wall. Beneath it, in the middle of the picture, will be seen a thrombosed vein.

Figure 94 shows a microscopic field through the orbital tissue showing thrombi in a longitudinally cut vein.

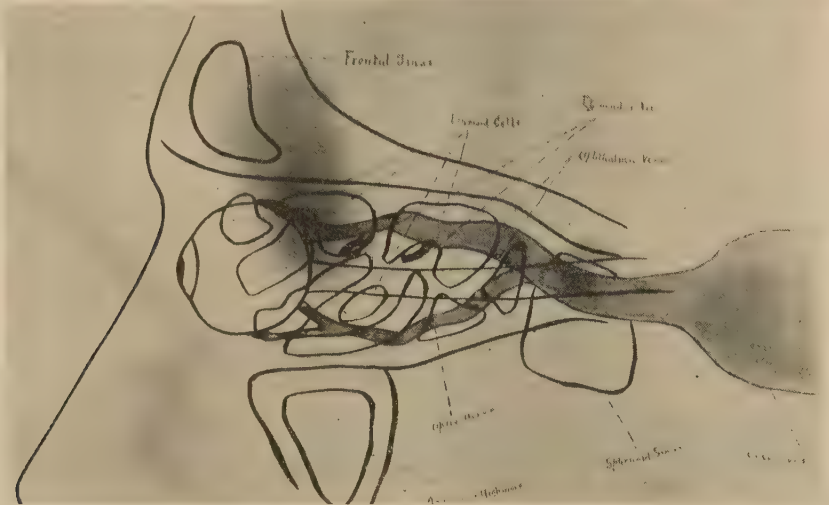


Fig. 92.

Figure 95 shows a fairly organized thrombus in the central vein. Such a pathological condition in a living head must be accompanied by a consider-

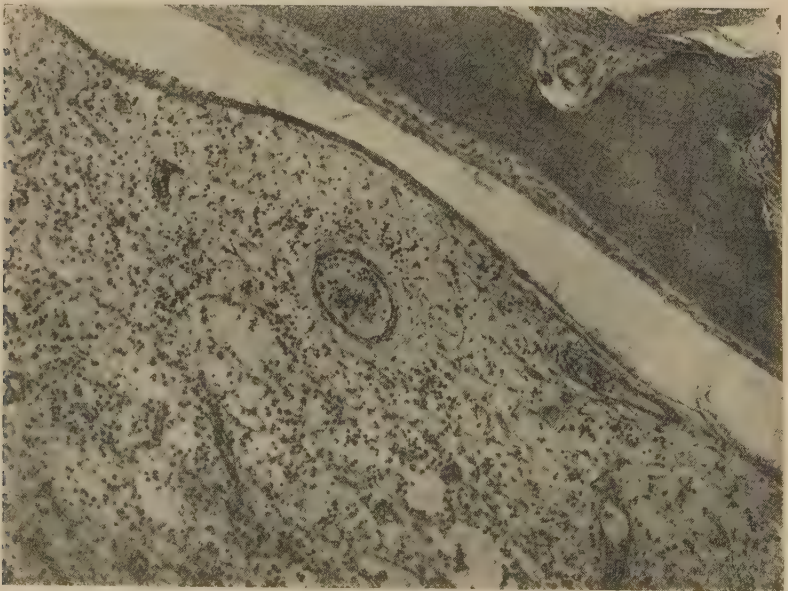


Fig. 93.

able swelling and edema of all tissues whose venous drainage is via the thrombosed vein. In the particular case under discussion the drainage

from the optic nerve and the orbit is into the thrombosed ophthalmic vein, the edema of the orbit producing an exophthalmos, the edema in and about

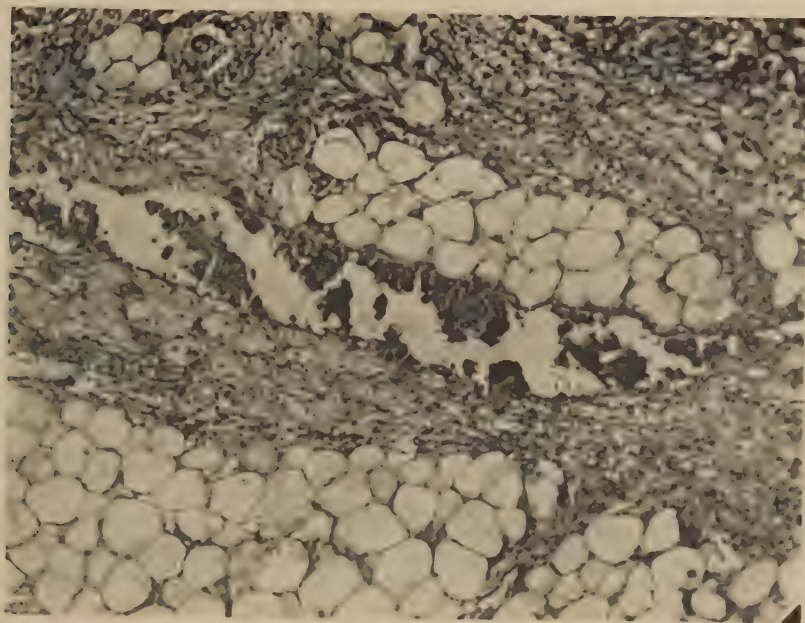


Fig. 94.

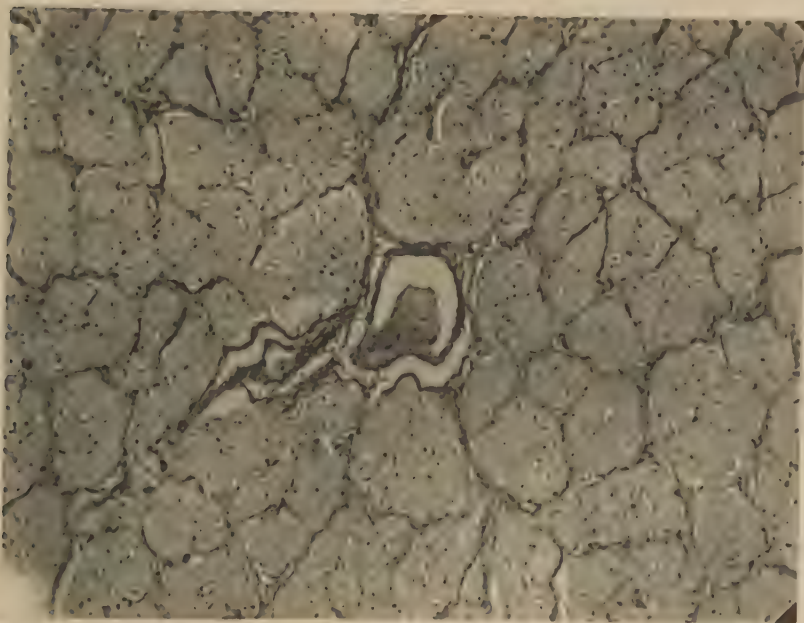


Fig. 95

the optic nerve producing pressure, neuritis, and paralysis. These illustrations show one way at least in which the optic nerve may be affected from diseased accessory sinuses.

In my opinion one of the most frequent and serious sequelæ of chronic sinusitis in children is atrophic rhinitis and ozena.

**Treatment.**—Two cardinal principles should govern all curative efforts: namely, metabolic improvement and drainage establishment; drainage not only of the diseased cell into the nose, but drainage from the nose.

A child having obstructed breathing not only harbors disease-producing germs and irritating matter within the nose, but is deprived of nature's means of clearing the various cells and sinuses, namely, nose blowing, sniffing, and hawking. To know how important are these forces puncture an antrum with a cannula, fill the cannula with a colored fluid, and ask the patient to sniff and hawk. The fluid in the cannula disappears immediately to appear in nose and throat. A process of blowing produces the opposite effect, that is, the fluid is blown from the cannula. Therefore, with a child in hand suffering from any form of sinusitis one should see to it that all adenoid tissue is removed from the nasopharynx. The nose should be cleared of secretion by use of Haskin's catheter suction and the sinuses will be much assisted in emptying themselves by the use of general suction which will be obtained by attaching to any negative pressure apparatus any cone-shaped hollow tip that, applied to the nostril, closes it. In young children I have found that the top of a medicine dropper perforated or the tip of an ear diagnosis tube makes an excellent tip. They may be attached to any ordinary soft-rubber suction bulb. An interruption or retaining bottle should be inserted between the tip and the suction bulb.

In older children one of the various suction apparatuses on the market may be used. In order to obtain negative pressure in the nose or sinuses the nose must be closed from the nasopharynx, one nostril is closed by the tip of the suction apparatus and the other by the finger of the operator placed on the ala of the other nostril, pressing it against the septum. The young child frequently closes the nasopharynx from the nose while crying and the older child will close it by saying K-K-K. This method of suction not only has a tendency to drain the sinuses, but produces a "Bier's hyperemia" of the lining membrane—a considerable help in restoring the membrane to health. Very weak solutions of cocaine ( $\frac{1}{2}$  of 1 per cent.) with possibly the addition of a little adrenaline sprayed into the nose favor drainage from the sinuses, as does also placing the child in a steam or vapor tent. During the fever stage the patient should be kept in bed. Vaccines, autogenous or stock, may be used, although their status is not as yet definitely settled. Many sinuses will be entirely relieved by this simple and conservative treatment.

Douching of the nose is a difficult matter in the first half of the childhood period. If one feels he must douche the head of a very young or obstreperous child, the child should be sheet bound, laid face down on a table with head well over its end, when the douching may be done.

The tendency of acute sinusitis is to recovery. With the aid of such procedures as above outlined I believe nearly 100 per cent. of acute cases will recover.

Children suffering from any form of sinus disease should have all the benefits of good air and sunshine. Where practical a change of climate is of great advantage.

*Chronic Cases.*—Many chronic cases will recover under the foregoing treatment if persisted in for a somewhat longer time than is usually re-

quired in acute cases. Others refuse to improve beyond a certain point. Such cases and those fulminating cases which develop so as to show danger symptoms early are surgical. Here is demanded not technic alone but judgment.

The various operations for the relief of sinus disease have been described in a previous chapter. As applied to children my feeling as to operation on the various sinuses is as follows:

*The Frontal Sinus.*—No attempt should be made to open the frontal sinus intranasally in children under twelve years of age. If the frontal sinus is diseased, the ethmoids are probably coincidentally diseased and a complete Killian operation had best be done.

*Ethmoidal Cells.*—The ethmoidal cells are frequently diseased independently, at least, of the frontal sinus. The safest and most promising operation is through an external incision doing the second part of the Killian operation. Exenteration of the ethmoid cells intranasally under general anesthesia is a dangerous procedure.

*Sphenoidal Sinus.*—In exenterating the ethmoidal cells one should, if there be any question of sphenoidal disease, enter that sinus at the same time. The walls of the sphenoidal sinus cannot be curetted without danger.

*Maxillary Antrum.*—Probably the maxillary antrum is the sinus most frequently demanding operation during childhood. In children whose permanent teeth have erupted the problem is simple. Assuming that the sinus has refused to clear up after an opening into the sinus has been made high up under the inferior turbinated bone, through which a considerable washing and medication has been done, the Caldwell-Luc operation should be performed. In younger children whose second teeth have not descended one must exercise judgment in determining whether to enter the antrum through the canine fossa at the expense of one or two teeth or to delay the radical operation until such time as the second teeth shall have erupted. I have never seen bad results from the sacrifice of a tooth. I have seen no lack of facial development nor over-production of osseous tissue from such procedure, except the case be malignant. The contention that these direful results do follow radical operation through the canine fossa in children I believe to be argued from academic premises.

As sinus disease in children becomes better known and its seriousness more frequently recognized by the general practitioner and pediatrician, greater prophylactic measures will be instituted to the end, let us hope, that the occurrence of this serious malady may be ever on the wane.

LEWIS A. COFFIN.

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## COMPLICATIONS OF NASAL SINUS DISEASE

A large number of diseases have their origin in affections of the accessory nasal sinuses. The occurrence of such complications is so much dependent upon the anatomy of these cells and sinuses that a thorough understanding of their structure and relationship is essential to a comprehensive knowledge of the diseases arising from them. Such anatomy is given in detail in the Section on Anatomy of the Nose and will not be re-

peated here except in so far as may be necessary to emphasize the great importance of those relationships and structures to the pathology, etiology, and treatment of the secondary affections.

These complications have, until rather recently, received but scant attention from medical investigators and medical writers. Even at present surgical authors, including to a notable extent those who specialize in surgery of the head, either have made no mention of this subject, or have dismissed it with far too little consideration. Internists have been foremost in their investigation of focal infection, and to them credit is due for many recent advances in etiology and treatment. The subject still deserves wider recognition for it is true beyond question that serious and even fatal disease not infrequently originates solely in the labyrinth of the paranasal sinuses. It is equally true that many ailments originating in these foci, while not immediately serious or fatal, finally become so, and are, meanwhile, a menace to the comfort and well-being of those so afflicted.

The importance of the mastoid antrum and mastoid cells as etiologic factors in many local and general diseases was long ago ably investigated and emphasized by MacEwen, but strangely enough, as pointed out by Ballance, the relationship of the nasal sinuses to the production of similar complications has attracted less attention.

**Causation.**—All complications of sinus disease are due primarily to previous bacterial invasion of one or more sinuses. Every cause operating to produce the primary sinus affection is, therefore, a cause of the complication. Since but a comparatively small percentage of cases of sinus infection are followed by complication it becomes necessary to seek reasons for its occurrence in some and not in all. Such reasons are based either on the character of the anatomy of the given sinus or upon the virulence of the invading bacteria. The main reasons are, therefore, either anatomical or pathological.

1. *Anatomical.* (a) *The Position of the Sinuses.*—The situation of the sinuses at the top of the respiratory tract, wholly above the digestive apparatus, favors the entrance by gravity of septic material from the sinus into the pharynx, larynx, and bronchial tree, or into the stomach and intestinal tract. This disposal of pus and mucus is not only true of children who swallow everything, but also of adults, who, try as they may to clear and expectorate the products from the infected cells, will not be able wholly to do so; a certain portion always finds its way toward stomach or lung. Gravity is therefore, at least in part, often responsible for certain respiratory and gastro-intestinal complications.

(b) *The Environment of the Sinuses.*—The anatomical relationship of the sinuses is so intimate to many important structures that disease of a serious nature could not long exist in the cell without rupture and invasion of adjacent sterile tissues. In many skulls the roofs of the ethmoidal cells and frontal sinuses form the greater portion of the floor of the anterior cranial fossa. The lamella of bone which forms this roof and floor, and which separates the septic contents of the sinuses from the sterile intracranial tissues of this fossa, is exceedingly thin, often paper thin. When the contents of one of the sinuses is under pressure, such as must occur when its normal drainage is blocked, this shell of bone gives way, and provides leakage from sinus to dura. The osseous walls of the sphenoidal sinuses are likewise thin, and an outbreak of their infected contents may occur as just described; the

septic fluid escapes in this instance into the middle cranial fossa, where meningitis or brain abscess would, of course, at once be set up.

The maxillary sinuses have no immediate relationship to the brain. Their walls are thin in places, especially on the orbital floor, canine fossa, and portions of the posterior and inner plates. Under pressure due to blocking of nasal drainage, antral abscess may rupture into the nose, mouth, orbit, cheek, or pterygomaxillary fossa.

Congenital defects or dehiscences in the wall of any of the sinuses favor the outbreak of pent-up pus into adjacent structures, with serious complication. In this way the mouth, orbit, meninges, or brain are sometimes involved. The proximity of the pituitary body, cavernous sinus, and optic tracts to the sphenoid and posterior ethmoidal sinuses predisposes these important structures to infection. Nearness of the sixth nerve to these same cells likewise accounts for affections of the ocular muscle which it supplies.

(c) *Circulatory*.—Venous blood from those portions of the frontal, ethmoidal, and sphenoidal sinuses which lie immediately adjacent to the dura mater, finally empties in most part into the cavernous sinuses. Numerous small venous radicals form communication between the veins of the mucous membrane of the sinuses and the venules of the dura mater. Lymph-vessels also communicate directly and freely between the sinuses and dura. The possibility is therefore always present of any suppurative process within these sinuses extending to the dura or cavernous sinuses through intercommunicating lymph and venous channels, in which case serious intracranial disease may be transferred without visible rupture of the sinus wall. The anterior ethmoidal veins empty into the ophthalmic veins and may carry septic products thence to the cavernous sinuses. The ethmoidal veins also anastomose with numerous orbital veins, which may account for orbital complication from ethmoidal disease without actual destruction of any part of the intervening osseous wall.

2. *Pathological*. (a) *Local*.—The pathology of local complications is similar to that of the parent disease, the original focus in the offending sinus. The pathology of the complication is that which characterizes an infection, and the resulting products of infection. In cases of sinus disease where nasal drainage is good, and the infecting organism one of the milder types, complication of any kind is rare, except in those in whom resistance to disease is greatly impaired. On the contrary, when the invading organism is of virulent type, the onset violent and the drainage from the sinuses blocked, the inflammatory reaction in the mucous membrane will be correspondingly severe. As a result the membrane breaks down and exposes the underlying osseous wall to necrosis and rupture. In many instances serious complication occurs without visible rupture of the sinus wall. Infection of the veins of the diseased nasal sinus walls takes place, following which thrombophlebitis spreads through the communicating veins to the dura, cavernous sinus, or orbital structures where new foci of disease are established. In such instance cavernous sinus thrombosis, local or general meningitis, or orbital cellulitis will be the inevitable sequence. In the event the nasal sinus involvement is accompanied by blocked drainage, rupture and complication may arise from more gradual softening and necrosis of the osseous wall, even though the type of infection is but mildly virulent. Once the septic products of the diseased nasal sinus have entered the normally sterile

cranial cavity by any of these routes, the pathology there established is similar to that of brain abscess, sinus thrombosis, or meningitis originating from other sources. The pathology of these is given in detail in another section of this work.

(b) *General*.—The pathologic products of accessory nasal sinus disease may be absorbed directly into the lymph- or blood-streams, whence they are carried to distant parts of the body, giving rise to a general septic state which may be either violent or mild. When the blood-stream or lymph-channel is once invaded from an infected focus, persistence of its evil effects is the rule, even for a considerable time after the original source of such infection has been eradicated. Indeed after new foci are once established, elimination of the original source of the disease may have but little effect on the severity and progress of the complication.

The method whereby hematogenous invasion of new and distant tissue takes place is embolic. The bacterial products absorbed from the foci in the sinus are transported through the medium of the blood-stream just as are the minute emboli from a thrombus, final lodgment taking place in some selective tissue, as joint, bursa, tendon, or heart. The types of organism involved in this metastatic process are the same as those causing the original sinus disease, and may include all varieties. However, those most frequently responsible are the streptococci, pneumococci, and diphtheroid groups. When occurring in distant tissues as a cause of systemic disease bacteria are often of a metamorphosed type.

Complications of sinus infection also take place through the medium of the lymph-stream. Lymphogenous invasion occurs perhaps more often than hematogenous. Whereas the lymph-vessels more readily absorb bacterial products from the diseased sinuses, the defense mechanism of the lymph system is such that the invading bacteria are destroyed before reaching distant organs, and hence result in fewer complications than when hematogenous.

Since all complications of nasal sinus disease occur either in the immediate vicinity of the affected sinus, or at a considerable distance from the original focus of infection, further discussion of the subject may best be based upon a classification which recognizes the seat of the secondary affection.

#### COMPLICATIONS AFFECTING STRUCTURES DISTANT TO THE SEAT OF PRIMARY INVASION

The number of known complications is great and grows with the amount and character of investigation that is given to the subject of focal infection and its consequences. It would seem that there are comparatively few general diseases that are not either in whole or in part caused directly or indirectly by some focus of infection. Dr. Joseph S. Evans in a recent comprehensive discussion of this subject enumerates thirty-seven well-established more or less serious general diseases that have their origin largely from this source.

Chief among the diseases listed by Evans, complications which may arise from focal infection, are myocarditis, endocarditis, pericarditis, pyelitis, acute rheumatic fever, affections of the skeletal muscles, bursitis, and arthritis. Several diseases not enumerated should be added, among them asthma, bronchitis, and infective disorders involving the digestive tract.

While undoubtedly focal infection, chiefly in the head, may be responsible for all the many complications mentioned by Evans, none will be further considered here for the reason that all are general complications which most concern the internist and are already well described in recent works on general practice.

Attention should, however, be called to the fact that the several foci of infection in the head other than the nasal sinuses may be involved either separately or together in the production of any general complication. For example, an arthritis may be the sole and direct result of a suppurating antrum, or it may be due partly to the antrum and partly to a coexistent tonsil infection. Observers and writers on general medical subjects are more familiar with focal infections arising in the teeth, tonsils, gall-bladder, etc., for the reason, no doubt, that the latter sources are more easily investigated. Hence they have most often assigned the cause of general complications to sources other than the sinuses. Tice, in a recent article on the etiology of pericarditis, mentions all the foci of infection in the body except the sinuses. Robert Soutter in an enumeration of the foci causative of arthritis likewise makes no mention of the sinuses. Other internists have also overlooked them. The number, size, and location of these sinuses and the frequency with which they are diseased makes it certain that, with the possible exception of the ear and its labyrinth of associated cells, the nasal sinuses stand first among the infective foci responsible for serious complicating disease.

**Treatment.**—Therapeutic measures directed to the nose or given inwardly have little or no value, except, perhaps, in acute cases of mild severity. When severe and chronic such treatment is but a means of procrastination and is, therefore, harmful. Climate is the one most helpful measure aside from surgery. Many who suffer constantly from sinus disease and its consequences, during the cold changeable winter months, especially in smoky cities, are greatly relieved by residence in a warm country where the air is free from impurities. Extreme southern California and south Florida are best. A careful study of the beneficial effects of climate on this class of disease, carried out in several of the most favorable portions of the United States and Europe, leads to the conclusion that extreme southern Florida is most suited, chiefly for the reason that there not only is the air perfectly clean and pure, but it is also soft and warm throughout the whole twenty-four hours, and in all seasons.

The treatment should, when possible, be prophylactic. The complications should be prevented by curing the sinus disease before the systemic infection has taken place. Patients with sinus infection should be warned concerning the fact that serious heart, rheumatic, kidney, and other diseases may result from their "catarrhal trouble" and should be urged to submit to operative or other measures before the actual occurrence of some complication.

Surgical measures directed to the cure of the primary focus are necessary in most cases. When chronic, and when the sinus is filled with the products of infection, including obstructive polypi, trivial surgical measures are perhaps worse than procrastination. Ventilation, drainage, and complete removal of all disease within such a sinus are clearly indicated. Radical operations, usually of an extranasal type, will be followed by satisfactory results. The technic of such operative measures is given elsewhere.

### COMPLICATIONS AFFECTING THE ENVIRONMENTAL STRUCTURES OF THE SINUSES

This group falls wholly within the field of the rhinologist. Environmental complications occur chiefly as a result of the position of the sinuses, the extreme thinness of the sinus walls, and the presence of congenital or necrotic dehiscences in the walls. Such complications also follow the transport of infective fluids through the medium of the intercommunicating veins. The environmental affections secondary to sinus disease are either extracranial or intracranial.

Chief among the complications that occur in structures outside the skull are minor facial neuralgia, nasal polypi, osseous necrosis, abscess of the mouth and cheek, orbital cellulitis and abscess, and optic neuritis. Those affecting intracranial tissues are meningitis, epidural abscess, and brain abscess. In the limited space of this section it is clearly impossible to do more than detail a few bare facts concerning each disease.

#### GROUP I. COMPLICATIONS AFFECTING EXTRACRANIAL STRUCTURES

(a) **Minor Facial Neuralgia.**—This disease may have its origin in any one or all of the infected nasal sinuses, but the maxillary, sphenoid, and frontal are the most frequent seats. The term "minor neuralgia" is used to distinguish it from major neuralgia, or *tic douloureux*, which latter Frazier believes is solely an affection of the Gasserian ganglion, whereas he regards the minor variety as centering in the nasal ganglion, the peripheral sensory nerves being primarily affected by the diseased sinus. The peripheral nerves in *tic douloureux* are not involved.

*Diagnosis.*—The chief symptom is pain of a continuous character. Sometimes the pain is lancinating, but more often is only an ache with exacerbations of severity. It is never spasmodic, never incited by trivial means. Suppuration is present in one or more nasal sinuses in a large percentage of cases, but this fact may not be determined with certainty except after repeated, careful examination for the reason that not infrequently the sinus disease becomes quiescent, in which case evidence of the suppuration will not always be found in the nose. Good roentgenograms will be of great assistance. Minor neuralgia must be distinguished from the major variety, the pain of which is always spasmodic, always lancinating, and always incited by trivial causes.

*Treatment.*—A long list of remedies has been recommended. All are useless in cases solely caused by infected sinuses. Improved drainage of the sinuses, followed by proper methods of irrigation, will usually cure in acute or subacutely affected sinuses, and this plan should be followed. When the sinus disease is chronic its thorough eradication by surgical measures becomes necessary. Operative measures on the nasal sinuses have little or no effect in relief and cure of *tic douloureux*.

(b) **Polypi.**—These are usually classed among nasal diseases. Undoubtedly such growths are a complication of sinus disease. Sufficient evidence of this is presented in radical operation on the sinuses for chronic suppuration, when on widely exposing the sinuses to view it is seen that the mucous membrane is polypoid, that the polypi spring directly from it and have pushed their way toward outlets of least resistance, into the nostril.

The *symptoms* are chiefly nasal, with blocking, excessive discharge both forward and postnasal, a nasal voice, sneezing, and easy cold taking. Not infrequently bronchitis and asthma are present.

*Treatment*.—Medicines taken inwardly, sprays and applications locally are never curative. Removal of the polypi by intranasal methods gives relief for a varying length of time, but seldom cures. It is, however, sometimes the method of choice, especially in elderly patients who are immunized to their own infections, and who have no serious complication other than the polypi. In most cases cure of the polypi will depend on complete eradication of the disease in the sinus from which the new growths spring.

(c) **Abscess of the Mouth and Cheek**.—Necrosis of the osseous wall of the maxillary sinus occasionally takes place above the alveolar process and at or posterior to the canine fossa. Such loss of bone may be considerable, even in children, especially when following scarlatinal or other severe sinus infection.

*Symptoms*.—Pain and swelling of the face occur adjacent to the nose of the affected side. The temperature will be increased if already present. Pus from the maxillary sinus escapes and dissects its way over the external wall of the sinus causing an induration of the cheek, or it may loosen the mucoperiosteal membrane of the roof of the mouth, where it forms a sensitive tumorous mass.

*Diagnosis*.—The history of the case and the detection of the infected sinus should lead to a correct diagnosis of these sudden, painful tumors. Fluctuation may be made out. Such swellings should be differentiated from simple abscesses due to diseased teeth, and from sarcoma of the antrum or alveolus, which latter often suddenly presents in children, and its soft and fluctuating feel is not infrequently mistaken for abscess of a tooth or suppurating maxillary sinus. In sarcoma the adjacent teeth are loose without evidence of dental disease, and the tumefaction is often greater than that due to antral or dental abscess. Incision of the tumor is followed by escape of pus in abscess; of blood only in sarcoma.

*Treatment*.—If due to acute infection of the sinus, improving nasal drainage, lancing the abscess, and curetting away any remaining disease of the bone about the fistulous openings will be sufficient to cure. Curettage of the interior of the sinus is not indicated in acute cases. When the necrosis is extensive, one or more teeth must sometimes be sacrificed, since they are found to set in necrotic bone. When necrosis and abscess result from chronic sinus involvement, radical operative measures are required on the sinus, the interior of which should be exposed and its polypoid, infected mucous lining completely removed. The Caldwell-Luc or Denker's operation is best suited for such cases.

(d) **Retrobulbar Optic Neuritis**.—*Etiology*.—It has been established beyond question that this complication may follow suppuration of the sphenoidal sinus or posterior ethmoidal cells. It is also reasonably well proved that it may also originate from non-suppurative, hyperplastic inflammatory processes in these same sinuses. Anatomists and pathologists think this probable, while oculists and rhinologists have established it as a rather definite clinical fact. Onodi, Vail, White, Loeb, deSchweinitz, and Van der Hoeve believe such relationship unquestionable, while Cushing, from the neurologic point of view, thinks it impossible for retrobulbar

neuritis to have its origin from such a source, and says much harm has been done through operative measures based on the erroneous assumption that the optic ailment is a complication of non-suppurative sinus disease.

It is probable that the mode of infection is not always the same. The toxic products may reach the nerve by direct extension from the sinus, or they may be carried by either the lymph- or blood-stream. In case no actual suppuration exists in either sphenoid or postethmoidal cells, Sluder believes that optic neuritis may be accounted for on the theory that a hyperplastic process extends from one or both of these cells to the optic canal, which is thereby narrowed to such an extent that the optic nerve is seriously compressed in this portion of its course.

*Symptoms.*—There is usually a history of acute or chronic nasal disease. Vision of the affected eye is blurred. The visual difficulty comes rather suddenly and makes rapid progress, until shortly the patient is able to count fingers only when close. The field of vision is greatly restricted. The patient complains of pain and soreness on movement of the eye, which is also sore to pressure under the finger.

*Diagnosis.*—This should be made as early as possible. Early definite diagnosis is not easy, and may require the combined skill of the ophthalmologist, neurologist, internist, and rhinologist. When actual suppuration is present in the posterior group of sinuses this fact is helpful. Changes in the fundus are important and must be differentiated from those occurring from brain tumor and other intracranial disease. Roentgenograms are of assistance when sinus suppuration is present, but are negative in others. In optic neuritis without sinus suppuration it may be found that the nasal septum is deformed and the upper turbinates enlarged. White attributes the neuritis to this nasal pathology, which blocks sinus aëration and sinus drainage. Nowhere is rhinologic skill in examination more essential than in this class of case, for loss of vision may speedily result from opinions based on inefficient or indifferent investigation of the nasal and sinus pathology.

*Treatment.*—When clearly due to suppurating ethmoidal or sphenoidal sinuses, early eradication of these septic foci is urgently indicated. When suppuration is not present, and there is no history of previous sinus infection, milder measures may suffice. Some cases recover without surgical aid, hence medicinal and local nasal treatment is advisable when the ocular disease is mild or slow in progress. All cases of optic neuritis, however mild or slow of progress, should be under close observation and receive careful study, since otherwise opportunity may be lost to preserve function. Should progress be slow from expectant treatment, or should the blindness become progressively worse, surgery of the sinuses is indicated even though actual suppuration is not determined, and may not be present. When the nasal septum is deformed and the upper turbinates enlarged, these defects should be surgically corrected. The sinuses should not in such cases be dealt with radically. After removing the pathologic upper turbinates it has been found sufficient merely to uncap the postethmoidal cells and to open the sphenoid sufficiently to secure good ventilation and drainage. The value of these simple surgical measures is attested by many cases of progressive optic neuritis that have been cured or greatly relieved by such comparatively simple surgical measures.

(e) **Orbital Cellulitis and Orbital Abscess.**—This complication sometimes follows suppuration in the frontal and maxillary sinuses and the ethmoidal cells.

*Pathology.*—Pathogenic organisms may enter the orbital tissues through the anastomosing veins, through a dehiscence in any one of the orbital walls, or by direct process of necrosis and fistula.

*Symptoms.*—Swelling, edema, and redness accompanied by closure of the eyelids and intense pain occur at once. Pressure upon the eye sets up severe pain. The eyeball may protrude, and be turned upward, outward, or downward according to whether the infection originates in the antrum, ethmoid, or frontal sinus. Abscess formation with discharge of pus outwardly sometimes takes place, resulting in fistula. If not relieved great destruction of the orbital tissues may occur, with total loss of vision in the affected eye.

*Diagnosis.*—The points upon which a diagnosis of this condition may be made are the presence of a suppurating adjacent sinus, and the symptoms and history of the case. Cavernous sinus thrombosis gives rise to symptoms quite similar to those caused by adjacent sinus disease, and must be differentiated.

*Treatment.*—Early eradication of the focus of infection in the sinus, opening the abscess of the orbit, and provision for adequate drainage are imperative, since delay will result in loss of the affected eye.

## GROUP II. COMPLICATIONS AFFECTING THE INTRACRANIAL STRUCTURES

These include epidural abscess, localized and general meningitis, and abscess of the brain. Normally the intracranial contents are sterile. The several pathways by which infection is carried from a sinus to the dura have already been discussed. The entrance of pathogenic products into the cranial cavity from osseous erosion will with certainty be followed by epidural abscess, localized meningitis, and possibly by general meningitis and brain abscess. If entrance of infection occurs through the blood-stream intradural abscess is probable.

(a) **Epidural Abscess.**—Following the rupture of one of the nasal sinuses which contains pent-up pathologic exudates, pus in varying quantity enters the cranial cavity, dissects adjacent dura from the inner table of the skull, and at once becomes an epidural abscess of direct invasion. Defense activity is immediately aroused in the invaded portion of the dura, and if the defense mechanism is sufficient, or the invading bacteria mild, the abscess will at once be limited by a wall of protecting adhesions between dura and bone. In such event the abscess may remain quiescent for an indefinite length of time, especially if improved drainage into the sinus and nose is maintained.

*Symptoms.*—The development of an epidural abscess may not be accompanied by definite symptoms. There is a history of nasal disease with nasal or postnasal discharge. Headache is common. Fever accompanies the headache when the intracranial invasion first takes place, but subsides when the abscess becomes limited by adhesions. There may be loss of strength, the mental faculties may be dulled, and the patient's physical condition may be below normal. All these, however, are but the more or less exaggerated symptoms of the original sinus disease.

*Diagnosis.*—The presence of an epidural abscess may be wholly unsuspected, and is discovered only during an operation on the affected sinus. Headache, lassitude, mental dulness, and other symptoms are characteristic of many local and general infectious diseases. In all cases of pronounced headache occurring in connection with nasal sinus disease, especially if sudden, persistent, and accompanied by a rise in temperature, invasion of the cranial cavity should be suspected. Roentgenographic studies and all other known means helpful to diagnosis should be made, and careful analysis of the evidence thus obtained may point rather conclusively to epidural abscess.

*Treatment.*—Surgical measures alone promise any hope of permanent relief or cure, and should be carried out without delay. The abscess should never be attached by any route except that which leads through the affected sinus. Thorough removal of disease from the sinus is accomplished as a first step, following which the abscess receives attention. If a fistula leads to the abscess cavity this should be enlarged and all adjacent necrosed bone should be removed. It is never wise to open the dura or to curet the abscess. The former is unnecessary and the latter dangerous for the reason that protective adhesions would be broken down and general meningitis would certainly follow.

(b) **Meningitis.**—Local meningitis is immediately set up in every instance of invasion of the cranial cavity by bacterial organisms. It is Nature's first line of defense and, if successful in combating the infection, may forever prevent further spread of the disease. Localized meningitis occurs almost coincident with the epidural abscess, and therefore its symptoms are identical with those of that complication.

*General meningitis* never occurs unless the invading organisms are virulent and the protecting mechanism of the patient is impaired. When virulency is great and the defense weak, spread of the disease to the arachnoid and pia is inevitable, and general suppurative meningitis the result.

*Symptoms.*—The symptoms of general meningitis are usually pronounced. A severe chill occurs, which is followed by a rapid rise in temperature and a correspondingly rapid pulse. The patient complains of headache of distressing severity. Vomiting and constipation are almost constant symptoms. Photophobia comes on early, the patient demands darkness, turns away from the faintest light, covers the head, and cries aloud from pain. When progress is rapid convulsions often occur. The eyes become injected and squint is common. Rigidity of the neck is marked. Stupor and coma supervene and death speedily follows.

*Diagnosis.*—This is based chiefly upon the history of sinus suppuration, and upon the actual discovery of such a focus by careful rhinoscopic examination. The symptoms are strongly diagnostic. Evidences of intracranial involvement are often present in the fundi of the eyes, but the same conditions of the eye may exist in other brain diseases, and must be differentiated. The general symptoms of meningitis often closely simulate those of infectious diseases, especially typhoid fever, smallpox, and scarlet fever. Spinal puncture and the withdrawal of spinal fluid will show in meningitis a much increased intradural pressure, sometimes as high as 250 mm. Hg. The appearance of the fluid withdrawn is often clouded or purulent. Laboratory examination of the fluid shows a very greatly increased

number of cellular elements. These and other symptoms which may be present should leave little doubt as to the existence of meningitis.

*Treatment.*—General medication, including several specially prepared sera, has been advocated and used with some success. It could scarcely be expected, however, that such medication could cure a widely distributed suppurative process that is completely pent-up.

The original focus of infection in the sinus should be cleaned out in early cases, even though this will have little effect on the meningitis, once this disease is well established. Free exit in so far as possible must be given to intradural exudates. Whether the original source of the infection was in the ear or in one of the nasal sinuses the resulting products are the same, and the same operative procedures are indicated. These products accumulate in the subdural and subarachnoid spaces, but to a greater extent in the several cisterna below the posterior brain. The first and best principle of good surgery—to drain adequately all infected cavities—should be followed in the surgical management of general meningitis. The technic for performing such operations is given in another chapter.

(c) **Brain Abscess.**—This complication, occurring as a result of sinus disease, is, according to Ballance, relatively more frequent than general meningitis. Brain abscess from this cause is less frequent than abscess due to suppuration in the temporal bone.

*Pathology.*—Brain abscess is always a complication of infective processes originating outside the skull. When secondary to sinus disease it results either from rupture of the adjacent sinus wall with direct entrance of pus, or from a transference of septic material from sinus to brain through the intercommunicating veins. When abscess of the brain follows an actual outbreak of pus from the sinus, the pus does not at the same time penetrate the dura and enter the substance of the brain. Its entrance into the brain occurs subsequently to the formation of an epidural abscess, as previously described, and to the limitation of such abscess by the localized meningitis which has been set up. Brain abscess, therefore, is a complication of the slower moving pathogenic process, and probably never occurs when the invading organisms are virulent, unchecked, and end in general suppurative meningitis.

When once tightly sealed by the localized meningitis further accumulation of pus in the epidural abscess cavity results in increased pressure upon the adjacent dura which in time yields with consequent invasion of intracranial tissue. Such invasion does not result in flooding the adjacent brain structures. The penetration of the dura by pus is gradual and occurs only after dura, arachnoid, and pia are bound together as one by inflammatory adhesions. The pathway of invasion is thus safeguarded against leakage into the subdural space with consequent meningitis. Because well supplied with blood and lymph the cortex of the brain is least vulnerable to infection, and hence the white matter is the most usual site of intradural brain abscess. The progress of the infection from its source in the sinus to its destination in the white substance of the brain is marked by an inflammatory, fibrous tract which leads from one to the other. Ballance, Bergmann, and others have aptly referred to this as a "stalk." This stalk may remain open and provide drainage from the brain abscess into the sinus. In many cases when chronicity is established the brain abscess becomes encapsulated, and the stalk may disappear, leaving but a trace or even no trace of its former

course. The abscess may be multiple. As it enlarges it encroaches upon both cortical and deep cerebral structures, all of which may be crowded aside gradually and hence often without serious disturbance of function. Rupture of the abscess into one of the ventricles is not uncommon. If the intracranial tissue is hematogenous, no stalk is present and localized meningitis does not occur.

*Symptoms.*—The symptoms of brain abscess from any source are often vague and indefinite. When the complication occurs subsequent to nasal sinus disease it is often yet more vague because of its situation in the silent areas of the frontal lobe. The symptoms of abscess in the frontal lobe are, for that reason, often referred to as “latent.” The symptoms of brain abscess are so frequently similar to those of the sinus affection, or of general disease, that when arising from the abscess *per se*, are sometimes completely obscured by the former. Especially is this true when once the brain abscess has become chronic and encysted.

The symptoms most commonly present are headache, vomiting, choked disk, constipation, subnormal temperature, and slow pulse. All these symptoms are seldom present in any one case. A well-developed brain abscess may give rise to but two or three well-defined symptoms. Headache is the most frequent and is present with greater or less persistence and severity in the vast majority of cases. In brain abscess of sinus origin the headache is usually frontal, dull and heavy, and sometimes excruciating. Even when the abscess is in the frontal lobe, the headache may be occipital or parietal.

Except at the onset when localized meningitis is present, or later when rupture of the abscess has occurred, fever is not present. On the contrary, in uncomplicated cases of abscess of the brain, subnormal temperature accompanied by slow pulse and lowered respiratory rate is common.

Next to headache vomiting is the most frequent symptom. Its occurrence varies in different cases. At the onset of the abscess, at the time of rupture or extension, vomiting may be a prominent symptom. In encysted abscess it more rarely occurs. The vomiting is of a “purposeless,” “cerebral” character. The stomach contents are ejected without nausea and the patient may wish food immediately afterward.

Occasionally the symptoms of brain abscess closely simulate those of acute general infection, in which event the temperature is high and is accompanied by acute delirium or even mania. This, the fulminating type, becomes rapidly fatal.

*Diagnosis.*—The first and most essential point in diagnosis is to establish the fact that acute or chronic infection is present or has been present in one or more nasal sinuses. Failure to discover actual pus in the nose at one examination should not be construed to mean that no infection is present, for the brain abscess may have taken place long ago when the sinus was active, whereas at the time of examination it may have been entirely quiescent. Repeated examination of the sinuses by all known and useful methods are, therefore, necessary to accurate final conclusions.

Headache, vomiting, and choked disk are present at some period in the history of nearly every case, although all are seldom found at once except in those most typical. Other symptoms, as subnormal temperature, slow pulse, and obstinate constipation, often accompany brain abscess, and when present are great aids to diagnosis.

Headache, while often not more severe than that which accompanies uncomplicated sinus disease, is often intense and of a type which arouses suspicion of intracranial complication. Its behavior differs from common headache. It is sometimes acutely severe and paroxysmal. After an interval of freedom from pain the patient suddenly grasps the head between the hands, appears in serious distress, and sometimes cries aloud from pain. The headache leaves as suddenly as it came and there is an interval of complete freedom during which the patient seems almost normal. Association of nasal sinus disease with headache of this type should lead to strong suspicion of abscess.

Vomiting is not a symptom in all cases, but when present, projectile, and occurring without nausea, is strong evidence of intracranial complication. Choked disk does not always accompany this complication, and even if found is not pathognomonic of brain abscess. It signifies intracranial pressure from some cause which may be from brain tumor as well as from abscess. However, when taken in connection with the other symptoms already enumerated, choked disk becomes a clinching argument in favor of brain abscess.

*Treatment.*—While a few cases of brain abscess are recorded where cure resulted from rupture of the abscess through the pathway of entrance, the disease is essentially a surgical one. When the diagnosis is certain, or even highly probable, drainage of the abscess is necessary and should not be delayed. The technic of the operative surgery on the brain is given in another section, but a few observations on the surgery of brain abscess complicating sinus disease should be made here.

The greatest percentage of recoveries following operative procedures on brain abscess due to focal infection from the sinuses has followed the plan of first eradicating the disease in the sinus and then draining the abscess by entering it through the stalk or through the inflammatory pathway from the sinus to the abscess. This method has the objection of draining an intracranial structure through a septic field. This objection can, however, be largely overcome. If the operator enters the abscess in the manner stated no fresh intracranial tissue is wounded, since the pathway of incision lies wholly within the adhesive barriers of the pathway of entrance. The frontal sinus is the most frequent beginning of this pathway. If this sinus is thoroughly cleared of every infected particle of bone and polypoid membrane, it is thus sterilized and prepared as a safe field through which to drain the brain abscess. The abscess should be opened only after the sinus has been thus thoroughly cleaned and prepared. The subsequent problem is to maintain sterility of this field until healing of the abscess is complete. The chief difficulty in carrying out a sterile plan arises from the nose infection which enters through the nasofrontal duct and is a menace in all cases. At the time of operation on the sinus an adequate opening must be made from the sinus into the nose, which opening must subsequently be kept free. The turbinates and ethmoids are often polypoid in this type of brain abscess, and these if left will almost certainly block the drainage and reinfect the sinus during the healing. If, therefore, time will permit at the original operation on the sinus and brain abscess, any obstructing polypi or turbinate should at the same time be removed. Time will usually permit this if previous organization of the operating room forces is made. Thoroughness in removing all the infected tissues that originally caused the

abscess, and that will certainly reinfect the wound if left behind, is so essential to the cure of the brain complication that every care and thought should be bestowed upon it. A large percentage of all frontal lobe brain abscesses will be cured if adequate drainage into a sterile field is secured and maintained. Most cases die when this is not accomplished.

JOHN F. BARNHILL.

### THE SINUSES IN RELATION TO EYE DISORDERS

In the medical literature of a century ago various eye disturbances were attributed to "snuffles" or some similarly vague intranasal disease. While there has been a steady accumulation of data on the relationship between eye diseases and nasal infection, it was but recently that anything like a scientific explanation could be made. This, naturally, followed the epoch-making study of Onodi<sup>1</sup> in 1908 on the anatomical relationship between the accessory sinuses and the optic nerve and later contributions by Loeb,<sup>2</sup> Skillern,<sup>3</sup> Hajek,<sup>4</sup> Schaeffer,<sup>5</sup> Sluder,<sup>6</sup> van der Hoeve,<sup>7</sup> deSchweinitz,<sup>8</sup> Knapp,<sup>9</sup> and others. It was not, however, until radiography had reached its present stage of development that infections within the various sinuses could be definitely located or excluded.

The sinuses, owing to their intimate relationship with the orbit, have been considered the most common source of eye infections. The teeth, tonsils, and other regions being more remote have not received the attention they warrant. Infections in teeth and tonsils, at least in my cases,<sup>14c, e</sup> have far outnumbered those within the sinuses.

The following is a brief résumé of the eye disorders attributed in whole or part to the accessory sinuses:

Thickening of the orbital periosteum by infection within an adjacent sinus is frequently the cause of persistent edema of the lids. Conjunctivitis, keratitis, iritis, scleritis, uveitis, choroiditis, cyclitis, retrobulbar and optic neuritis most ophthalmologists believe may originate from infections in the sinuses. The contents of the orbit may be involved by the direct extension into it of a mucocele, pyocele, or neoplasm. Muscular asthenopia and loss of accommodation may result from a neuritis originating from infections in the sinuses.

Whenever cases with eye disturbances are referred to the rhinologist, a careful study should be made of the accessory sinuses, using every available method of arriving at a correct diagnosis, such as roentgen ray, transillumination, nasopharyngoscope, suction, and, above all, most careful inspection both before and after shrinking the tissues in the nose. It is never wise to advise the removal of the turbinates and the opening of the sinuses unless one has definite evidence that these structures are harboring infection. One hears so often the remark that "it does no harm to open the sinuses." This is a great fallacy and has justly cast aspersions on rhinologists as lacking in sufficient scientific skill to diagnose infections within the sinuses and correctly evaluate intranasal surgery. If, after investigation, one finds an infected sinus that might be responsible for the eye disturbance, it should be opened. The exenteration operations, unless practically all the sinuses are involved, are not justifiable, as radical operations rarely produce radical

cures. While the offending sinus should be carefully and thoroughly opened, the normal function of the nose should be as little impaired as possible. The brilliant results following intranasal surgery occupy too prominent a place in rhinological literature to the exclusion of what can be accomplished by less spectacular methods. In treating the aged and infirm excellent results frequently follow the use of alkaline solutions, of argyrol packs, of ephedrine, of hot mineral oils, and of suction and ointments. It is much wiser to use these simple remedies than unnecessarily to mutilate the intranasal structures. Then, too, one should consider the general condition of the patient, his manner of life, the atmosphere in which he lives. Do not forget the individual in looking for some "pet" intranasal operation. The mental unbalance, even after a minor operation, may be more distressing than the condition it was hoped to relieve.

The most important of the eye disturbances arising from infections in the sinuses are those involving the optic nerve. This is largely due to the fact that the optic nerve is particularly susceptible to the action of toxins. The focus of infection in many instances is located with great difficulty. For this reason special stress will be laid on retrobulbar and optic neuritis, although it is expected that much of this discussion will be found applicable to the other eye disturbances arising from focal infection.

**The Anatomy.**—The anatomical relations between the optic nerve and accessory sinuses are of vital importance. Onodi<sup>1</sup> investigated this relationship thoroughly and his findings furnish the anatomic foundation for the theory of blindness from accessory sinus disease. He demonstrated by eleven distinct anatomic findings the possibility of double-sided and contralateral blindness from one-sided sinus infection. "Our observations," Onodi<sup>1</sup> says, "have shown that the wall between the last ethmoid cell and the canalis opticus is nearly always as thin as tissue paper; dehiscences in the walls of the accessory cavities have been found, where the diseased mucosa may come into direct contact either with the dura mater or the optic nerve sheath."

Normally, according to Loeb,<sup>2</sup> "the optic nerve may be described as passing externally from the chiasm along the roof or lateral wall of the sphenoid and in close relation with the ethmoid labyrinth *only* at the posterior external angle of the last cell." The sphenoids and posterior ethmoids are the only sinuses in intimate relation with the optic nerve, and to reach the tissue adjacent to the nerve the direct and logical route is through these structures and not through the entire ethmoid labyrinth.

**The Diagnosis.**—The diagnosis is naturally of major import. The symptoms in the ordinary cases of acute optic or retrobulbar neuritis are quite typical. Associated with the loss of vision there may be discomfort about the eye or lameness on moving it. Occasionally there is slight exophthalmos, this condition indicating an inflammatory process in the orbit. Pupillary changes and ptosis are found occasionally. There is frequently nyctalopia, a central scotoma for colors, enlargement of the blind spot, and contraction of the fields. The patients generally say that things at first were blurry when looking straight ahead, but comparatively clear when viewed sideways. Changes in the fundus are of great diagnostic value, varying as they do from the normal to a commencing pallor in some cases, and in others to a marked redness and swelling about the nerve, with engorgement and tortuosity of the retinal veins. It is essential that these inflammatory swellings be differentiated from the edema due to intracranial pressure.

*Papilledema.*—The question of papilledema ever originating from infections in the sinuses requires elucidation. I have on record about 125 cases with optic nerve disturbances apparently arising from infections in the accessory sinuses, teeth, or tonsils. Of this number but 5 (or 4 per cent.) have shown edema of the disk. In each case it was unilateral, transitory, and but moderate in extent, *i. e.*, between 2 and 3 diopters. In all there was sudden and marked loss of vision, one recovered spontaneously, the others after opening some of the sinuses or removing infected teeth or tonsils. It can, therefore, be considered a rare condition, entirely distinct from the papilledemas associated with brain tumors, brain abscesses, meningitis, and lateral sinus infections, and would better be designated by a different name. I<sup>4d</sup> have suggested "optic neuritis with edema" to distinguish it from the true papilledemas due to increased intracranial pressure.

*The Middle Turbinate.*—While there may be so much pathology in the nose that there is no question as to the source of the infection, in many cases the nose appears practically normal. The one vital point to determine in the nasal examination is the size and position of the middle and superior turbinates. Do they block the ventilation of the posterior sinuses? It takes but moderate obstruction to interfere with the ventilation and the mistake is frequently made of expecting to discover marked changes; while, on the other hand, whenever there is no blocking of the normal openings of the posterior sinuses, this region can usually be ruled out as a causative factor in the neuritis. The following is quoted from Stark,<sup>10</sup> who expresses well this thought: "From a nasal standpoint we must not expect to find the common symptoms of sinus infection, pus, polypus, history of nasal discharge, etc., as we are dealing with a closed sinus; otherwise we should not have pressure. The deflected septum and middle turbinate tightly pressed against the lateral wall should always be suspected." Over half the cases give a history of a recent coryza or a prior influenza, but in a few there is no history of infection. It is imperative that careful radiographs should be made of the accessory sinuses and optic canals. Whenever the sinuses are negative, the teeth and tonsils should be suspected. Tonsils at all suspicious, with teeth and sinuses negative, should be removed. The diagnosis many times must be made largely by exclusion. In all cases the patient should undergo a thorough physical and neurological examination. One should consider in turn blood, urine, hysteria, pellagra, lues, tobacco, alcohol, lead, arsenic, quinine, etc. While investigating, time is an important element. Practically all necessary tests can be made within forty-eight hours.

*Brain Tumors.*—Pituitary disease, brain tumor, and multiple sclerosis, while not usually producing such sudden loss of vision, must always be borne in mind. As a matter of routine the various cranial nerves and lobes of the brain are tested. Not a few cases with brain tumor have had various nasal operations before they were correctly diagnosed. The possibility must always be borne in mind that even in patients with definite nasal pathology the loss of vision might be due to intracranial neoplasms or multiple sclerosis. The history of the onset of the amblyopia is so typical, either of its nasal or central origin, that this in itself should put one on guard. The slow progressive loss of vision, nearly always bilateral in the cases of central origin, warrants at least the necessary time for investigation; while in the sudden unilateral loss of vision cases, especially with normal

optic canals, needless haste is unnecessary, as many will recover spontaneously.

**Etiology.**—The earlier writers considered the mere presence of pus in the sinuses the all-sufficient explanation for the disturbance about the optic nerve, and while this may undoubtedly be the cause in some instances, it is by no means the usual or the only one. The nerve is ordinarily protected by the barrier thrown out by inflammatory processes, so that it is rarely involved unless through some anatomic abnormality. Hyperplasia has been emphasized by others as the chief cause, and while hyperplastic tissue may involve the posterior sinuses, it has seemed from a study of numerous sections and cases that it would better be considered a predisposing factor rather than the principal etiologic condition. Hyperplasia undoubtedly renders the sinuses more vulnerable. The etiology seemed to be explained in most of my cases by blood-stream infection from the antra, teeth, or tonsils.

**Pathology.**—(1) Direct extension; (2) toxemia from some infective process; (3) bacteremia or focal infection; (4) hyperplasia; (5) anaphylaxis.

1. By *direct extension* infections in the posterior sinuses may extend by continuity of tissue to the orbit and produce an orbital abscess or a cellulitis, but rarely, if ever, an optic neuritis or an iritis. Infection in a more remote sinus (an antrum for instance) usually becomes walled off, but should the infection persist, there may be forced into the system bacteria and toxins which, through the blood-stream, can produce an optic neuritis.

2. *Toxemia.*—It is conceded that retrobulbar and optic neuritis can be caused by alcohol, lead, tobacco, quinine, optochin, arsenic, lues, etc., so that, reasoning by analogy, there is little doubt that toxins originating in the accessory sinuses, teeth or tonsils, or, for that matter, anywhere in the body, may have similar action on the optic nerve.

3. *Bacteremia.*—It has been demonstrated by Billings that infectious micro-organisms may be carried in the blood-stream or by the lymph-channels from the foci of infection in the teeth, tonsils, and accessory sinuses to the terminal blood-vessels in various regions of the body. He has shown how the inoculated blood-vessels become more or less occluded by endothelial proliferation and leukocytic infiltration, and that the bacteria escape through the vessel walls into adjacent tissue, so there would seem little doubt that bacteria may also travel via the blood-stream and lymph-channels to the various structures making up the eye and to the optic nerve. Hematogenous infection from some focus in teeth, tonsils, or sinuses furnishes the best explanation of the pathology of these optic nerve disturbances due to infection.

4. *Hyperplasia.*—As a predisposing factor hyperplasia is probably of importance. It undoubtedly renders the accessory sinuses more vulnerable. Hyperplasia plus infection is, however, of far greater consequence than the mere fact that the tissue has become hyperplastic.

5. *Anaphylaxis.*—Stark<sup>10</sup> has advanced the theory that there is a "sensitization of the tissues of both the sinus and the orbit by the bacterial proteins, producing an allergy resulting in a localized anaphylactic reaction each time the individual comes in contact with a fresh infection of the same bacteria in the nose, teeth, tonsils, or other parts of the body. For that reason some of these cases give a history of attacks resembling hay-fever, or acute coryza, shortly previous to the eye trouble."

**Prognosis.**—The prognosis depends largely upon the duration and extent of the loss of vision, the condition of the fundus, the virulence and site of the infection, and the size of the optic canal.

*Duration.*—The conclusions reached from an analysis of many cases were that unless there was improvement under treatment within a week there was danger of permanent impairment of vision, while in cases of more than two months' standing little could be expected except possibly checking the loss of vision by the removal of some definite focus of infection.

*The Amount of Vision.*—It was also found that the demand for early operative interference in total loss of vision was far more imperative than when the loss was but partial.

*Condition of the Fundus.*—When the nerve appears normal one might, with safety, delay operating longer than when there is increasing engorgement or commencing pallor.

*The Virulence and Site of the Infection.*—As in all the other types of infection, so in that producing optic neuritis, the micro-organisms differ greatly in virulence. When the infection is of the virulent type, there is probably considerable exudate about the nerve or even within its sheath. The optic nerve is really not a nerve, but a part of the brain. It is easily destroyed and does not regenerate. Parsons<sup>12</sup> says of it: "The so-called optic nerve, together with certain parts of the retina, constitutes a lobe of the brain, and has therefore the characteristics of the central nervous system. Hence the nerve-fibers are devoid of a sheath of Schwann and the interstitial substance is neuroglia." If the infection is so located that it can be easily discovered and removed, the prognosis is much better than when the site is difficult to locate and, when found, to eliminate.

**The Optic Canal.**—The *size of the optic canal*. That the osseous optic canals normally afford great protection to their neural contents cannot be doubted, but when these canals become narrowed and the enveloping sheath of the nerve, or the nerve itself, swollen, the unyielding walls may cause constriction sufficient to produce atrophy. Piersol<sup>13</sup> gives the diameter of the optic nerve as from 3 to 4 mm. In a canal of 4 mm., therefore, only a moderate swelling would cause a pressure so severe that degeneration of the nerve would rapidly ensue.

The important thing to determine in a study of the optic canal is its size and shape. Adjacent pathology, while always sought, is not so easy to determine, and many cases require surgical interference where it is not evident.

The average diameter of the optic canal, as determined by my measurements in 164 adult skulls, was 5.17 mm.<sup>14a</sup> The canals were usually found to be round irrespective of the way examined, *i. e.*, by holding them up to the light, by the fluoroscope, or the Roentgen ray. The top posteriorly was occasionally flattened and in a few cases the canal appeared distinctly oval with the horizontal diameter usually the larger. Excessive pneumatization of the sphenoid adjacent to the canal is usually associated with diminution of its caliber and a thinning of the bone. The diameter of the optic canal at birth is nearly of adult size, but very short. There is a rather close analogy between it and the annulus tympanicus.

The canal appears in a radiograph of the orbit slightly larger than its actual size. The average size in radiograms, as determined by the filming of 25 doctors and nurses, was 5.35 mm. About 10 per cent. of these canals

appeared oval. In a similar number of cases with optic nerve involvement the canals averaged  $\frac{2}{3}$  mm. less and 50 per cent. were oval. In a correct radiogram of the orbit the canal should appear close to the bony rim in the lower outer quadrant.

In an analysis of 36 cases with optic nerve involvement<sup>14b</sup> it was found that 50 per cent. of the 4-mm. canal cases were unimproved. All the 4.5-mm. canal cases recovered with practically normal vision. Normal vision was also obtained in all the 5-mm. canal cases but two—one had a brain tumor and the other multiple sclerosis.

Total unilateral blindness of sixteen days' duration in a recent 5-mm. canal case, however, did not improve after removing infected tonsil stumps and ventilating the posterior sinuses, so that implicit reliance cannot be

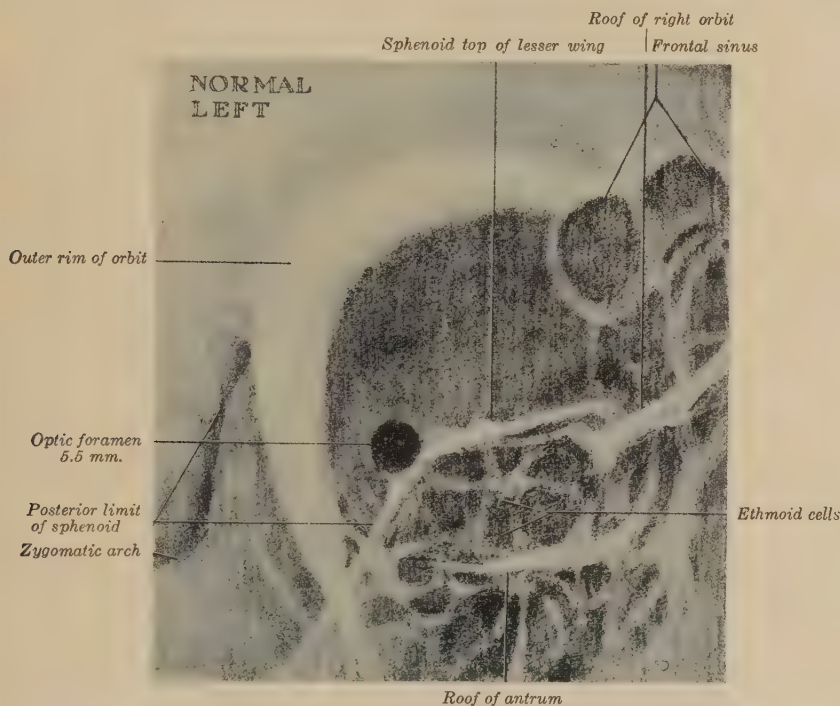


Fig. 96.—The normal optic canal in its correct position and its relationship to the adjacent sinuses and rim of the orbit.

placed on the diameter of the optic canals. Their size and shape without doubt furnish a valuable guide in both prognosis and treatment.

**Treatment.**—The treatment of each case is a study in itself. Some are so mild that spontaneous recovery rapidly ensues. It would be a grave error to submit this type to any unnecessary surgical intervention. Other cases, untreated, may remain permanently blind. As long as the patient has vision which can be checked up at frequent intervals a reasonable delay is justifiable. Local medication may be tried, but if the vision is nil for four or five days the case requires most serious consideration. Every focus of infection which might be responsible for the condition should be eliminated.

While it has been proved in a general way that small optic canals are

found in the more severe types of cases and that, as a rule, the size of the canals indicates the appropriate method of treatment, the mere fact that we have a fairly normal canal does not absolutely preclude the possibility of atrophy. In the small canal cases associated with complete loss of vision early ventilation of the posterior sinuses is indicated, *i. e.*, as soon as a reasonably correct diagnosis can be made. When this total loss of vision occurs with a moderate size canal a little more delay might be permissible, local treatment may be tried, but it would never be advisable to permit a totally blind patient to go more than five or six days without attempting to relieve the engorgement about the nerve by ventilation of the posterior sinuses, unless some other very definite focus has been found.

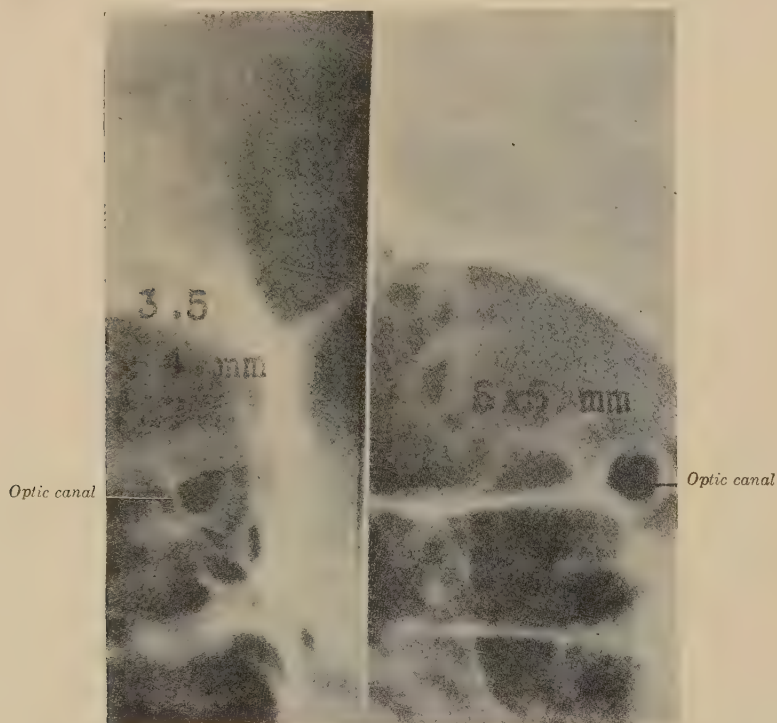


Fig. 97.—Shows the comparison in size between the small and the normal sized canals. There was complete optic atrophy in this case.

Even if there is some discoverable focus, obstruction to the posterior sinuses, if present, should also be eliminated, as an exudate can rapidly produce optic atrophy even in a 5-mm. canal. The focus in every case should be sought, and if found, eliminated, be it in teeth, tonsils, sinuses, or other regions of the body.

The *local treatment* mentioned above is a 3 per cent. spray of ephedrine about the middle turbinate, followed by hot irrigation and argyrol packs. Ointments and an ephedrine spray are also prescribed for home use.

*Surgical Treatment.*—When no definite explanation can be found for sudden and persistent loss of vision, and also in cases with small optic canals and marked loss of vision even when some focus has been found and eliminated, the operation advocated as a therapeutic measure is the re-

removal of the middle turbinate, a larger opening in the front wall of the sphenoid and the uncapping of the posterior ethmoid cell—a simple and comparatively safe surgical procedure to one at all skilled in intranasal technic. The percentages of these cases has from year to year become lower and may eventually vanish. This percentage is now somewhat under ten. In my last 30 cases the sphenoids were opened but twice, and in neither case was it justifiable, as both would undoubtedly have recovered simply by the removal of the focus.

The complete ethmoid exenteration, unless the cells are definitely infected, is never a justifiable or necessary procedure.

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## NEOPLASMS OF THE PHARYNX AND NASOPHARYNX

### BENIGN GROWTHS

Benign neoplasms are rare in the pharynx and the nasopharynx. They include the fibroma, the hemangioma, the papilloma, the lipoma, and the teratoma. The fibroma of the nasopharynx will not be considered here, as it is treated in detail in another section.

**Fibroma** occasionally arises in the posterior wall of the pharynx in the form of a smooth, rounded, sometimes lobulated tumor covered by a nor-

mal mucosa to which it is non-adherent. It may grow to a size sufficient to fill the faucial cavity and markedly to interfere with swallowing and breathing. A neurofibroma the size of a large goose egg was recently seen by the writer arising from the prevertebral fascia. In extreme cases of this kind a tracheotomy may have to be done to relieve dyspnea.

**Lipoma** is extremely rare in the pharynx. The posterior wall, the ary-epiglottic fold or the anterior surface of the epiglottis, and the base of the tongue are the usual sites.<sup>1</sup> It forms a smooth swelling and is non-adherent to the mucosa under which it lies. It may be small, sessile, and unilobular; occasionally it is large, multilobular, and pedunculated. It has the elastic feel of fluid under pressure and for this reason it may be mistaken for a cyst or an abscess.

*Treatment of Fibroma and Lipoma.*—Tracheotomy may have to be done on account of the size and the situation of the growth. As a rule both forms of tumor may be removed quite easily by dissection, as they shell out from their capsules without difficulty. For the smaller tumors the Rose position and incision through the pharyngeal wall is to be preferred. In cases of very large tumors it will be found advisable to approach the growth from the outside through an incision paralleling the anterior border of the sternomastoid. The large vessels may then be retracted and the growth shelled out by blunt dissection with the finger. As these tumors are under the mucosa and are non-adherent to it, their dissection in this way leaves the pharynx unopened.

**Hemangioma** of the posterior pharyngeal wall is occasionally seen in the form of flat, sessile, non-ulcerated growths.<sup>2</sup> The surface is of a bluish color and often has a serpentine lobulation. Spontaneous hemorrhage is not usual, though bleeding is easily induced by trauma. Hemangioma of the nasopharynx is, in the majority of cases, an extension of the same growth arising in the posterior ethmoid region. Occasionally it is primary in the nasopharynx. In this region it is an infiltrating growth and tends to invade the nose and the sinuses and to destroy their bony walls. The symptoms are slowly increasing nasal obstruction and repeated severe epistaxis. It may take years to develop to a point where the obstruction in the nose is complete. The diagnosis is based on the history of frequent nosebleeds, the slowly developing nasal obstruction, and the presence of a friable tumor mass more or less completely filling the nasopharynx.

*Treatment.*—On account of the severe hemorrhage that always takes place when these growths are attacked surgically, radium is the method of choice. Seeds of radium emanation of 1 or 2 millicuries may be directly inserted into the growth, six or eight seeds being used at one sitting. These seeds may be of glass or of gold. The gold seeds are the better, since the screening action of the gold prevents any marked reaction in the tissues. When the tumor is in the nasopharynx it will be found advantageous to place the patient in the Rose position and to plant the seeds in the tumor by thrusting the carrier directly through the soft palate. Great accuracy in placement is obtained in this way. A platinum tube 0.2 millimeter in thickness and containing 10 milligrams of the element radium may be inserted into the growth and allowed to remain for four or five days; or gamma radiation may be employed from the outside.

**Papilloma** is very rarely seen in the vault of the pharynx. It may reach the size of a hen's egg.<sup>3</sup> It causes no symptoms other than nasal obstruc-

tion. When pedunculated it is easily removed with the snare. The base should be treated with radium or with the galvanocautery to prevent recurrence. Sessile papillary growths in this region should be regarded with suspicion until the microscope shows them to be benign. A piece of the deeper tissue should be taken for biopsy, as it often will show well-marked evidence of malignancy when examination of the more superficial parts of the tumor is not conclusive in this regard.

**Teratoma** (hairy pharyngeal polyp) is sometimes found in the pharynx, though the nasopharynx is the usual site. It is a congenital cyst containing skin, hair, cartilage, and bone. It may be removed with the snare and the base cauterized to prevent recurrence.

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#### MALIGNANT NEOPLASMS

Malignant tumors of the pharynx and the nasopharynx are more common than benign growths. They include the carcinoma, the sarcoma, and the malignant chordoma. In the pharynx the carcinoma is more common than the sarcoma. It rarely occurs in this situation before the fortieth year. In the nasopharynx, however, it occasionally is seen in comparatively young people. The writer has had one case in a patient of twenty-three; and Crowe<sup>1</sup> has reported several cases under twenty years of age. In the nasopharynx, sarcoma has been said to be twice as common as carcinoma. The reports of New<sup>2</sup> and Woltman<sup>3</sup> are hardly in accord with this statement, as a majority of their cases were epidermoid carcinomas. Sarcoma is not confined to any age. It occurs as the round-cell (large or small) sarcoma, the spindle-cell sarcoma, the myxosarcoma, the fibrosarcoma, or the lymphosarcoma. The last two are by far the most common. The fibrosarcoma has all the symptoms and gross characteristics of the nasopharyngeal fibroma, including its tendency to excessive hemorrhage and to invasion of the nose and the sinuses. The fibroma, however, has a tendency to spontaneous regression after a variable period of growth, which is by no means shared by the fibrosarcoma. The one cannot be distinguished from the other except by microscopic examination. Their treatment may be conducted along similar lines, for a detailed description of which the reader is referred to the section on Nasopharyngeal Fibroma. The myxosarcoma and the spindle-cell sarcoma may occur in an encapsulated pedunculated form. Such tumors are not highly malignant; they cause few symptoms other than nasal obstruction, possible ear symptoms due to eustachian obstruction, and bulging of the palate. They are easily removed with the snare. Subsequent radium treatment of the base should be given to prevent recurrence. A fair proportion of all the malignant tumors in this region, however, are epidermoid carcinomas and may give no symptoms referable to the nasopharynx; an early diagnosis is, therefore, often extremely difficult. The reason for this is that the growth is small, arising on one side in the fossa of Rosenmüller; and, aside from unilateral pain in the ear, tinnitus, and deafness, does not cause any local disturbance. The earliest other symptoms are the result

of involvement either of the glands of the neck or of one or more of the numerous nerve structures, intracranial or extracranial, that lie in the immediate vicinity. These include the gasserian ganglion, the second, third, and fourth nerves, the second and third division of the fifth, the sixth, seventh, ninth, tenth, eleventh, and twelfth nerves. The symptom-complex, therefore, may be most diverse. It is usually unilateral and consists of various combinations of blindness, due to choked disk or optic atrophy, diplopia and ptosis, headache, pain or paresthesia of the face, pain in the jaw, in the mastoid, occipital or shoulder regions, paralysis of the facial muscles, of the tongue, palate, pharynx or larynx, or of the sternomastoid and the trapezius muscles of the affected side. The sixth nerve is most frequently involved, and diplopia, with or without the trigeminal syndrome, is the most common early symptom. The trigeminal and the jugular foramen syndromes come next in the order of frequency.<sup>3</sup> When there is extension of the growth to the pterygoid region, ankylosis of the jaw may be present. When any or a combination of the above symptoms are present, and in all cases of suspected hypophyseal tumor, the nasopharynx should be examined carefully for neoplasm.

**Carcinoma** of the pharynx is usually of the epidermoid variety. Adenocarcinoma is met with occasionally. The growth may be situated anywhere on the walls of the pharynx, though the lower lateral walls are the ones most often involved. It forms a hard almost cartilaginous swelling which breaks down early in the center, leaving an ulcerated, granular surface covered with mucopus and surrounded by a crater-like rim of very dense consistency. The early symptoms are a vague feeling of fulness or of foreign body in the throat and slowly developing pain. When the growth involves the upper end of the esophagus, dysphagia is a prominent symptom. Pain is rather unusual until ulceration occurs; it then becomes marked, especially on swallowing, and radiates from the site of the growth to the ear of the same side. The glands of the neck may become involved early.

**Sarcoma** of the pharynx may arise either in the posterior or one of the lateral walls. It may show as a distinct tumor which ulcerates rather late or as a somewhat diffuse swelling. Lymphosarcoma is the usual type.

*Treatment.*—In the pharynx the sarcoma should receive radium treatment. Most of the carcinomas are inoperable if they occur low down in the pharynx. When they do not involve the upper end of the esophagus, and especially when they are situated on the posterior wall, diathermy is the most effectual form of treatment.<sup>4</sup> In the nasopharynx, if the tumor is discovered early and if there are no metastases in the neck and no involvement of the cranial nerves, the writer feels that operation with immediate radiation offers the best chance of ultimate success. This does not apply to the fibrosarcoma, in which radiation is the treatment of choice. The operation should be done in the Rose position, the palate split, and every vestige of the tumor removed, after which the surfaces should be thoroughly radiated, the radium being placed in position while the patient is still under ether. For dosage and method of application, see paragraphs on treatment of malignant tumors of the nasal sinuses. In those cases in which metastasis has taken place, and the cranial nerves involved, palliative treatment is indicated. It consists in irradiation, either in the form of the deep Roentgen ray, of radium emanation seeds inserted directly into the growth, or of gamma radiation from the outside. These methods often do much in re-

tarding the rate of growth of the tumor and in diminishing pain and discomfort.

**Malignant chordoma** is a tumor arising from the remains of the primitive notochord of the embryo. The majority of cases arise either at the base of the brain or in the region of the coccyx or sacrum.<sup>5</sup> But 3 cases have been reported arising primarily in the nasopharynx. The growth is non-adherent to the mucosa and may simulate closely a fibroid polyp with a rather broad base.<sup>6</sup> When it does not involve the cranial cavity or any of the cranial nerves, the symptoms are nasal obstruction and bulging of the soft palate. Any or all of the symptoms enumerated above as occurring in the more common malignancies of the nasopharynx may be present, however, when one or more of the nerve structures in the immediate vicinity are implicated. The prognosis is exceedingly grave, though freedom from symptoms for a year after removal has been reported in a case showing no nerve involvement.<sup>6</sup> The treatment is the same as for other malignant growths of the nasopharynx.

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## NEOPLASMS OF THE NOSE AND THE NASAL ACCESSORY SINUSES

### BENIGN NEOPLASMS

Benign growths are rare in the nose and in the sinuses. They include the papilloma, the osteoma, the chondroma, and the osteochondroma, the hemangioma, the glioma, the fibroma, the mixed tumor,\* the adenoma, the lymphangioma, and the lipoma. The fibroma will not be considered here, as the subject is taken up fully in another section.

**Papilloma.**—Papilloma occurs in the nose in two forms, the hard and the soft. The first is occasionally seen, just within the margin of the vestibule, in the form of pedunculated cauliflower growths with hard, almost horny surfaces. It has all the characteristics of the common dermal wart. Papilloma arising posterior to the mucocutaneous junction is usually of the soft variety. The septum anteriorly was the point of origin in a majority of the 45 reported cases,<sup>1</sup> though the inferior or middle turbinate,<sup>2</sup> the frontal,<sup>3</sup> ethmoid,<sup>4</sup> or sphenoidal<sup>5</sup> sinus was each involved primarily in one or more cases. These growths are pedunculated, may be single or multiple, and have the usual cauliflower structure of papilloma elsewhere. They bleed easily when touched with a probe and often spontaneously. Nasal obstruction is usually their only other symptom, though the tendency to

\* The writer has found the mixed tumor a highly malignant one when it occurs as a secondary growth from a primary tumor of the parotid gland or the lacrimal sac. These tumors form metastases rapidly, especially in the bones of the face and the head.

recur after removal, always marked, may be so pronounced and the growth so free as to produce serious pressure symptoms on neighboring parts.

*Diagnosis.*—There should be no confusion between papilloma and the papillary hypertrophy so often seen at the posterior end of the lower turbinate and less frequently elsewhere. Here chronic inflammation has produced an hypertrophy of the normal papillæ of the mucosa which are covered by a normal epithelium. Papilloma, on the other hand, is typically neoplastic and is composed of branching fibrous stalks surrounded by enormously thickened epithelium which is everywhere separated from the fibrous tissue by a perfectly definite basal outline. In malignant epithelioma this basal outline is obscured by the invasion of the epithelium into the fibrous tissue. Papillary tumors occurring in the cancer age and suspected of being malignant may show nothing of this invasion if the piece taken for biopsy is too superficial; whereas the deeper parts of the growth may give unquestioned evidence of malignancy. This is the only serious difficulty connected with the diagnosis.

*Treatment.*—This consists in thorough removal and cauterization of the base with the electrocautery to prevent recurrence. This may occur in spite of all care, and is probably due to implantation during operation. Radium is successful in many cases when used alone; it is an invaluable adjunct used immediately after surgical removal to prevent such implantation. The method is described in the paragraph on the treatment of malignant disease.

**Osteoma.**—Osteoma is rare in the nasal passages and in the sinuses. In the majority of instances the point of origin is the frontal,<sup>6</sup> though the ethmoid, sphenoid, and antrum are occasionally involved primarily.<sup>7-8</sup> It is usually single, but multiple osteomas may occur involving a wide area of one or more of the sinuses or the bones of the face<sup>9</sup>; or several of the sinuses may be included by extension from a single growth.<sup>10-11</sup> It is essentially a tumor of youth, over 50 per cent. of the reported cases occurring in the second decade of life, and the third decade accounting for 30 per cent. of the remainder.<sup>12</sup> Several hypotheses have been advanced regarding its histogenesis; but whether it arises from the bony diploë, from the periosteum, from ossification of the schneiderian membrane, or from ossification of remnants of fetal cartilage is a moot question.

*Etiology.*—Cases that have given the history of more or less severe injury before the tumor developed are too numerous to be considered altogether coincidental. A fair proportion have had co-existing sinus disease. It is probable that in the first group injury is a contributing cause only, the spark that ignites the tinder of a quiescent congenital fault in the bone. In the second group inflammatory disease may act in the same way, though it is quite likely that the relationship is reversed and that the sinus disease results from imperfect drainage brought about by pressure of the tumor.

*Varieties.*—Osteomas are of two kinds, the hard and the soft. The first is of ivory density, which resists any attempt at comminution with the curette, chisel, or rongeur. The second varies in consistency, but usually gives little trouble in its complete exenteration with those instruments. Fortunately these tumors are usually pedunculated and their pedicles easily broken, so that the hard variety, although it cannot be removed piecemeal, may be dealt with successfully *en masse*.

*Symptoms.*—Osteoma grows slowly and may attain considerable size without giving symptoms other than facial disfigurement. Nasal obstruction, suppurative sinus disease with polyp formation, deafness from eustachian obstruction, anosmia, pain, and exophthalmos may be present according to the size of the growth and the parts that receive its pressure. The cribriform plate or posterior frontal plate may be eroded and meningitis or brain abscess ensue.

*Diagnosis* is seldom difficult. The character of the mass, its density, its slow growth together with the above symptoms make it self-evident. Roentgenograms outline the extent of the tumor perfectly.

*Prognosis.*—The prognosis is good unless the meninges have been exposed, in which event the outlook is doubtful.

The *treatment* is surgical removal, which should be done at the earliest possible time before important structures have been destroyed by pressure or the meninges laid bare. When the tumor is confined to the frontal, the usual incision over the brow and removal of the anterior plate is sufficient to give access to the growth. If the antrum is involved alone, the Caldwell-Luc operation may be done. Ethmoid osteoma, whether confined to that region or involving one or more of the other sinuses, may be approached through a lateral incision in the cheek. (Vide description of author's operation for malignant disease of the sinuses.) The size of the bony openings in the maxillary and the ethmoid regions can be determined in the individual case only by the size and hardness of the tumor and the space necessary for its delivery.

**Chondroma and Osteochondroma.**—Both are exceedingly rare. Chondroma of the septum occurs as an encapsulated tumor of the hyaline cartilage type.<sup>13</sup> In the sinuses the osteochondroma is the more usual,<sup>14</sup> though chondroma has been reported.<sup>15</sup> These tumors grow by expansion, and their clinical appearance and symptoms make them indistinguishable from the osteoma.

**Hemangioma.**—The so-called "bleeding polyps of the septum" arising from Kiesselbach's area, or similar growths springing from the floor of the nose or the anterior end of the lower turbinate, may be true angiomas or simple granulations of an exaggerated type. They form sessile or pedunculated tumors, varying in size from a pea to a marble, dark red in color, and of soft consistency. The surface is usually stippled, which gives the growth the appearance of a ripe raspberry. It bleeds easily on palpation and often spontaneously. Nasal obstruction and epistaxis are the only symptoms.

*Treatment.*—Remove with the snare and cauterize the base with the electrocautery to prevent local recurrence.

A much more serious form of cavernous hemangioma is that arising from the lateral wall of the nose in the ethmoid region. It was scarcely mentioned in the literature until Crowe<sup>16</sup> reported 7 cases in 1923. The writer has seen 2 similar cases. These tumors are benign in the sense that they do not form metastases; they are none the less dangerous, since they grow by infiltration and are therefore highly destructive to surrounding tissues. Their great vascularity also gives them a grave importance. They grow slowly, and frequent profuse epistaxis may be the only symptom for years. Eventually they may fill the nose, the sinuses, and even the nasopharynx, and erode away the bony walls of one or more of these cavities. Exophthalmos,

nasal obstruction, and facial disfigurement, one or all may be present. Both of the cases seen by the writer had erosion of the anterior wall of the frontal sinus where a tense, non-fluctuating swelling had in both instances been diagnosed as a lipoma. These tumors are soft, slightly lobulated, and of a dark purplish color.

*Diagnosis.*—This is made on the prolonged history of frequent epistaxis with gradually increasing nasal obstruction, and on the appearance of the tumor. If a piece is removed for biopsy, the bleeding may be difficult to control.

The *prognosis* is doubtful because of the extreme exsanguination that may occur.

The *treatment* may be either by surgical removal or by radium, and of the two, the latter is to be preferred because oft he profuse hemorrhage always encountered when the former is tried. Irradiation may effect a marked decrease in the size of the growth with a corresponding amelioration of the symptoms, or even its complete disappearance. Surgery cannot hope to remove one of these tumors at one sitting; when it is tried the external route is imperative, and the operator should be prepared to stop with much of the tumor still in place. In one of the writer's cases the growth gradually disappeared after such partial removal. Crowe has noted this phenomenon and attributes it to the removal of the point of origin or "nucleus" of the tumor.

**Glioma.**—This is a rare congenital tumor which results from an anomalous embryonic development of the anterior cerebral vesicle or of the olfactory lobe. Histologically it consists of a proliferating neuroglia (the supporting tissue of the central nervous system) together with a small amount of connective tissue. It never metastasizes, but pressure on neighboring structures may make it of serious import. It may be intranasal,<sup>17</sup> extranasal,<sup>18</sup> or a combination of both.<sup>19</sup> The extranasal form appears at the root of the nose as a tense cyst-like swelling, without fluctuation, but with the firm elastic feel of a lipoma. It may be differentiated from the meningocele by the absence of pulsation or of changes in size during effort. Its point of attachment is usually at or near the maxillo-lacrimal suture. The intranasal variety springs from the inner side of the same area, and appears in the naris as a polyp-shaped mass of firm consistency. In the combined form a bony dehiscence in the lacrimal region is filled by the isthmus connecting the external and internal lobes. A patent pedicle sometimes extends to the meninges,<sup>19</sup> which condition constitutes the one possible danger of surgical removal. It may be demonstrated by exploratory puncture of the tumor and the recovery of cerebrospinal fluid in the syringe. Gliomas give rise to no symptoms other than nasal obstruction and facial disfigurement. They are usually safely and easily removed surgically. Though not malignant, they tend to recur locally unless their removal is complete. When there is any suspicion that a connection exists with the brain, radium treatment may be tried. If surgical interference is undertaken, the point of attachment of the tumor should be carefully cauterized to prevent infection of the meninges.

**Mixed Tumors.**—Unlike the simple tumors which contain but one type of differentiated cell, these growths contain types of cells representing two or more of the germinal layers. These may be derived from different layers

or arise from immature fetal rests capable of differentiating into two or more types. They are of slow growth, are nearly always encapsulated, and are easily removed surgically. They are fairly common in the parotid and in the palate, but are rare in the nasal fossæ.<sup>20, 21</sup> They occasionally occur in the nasal sinuses as metastases from the parotid or the lacrimal gland. Under these circumstances they are decidedly malignant.<sup>26</sup>

**Adenoma, lipoma, and lymphangioma** are so rare in the nose and the sinuses that they may be dismissed with a word. A case of benign adenoma arising in the ethmoid,<sup>22</sup> so extensive that it filled the nasal cavity and required a lateral rhinotomy for its removal; 2 cases of lipoma, one of the antrum<sup>23</sup> and one in the naris just under the lower edge of the nasal bone,<sup>24</sup> and 1 case of cavernous lymphangioma<sup>25</sup> involving the anterior end of the lower turbinate, have been recorded.

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## MALIGNANT NEOPLASMS

Malignant neoplasms are fairly common in the nose and the accessory sinuses. The sarcomas were formerly thought to be much more common than the carcinomas, the reported ratio being about two to one. That such a disparity exists in the sinuses is doubtful, as 19 of New's 33 cases of malignant growths of the antrum, and 17 of the writer's 25 cases involving one or more of the sinuses, were carcinomas. Both forms of tumor occur in several varieties, the nomenclature of which has been based on widely different schemes. The first concerns itself with gross cell characteristics such as size and shape, examples of which are the small and the large round-cell sarcoma, the spindle-cell sarcoma, the giant-cell sarcoma, the squamous-cell (epidermoid) carcinoma, and the basal-cell carcinoma. The second is based on the type of cell differentiation in the tumor, and includes

the fibrosarcoma, the lymphosarcoma, the osteosarcoma, the chondrosarcoma, the myxosarcoma, and the melanosarcoma. A third indicates certain types of growth, such as the angiosarcoma and the adenocarcinoma. The psammoma,<sup>1</sup> a rare tumor in the sinuses, receives its name from the sand-like consistency given the growth by calcified hyaline concretions. It does not indicate a type of tumor, but an accidental calcified product of secretion or retrograde process.<sup>2</sup> It is usually a carcinoma. Malignant chordoma,<sup>3</sup> a tumor arising from the remains of the primitive notochord, may appear in the nose only as an extension from the pharynx or nasopharynx (vide Tumors of the Pharynx).

**Grades of Malignancy.**—These tumors vary tremendously in their malignancy, which depends not only on the type but also on certain characteristics of individual tumors of the same type. Those that grow by extension, such as the fibrosarcoma, or the encapsulated tumors, such as many of the spindle-cell and large round-cell sarcomas, are only mildly malignant and rarely form metastases. On the other hand, tumors that grow by infiltration of the surrounding tissues, of which the carcinomas, the lymphosarcomas, the small round cell and the osteosarcomas are typical, are usually highly malignant. The malignancy of a given tumor of any one type depends on two things: first, on the number of mitoses present in the tumor cells; and second, on the ratio of differentiated to undifferentiated cells in the growth. All cell growth tends to differentiate into some one type of cell, osteoblast, fibroblast, lymphoblast, etc. If the rate of growth is slow, this differentiation will be more or less perfect. When, however, the growth is rapid, no time is given for differentiation, which will be very imperfect or may not take place at all. Broders,<sup>4</sup> basing his opinion on the study of 2000 epitheliomas, has graded malignancy on this line: Grade 1, the least malignant, having one-fourth or less undifferentiated cells; and so on through the four grades. Of the tumor types found in the nose the epidermoid carcinoma is by far the most malignant. Other types of carcinoma in this region, on the other hand, may form metastases very slowly, their tendency in this regard being, in the writer's experience, in direct ratio to the amount of reactionary fibrosis (sometimes even bone formation) that has taken place in the growth. This may be so great as to give the tumor a density, and consequently a malignancy, closely approaching that of the epidermoid. Metastases usually first appear in the glands of the neck, though cerebral involvement may take place without any evidence of cervical extension. Metastasis to the dorsal vertebral bodies occurred in one of the writer's cases fifteen months after the removal of the primary growth, a carcinoma with marked reactionary fibrosis and bone formation in the stroma. There was no local recurrence in this case.<sup>12</sup> The basal cell is the least malignant type of carcinoma found in the nose. It also responds better to radiation than any of the others. Lymphosarcoma and the small round-cell sarcoma vary greatly as to metastatic extension. This may occur in the glands of the neck, or even become generalized, early in the course of the disease; or the growth may show for years no tendency to spread except by local infiltration. Metastases in the liver are perhaps more common than in any of the other viscera. The spindle-cell and the fibrosarcomas are the least malignant of all, the latter being in this respect about on a par with the simple fibroma, except in its tendency to local recurrence.<sup>12</sup>

**Age and Location.**—Carcinoma is rare in the nose or the sinuses under the fortieth year. The sarcomas may occur at any age, and although usually considered tumors of youth are almost as common after forty years as before it.<sup>5</sup> The ethmoid and the antrum are the most common points of origin. The frontal is rarely the seat of a primary growth, and even more rarely does it become involved by extension from the ethmoid. Extensions to the sphenoid are the rule in ethmoid malignancies that have existed for any length of time, though primary malignancy in the sphenoid is very rare.<sup>13</sup> The anterior end of the lower turbinate, the septum, and the floor of the nose are often involved primarily, the more usual form of tumor being the fibrosarcoma or the small round-cell sarcoma. In the antrum the epidermoid (squamous-cell) carcinoma is perhaps the most common form of tumor, as of New's 38 cases, 18 were of this variety and primary in the antrum. The same probably is true of the ethmoid, as of the writer's 25 cases 9 were epidermoid carcinomas.<sup>12</sup>

**Symptoms and Diagnosis.**—Malignant tumors of the anterior nares give rise to nasal obstruction, epistaxis, and crusting. Pain is unusual, though there may be a sense of soreness and immobility. When the opportunity is given there should be no difficulty in early diagnosis. The encapsulated tumors of the antrum simulate closely cysts of dental origin. Nasal obstruction and swelling of the cheek or palate, due to expansion of the growth, occur rather slowly. Pain is seldom present, and the diagnosis is usually made only at the time of the operation. The infiltrating tumors are by far the most important from the standpoint of an early diagnosis, on account of their greater malignancy and because the signs and symptoms by which they might be suspected, if not actually recognized, are obscure and often neglected. When the growth is primary in the antrum, pain in the alveolus is usually the first symptom, and is invariably attributed to the teeth, one or more of which may be pulled without relief. Unless a chronic empyema exists, a complication occasionally seen, there is no pus in the antrum or middle meatus at this time. Transillumination and roentgenograms give little or no evidence of trouble. Puncture through the inferior meatus with the suction syringe may show a serosanguineous fluid, sometimes slightly purulent, that cannot be accounted for by a simple empyema. Whether this sign is present or not, any case, especially if within the carcinoma age, with pain in the alveolus that cannot be explained on other grounds, should have the antrum opened through the canine fossa, a thorough exploration made, and the microscope made to settle any doubtful appearances. In no other way may these growths be caught in their incipency. Swelling of the cheek, accompanied by paresthesia (burning or tingling) or actual pain, are the next symptoms. The pain may be neuralgic in character or only a dull ache. Bulging of the naso-antral wall and the antro-orbital plate soon cause nasal obstruction and exophthalmos, the eye being pushed upward. Epistaxis occurs only after the tumor invades the ethmoid or erodes the naso-antral wall and appears in the anterior naris. Pus in the nose is found in a majority of the cases when the growth reaches a size sufficient to block the drainage of one or more of the sinuses. While epidermoid carcinoma invades the palate early, the softer tumors do not seem to have the same predilection, and appear in the mouth only late in the course of the disease. Invasion of the pterygomaxillary fossa is usually a late development. Restricted movement of the lower jaw and deep-

seated pain should arouse suspicion of such invasion, though moderate ankylosis of the jaw may be present when the posterior antral wall is intact.

Primary *tumors of the ethmoid* usually arise in the posterior cells, though the anterior cells in the lacrimal region may be the first involved. When chronic ethmoiditis with polyp formation is present, small neoplasms in this region are difficult to recognize, and are often removed in the course of an ethmoid exenteration and thrown away without examination. Such tissues should always receive a thorough scrutiny, and any that cannot without doubt be pronounced simple polyps should be sent to the pathologist for section. The anterior tumors may give all the symptoms of mucocele, swelling in the lacrimal region, exophthalmos and diplopia, and headache. There may be no bleeding, though epistaxis and nasal obstruction are the classical symptoms of ethmoid malignancy and occur early in a large percentage of all cases. Nasal discharge is not unusual and is due either to ulceration and necrosis of the tumor mass, to a suppurative sinus disease of much older date than the tumor, or to one that results from blocking of the sinus ostia by the growth itself. Pain is variable; in some cases it is absent altogether; in others it is severe, either in the frontal or temporal regions or referred to the eye. Exophthalmos, with the eye pushed forward, outward, and downward, is mild when the os planum is simply bulged by pressure; it becomes extreme when this plate is eroded and the tumor invades the orbital cavity. Diminished vision, even with optic atrophy, may be present. The septum is often either directly involved or eroded and the tumor appears in the opposite naris. Primary *tumors of the frontal* may be mistaken for chronic frontal sinusitis, and the correct diagnosis made only after the anterior plate is absorbed. The posterior plate may be eroded early and meningeal or cerebral symptoms ensue. Pain over the frontal in cases of antral and ethmoid tumors is not necessarily to be attributed to extension to that sinus, as such extension takes place rarely, but to chronic inflammatory changes brought about by lack of drainage. When the frontal is opened at operation, as it always should be if there is any opacity either by Roentgen ray or transillumination, it is usual to find in it more or less pus and polypoid material.

**Gross Characters.**—Ethmoid malignant tumors and those that appear in the nares from the antrum vary much in consistency. The epidermoid carcinoma is lobulated and firm; so too are many of the other forms of carcinoma if much reactionary fibrosis is present in the tumor. Most of these latter growths, however, are soft and succulent, as are the small round-cell sarcoma and the lymphosarcoma. The fibrosarcoma has much of the firmness of the simple fibroma. The majority of these tumors have a bluish-gray color as they are seen in the nares; occasionally they are pink, yellowish, brown, or even black. The melanosarcoma<sup>7</sup> accounts for most of the distinctly dark tumors.

**Prognosis.**—This has always been considered grave if not hopeless. Modern methods of treatment, however, have brought about a change for the better; and although today the outlook cannot be considered good, it is at least encouraging and bids fair to improve with better understanding of methods and technic. The results of New<sup>8</sup> in the treatment by cautery of that most malignant tumor, the squamous-cell carcinoma; the results of irradiation by Roentgen ray of malignant tumors of connective tissue origin,

reported by Portman and LaChapelle<sup>9</sup>; and the writer's experience with both sarcomas and carcinomas, treated by thorough operative removal of the growth followed by immediate radiation through a wide open wound,<sup>10</sup> all tend to make the prognosis hopeful, even in some of the most neglected cases. In the individual case an opinion must be based on a number of considerations. In general, tumors that have metastasized are inoperable and admit only of palliative treatment by radiation, which may retard the growth or diminish pain. Operation may be done on some of these cases, however, when the metastases are of limited extent, and of short duration

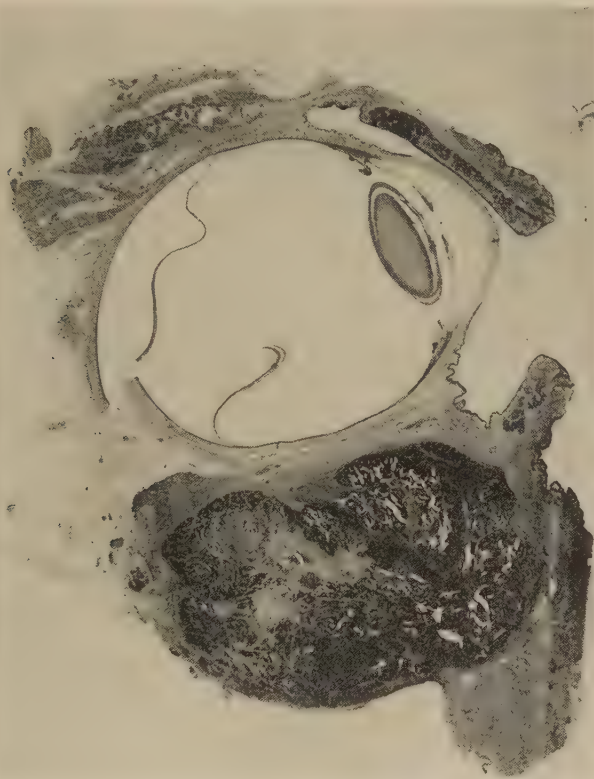


Fig. 98.\*—Basal cell carcinoma involving the ethmoid, antrum, and orbit; the orbit was filled with tumor from the posterior ethmoid which does not show in this section; no recurrence after twenty-eight months.

in the glands of the neck; the cervical lesions being dealt with later, either by complete dissection or by radiation. It may also be done to relieve pain and distress even when hope of permanent cure has been abandoned. The sarcomas are less malignant than the carcinomas; and of the sarcomas, the fibrosarcoma and the encapsulated round- or spindle-cell tumors are usually only locally malignant. A good prognosis may be given. In the lymphosarcoma and the small round-cell sarcoma the prognosis should be guarded, though good results may be obtained in many cases. The malignancy of carcinoma varies within wide limits, which have been discussed in a pre-

\* Figs. 98 to 114, inclusive, are reprinted from the author's article on Malignant Tumors of the Nasal Sinuses, in *Archives of Otolaryngology*, August, 1927.

vious paragraph. The prognosis should be based on the considerations there outlined.

**Treatment.**—The treatment of malignant disease of the sinuses demands close co-operation of the surgeon with the radiologist and pathologist.

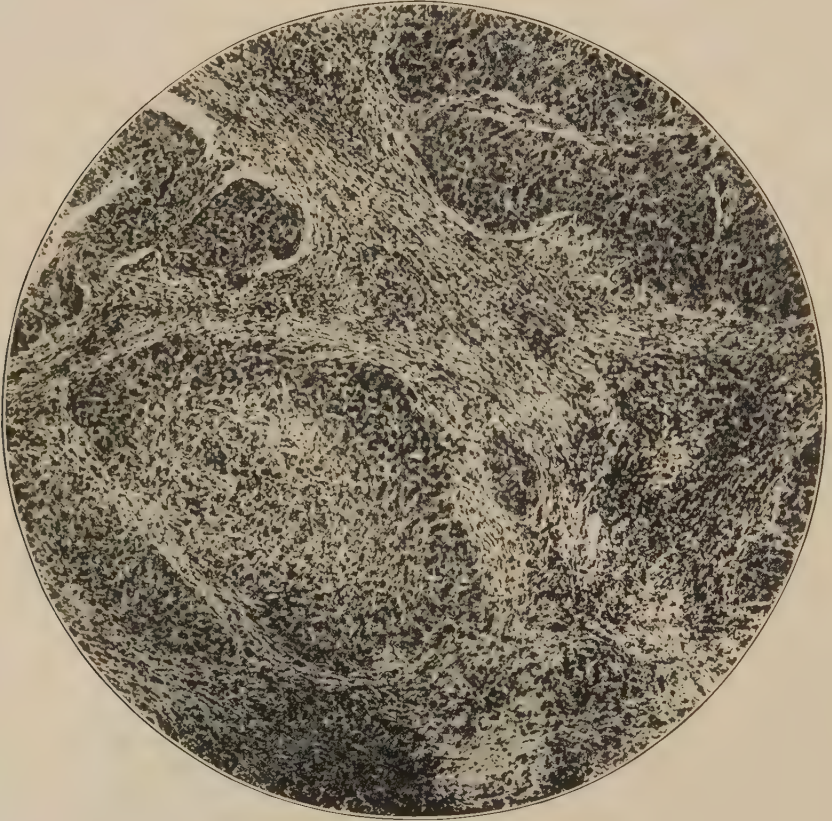


Fig. 99.—Basal cell carcinoma ( $\times 120$ ). Section taken from tumor shown in Fig. 98.



Fig. 100.—Showing orbit, ethmoid, and sphenoid.



Fig. 101.—Same patient as in Fig. 100; a chamois patch is kept in place with collodion.

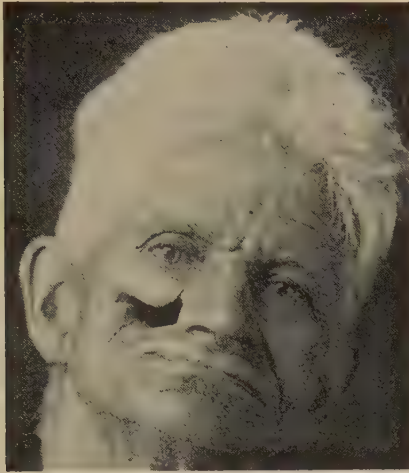


Fig. 102.—Highly malignant carcinoma (no cell differentiation) of antrum, ethmoid, sphenoid, and orbit; one of the 3 cases in which the eye was saved; no recurrence after two years and nine months.

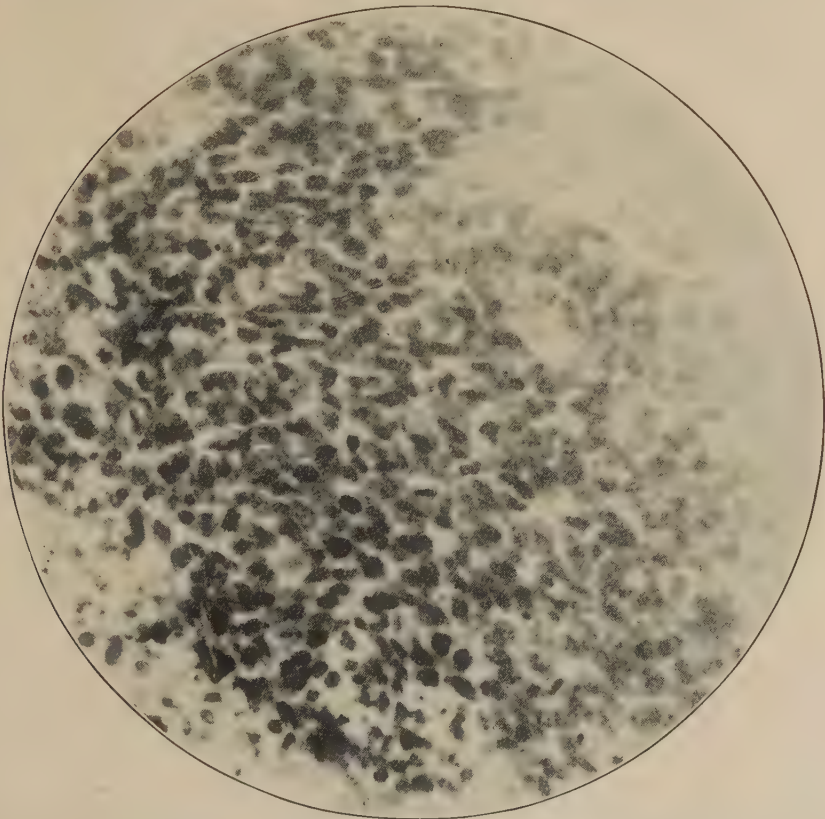


Fig. 103.—Highly malignant carcinoma (no cell differentiation;  $\times 500$ ); patient shown in Fig. 102.

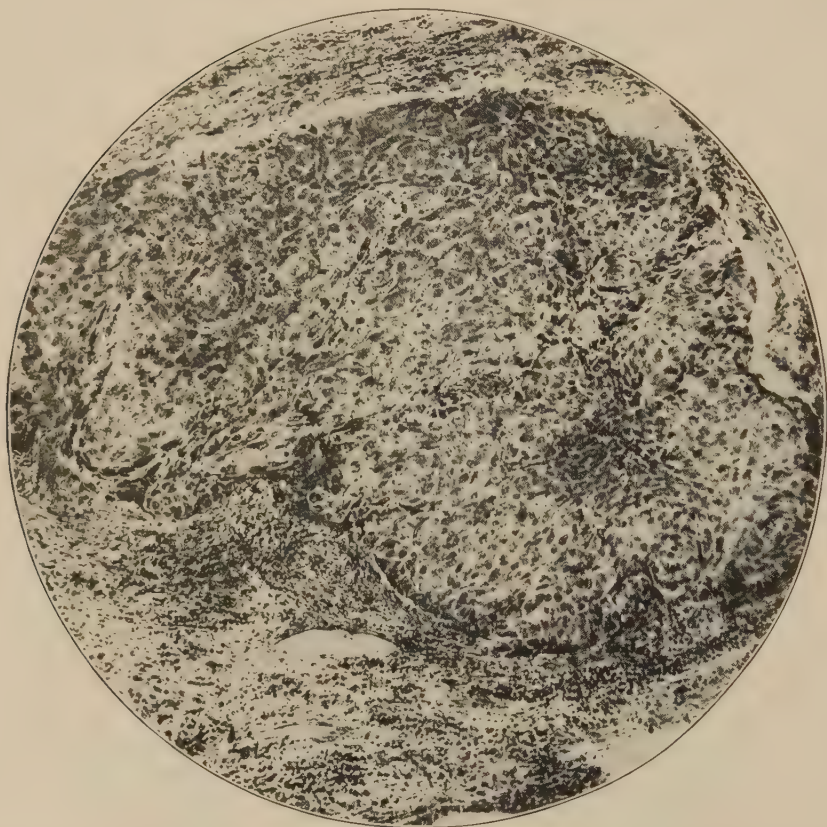


Fig. 104.—Epidermoid carcinoma ( $\times 120$ ) of moderate malignancy; patient shown in Figs. 105–107.



Fig. 105.—Tumor involved the left palate, antrum, ethmoid, sphenoid, and orbit; the right sphenoid and posterior ethmoid; the left sphenomaxillary fossa, malar bone, temporal muscle, and temporal fossa; the deformity of the nose is an old one and was not caused by the operation; patient well and without recurrence two years and ten months after operation.



Fig. 106.—Same patient as in Fig. 105; showing the tumor of the palate and alveolar process.



Fig. 107.—Same patient as in Figs. 105, 106; showing the patient without the plate which is being made under the direction of Dr. Kazanjian; this plate will cover both the opening in the palate and the opening in the face.

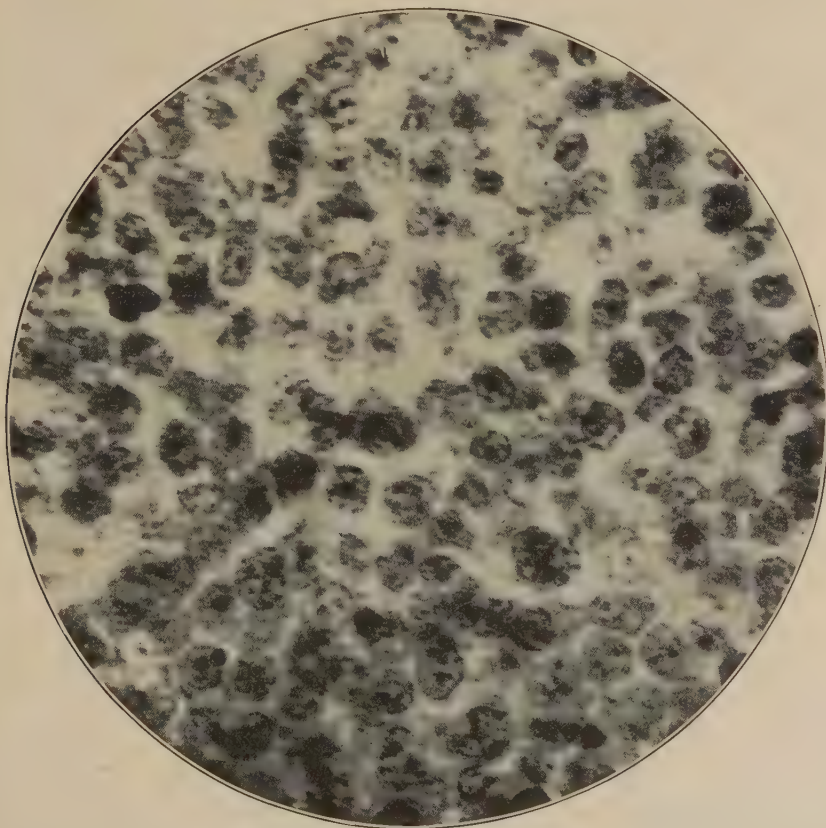


Fig. 108.—Lymphosarcoma (highly malignant;  $\times 1000$ ); tumor involved the right antrum, ethmoid, sphenoid, and orbit. The patient was well and without evidence of recurrence at the time of his death from cerebral hemorrhage five years after operation.

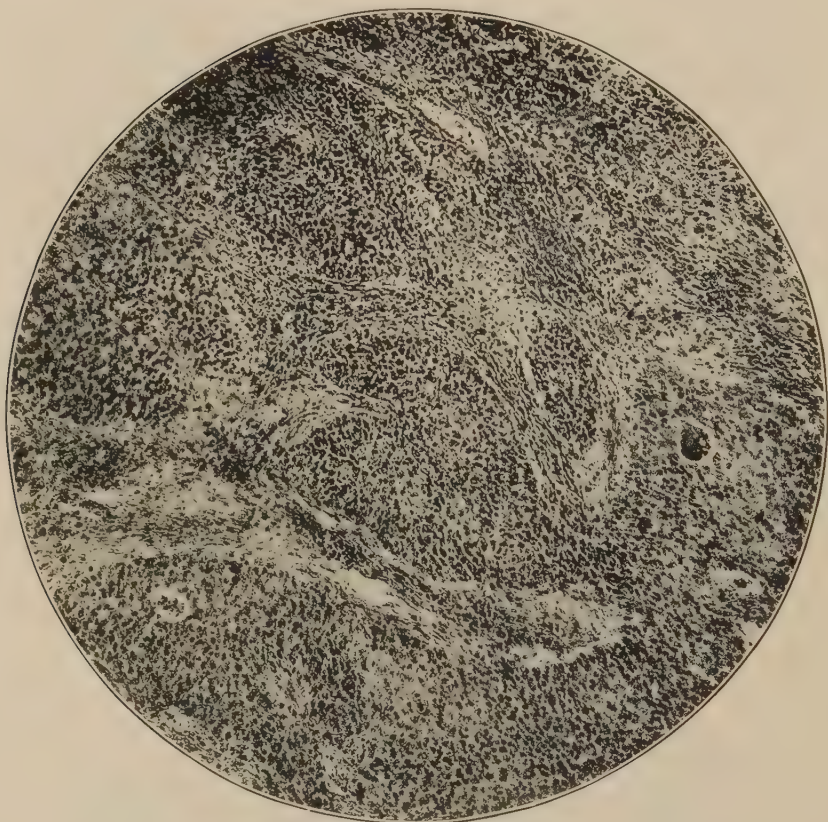


Fig. 109.—Highly malignant epidermoid carcinoma ( $\times 120$ ); tumor involved the left antrum, ethmoid, sphenoid, and orbit. Patient shown in Fig. 110.



Fig. 110.—Tumor involved the antrum, ethmoid, sphenoid, and orbit; one of the 3 cases in which the eye was saved; well and without recurrence six and a half years after operation; the plastic operation on the cheek was done fifteen months after the removal of the growth.

This does not mean that he must be an expert physicist, but that he should have a good working knowledge of the theory of radiation and a wide practical experience in its application, gained under the guidance of an expert. This is especially true in relation to postoperative radiation. The operator is the only one who knows the weakness of his own operation, the ins and outs and ramifications of the disease that he has removed. Be he ever so expert, the radiologist who attempts the treatment of a case without this knowledge will not be as successful as the man who has it. A great difference of opinion exists as to the advisability of removing a piece of the tumor tissue for diagnosis. The writer feels that this should

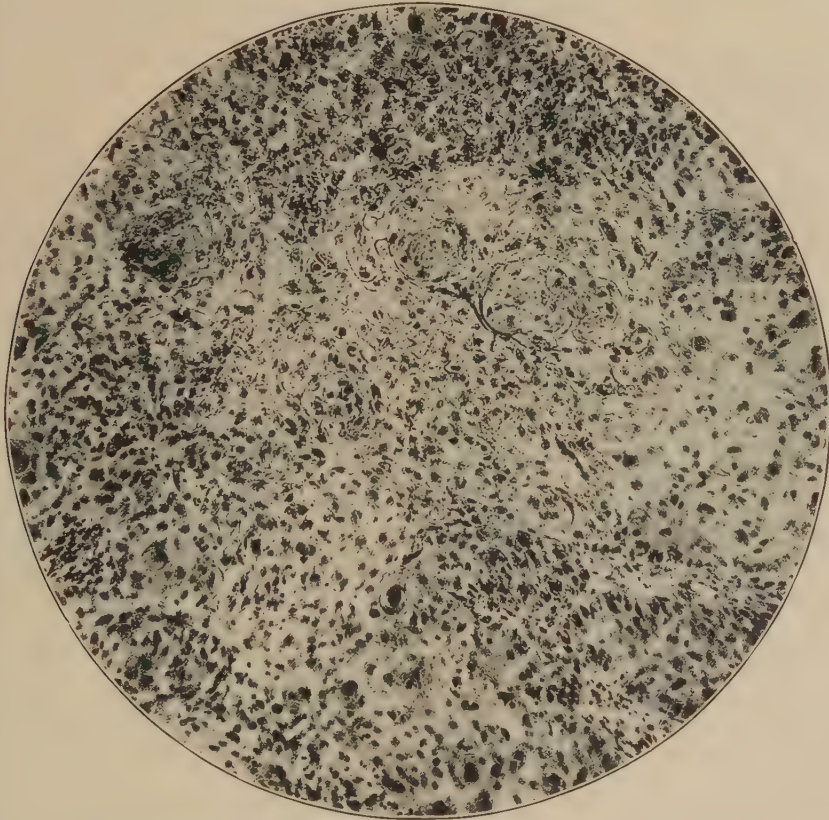


Fig. 111.—Highly malignant epidermoid carcinoma ( $\times 120$ ) showing slight attempts at pearl formation; patient shown in Figs. 112, 113.

be done in all cases; that the knowledge gained is indispensable to intelligent treatment; and that the danger of setting up metastases, at least from tumors in the region under consideration, is slight. Immediate cauterization of the cut surfaces will reduce this to a minimum. The pathologist should make his report as quickly as is consistent with a dependable diagnosis.

Three methods of treatment are available: first, irradiation, either with radium or the deep Roentgen ray; second, cauterization with soldering irons or by diathermy as used by New in tumors confined to the antrum;

and third, operative removal followed by immediate radiation while the patient is yet under ether, as practised by the writer for the past ten years. The last method was much facilitated by deliberately cutting away enough of the soft tissues of the cheek or lacrimal region to make the whole wound a surface one, which rendered subsequent inspection and radiation, or detection of the slightest recurrence, as easy as if the lesion were a dermal one. Radiation alone has given good results in certain classes of tumors, notably the sarcomas and particularly the fibrosarcomas. It cannot be depended upon, however, as many cases fail to respond satisfactorily or develop metastases during the necessarily long course of treatment. It has seemed to the writer that all of these cases except the fibrosarcomas should be operated upon; that the sooner the growth is removed, the better the chances of ultimate success; and that radiation should be reserved for post-



Fig. 112.—Epidermoid carcinoma (Fig. 111) involving the right antrum, alveolar process and palate, ethmoid, sphenoid, septum, and orbit; one of 3 cases in which the eye was saved; the turbinates on the left side show through the opening; patient well and without recurrence ten years and nine months after operation.



Fig. 113.—Same patient as in Fig. 112; she wears a dental plate to cover the opening in the palate; the opening in the cheek is filled with a piece of oiled paper—a device of the patient, who does not wish to have a plastic operation.

operative use. It may be tried, however, in some of the connective-tissue tumors that do not demand immediate removal to save important structures.

New's<sup>9</sup> method of cauterization, or rather slow cooking of the tumor by means of soldering irons, followed by radium treatment, has given excellent results in tumors confined to the antrum. Since 1924 New has substituted diathermy for the soldering iron. The avenue of approach is through the palate if the growth presents at that point; or above the alveolus, as in the Denker operation, if it does not.

The writer's method<sup>10</sup> of treatment is applicable to tumors of almost any extent in the sinuses and is designed to overcome certain difficulties of operative procedures in this field. In order to avoid the dangers either of implantation or of metastases, it is axiomatic in operating on any malignant disease that the tumor should be handled as little as possible, and

should be removed by incision through the surrounding normal tissues, the growth itself remaining intact. It is obvious that the only operation on malignancy in the sinuses that meets these requirements is the resection of the upper jaw as done by the general surgeon. This operation is to be avoided, as it is applicable only to tumors that are confined to the antrum, is unnecessarily disfiguring, has had a high operative mortality, and has not given satisfactory results. The rule must be ignored in operations on the frontal, ethmoid, or sphenoid, as the tumor not only cannot be taken out intact, but must be removed piecemeal by a process of exenteration. Added to this is the difficulty of detecting recurrence or of effective radiation in a cavity so extensive, some parts of which cannot be seen without the aid of a nasopharyngoscope. To counteract these inherent evils immediate radiation is applied while the patient is under ether, and the wound converted into a surface one by the removal of a skin flap over the antral or ethmoid regions as the case may require. These openings are not highly disfiguring\* and they allow a perfect view of the operative field, either for the purpose of detecting recurrence or for such subsequent treatment as the case may demand, either by radiation, diathermy, or cutting operation. A plastic operation may be done to cover the opening when recurrence has not taken place during a period of three years. In the less malignant tumors these openings should not be made.

**The Operation.**—This is based on Moure's lateral rhinotomy,<sup>11</sup> though the external incision has been modified as shown in Fig. 114. Its lower curve just misses opening the buccal cavity. Through this sweeping incision the anterior walls of the frontal, ethmoid, and antrum may be widely exposed and as much of them removed as is necessary in the individual case. The operation is always a bloody one, so much so that a preliminary tying of the external carotid is often done. This is usually not necessary, however, as the bleeding may be absolutely controlled by packing, and diminishes rapidly in inverse ratio to the amount of tumor tissue removed. The nasopharynx is first securely packed. This should be done with care, as it is of great importance that the pharynx should be free from blood. The ether, up to this time, is given by the ordinary cone method. A Rocci tube, inserted through the mouth into the lower pharynx, is now substituted, a strip of gauze being packed lightly around it in the pharynx to



Fig. 114.—The initial incision; epidermoid carcinoma involving the antrum, ethmoid, sphenoid, and orbit; dura exposed during operation; no meningitis; died (date unknown) of extensive recurrence.

\* During the last two years it has been my practice not to remove any of the soft tissues of the cheek, but to depend on keeping the wound open by means of gauze packing during convalescence and later by a stopper of dental molding compound which is pressed into the opening in the cheek while it is dilated with a nasal speculum. This plug maintains an opening very nearly the size of the front wall of the antrum and, while it is not as satisfactory as the larger openings, the ease with which a plastic operation may be done and the almost total absence of the subsequent deformity make this procedure more desirable in the majority of cases.

hold it securely in place. The writer has recently had a ring attached to one of the arms of a Jansen mouth-gag, through which the Rocci tube is passed. When its end is in the correct position in the lower pharynx it is held securely in place by two screws which hold the tube tightly in the ring. The position of the patient is semirecumbent. When the sinus walls have been well exposed, all the upper parts of the wound are packed, the front wall of the antrum removed, and the main mass of the tumor in the antrum, ethmoid, and sphenoid exenterated as rapidly as possible. In some of the softer tumors the finger is the best instrument for doing this. In the ethmoid and the sphenoid the tonsillar ring-punch is an invaluable instrument wherever space will allow of its use. The various curets are all serviceable. When the main mass of the tumor has been removed by these means, all excessive hemorrhage stops and the operator may be more deliberate about opening the frontal and the anterior ethmoid. Practically regardless of anatomical considerations all tumor tissue and all necrotic or soft bone should be removed. The bone is usually not directly involved except in sarcomas of bony origin. It seems rather to undergo a pressure necrosis or a necrosis due to the cutting off of the blood-supply. When all soft bone is removed, the remaining bony margins show little or no tendency toward malignant recurrence. When parts of the antro-orbital wall or of the os planum are gone, but the orbital tissues not invaded, the eye may be left, postoperative radiation of the parts being depended upon to check extension in that direction. The orbital fat is very resistant to infiltration by the tumor growth and, provided all of the gross tumor may be removed from the orbit without taking away too much of the supporting framework of the eyeball, it has been my custom not to remove the eye at the first operation. This does not subject the patient to any great extra hazard, as an exenteration may be done at any time whenever there is evidence of recurrence in the orbit. I have had 3 cases of this kind in which the eye was saved and in which no recurrence has taken place after periods of two, six, and ten years respectively. It is most important that no macroscopic tumor tissue remain in place at the end of the operation, since postoperative radiation is designed only to devitalize microscopic tumor cells that may have been implanted in the wound or set free in the lymphatics to become latent sources of metastases. Whenever possible, a small margin of healthy tissue should be removed. In the sphenoid and the ethmoid this is impossible, but it may be done without danger to important structures in the alveolus, the palate, and the septum. When all tumor tissue has been removed, a V-shaped flap is cut from the cheek,\* and the cavity lightly packed with gauze in the center of which the radium is placed.

**The Radiation.**—This should be begun immediately. In my earlier cases a tube of radium emanation, of a strength equal to 35 or 40 mg., screened with steel, was placed in the middle of the packing at the close of the operation. This gave an additional screening of at least 1.5 cm. of gauze, which was sufficient to prevent any actual burning of even the nearest surfaces. This tube was allowed to remain in place throughout convalescence. As the strength of the emanation is reduced by one-sixth every twenty-four hours, I have roughly calculated that these patients received a total dosage of something over 4000 millicurie hours of gamma

\* *Vide* note on p. 179.

radiation. In my later cases the dosage has been about the same, but platinum iridium tubes of 0.2 mm. thickness have replaced the steel tubes. Four tubes are used, each containing 10 milligrams of the element radium. Additional screening is obtained by placing the platinum tubes in a brass cylinder of 0.5 mm. thickness or within lead foil of 2 mm. thickness.

**Results.**—The operative mortality has been 16 per cent. The cases include every malignancy of the nose and the sinuses showing no evidence of metastases that the writer saw during a period of eight years ending in July, 1926. There were 17 carcinomas and 8 sarcomas. Seven of the carcinomas and 6 of the sarcomas are well and without local recurrence or metastases after periods of from two years and four months to ten years and seven months (52 per cent.).

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#### NASOPHARYNGEAL FIBROMA

**Pathology.**—The typical fibroma is a tumor composed mainly of connective tissue. While their structure varies greatly, they usually show fibroblasts, fibrils, blood, and lymph-vessels. The cells of the true fibroma are usually larger and more numerous than in adult connective tissue. In actively growing fibromas the chromatin content of the nuclei is also increased and the tissue has a distinctly neoplastic character. The tumor is composed of dense fibrous and elastic tissue which rarely shows calcification, cartilage, or bone. The cells are round-, spindle-, or star-shaped fibroblasts which are rather scanty except in certain foci of young connective tissue where they may be so numerous as to suggest fibrosarcoma. These tumors are very rich in blood-vessels, thin walled, and sometimes of large size. The surface of nasopharyngeal fibroma is covered by a thin mucous membrane, but there is nothing in the nature of a true capsule.<sup>1</sup> The blood-vessels being embedded in fibrous tissue do not normally collapse when cut, hence they may bleed profusely, even although small in size.

The presence of areas of spindle-cells, round cells, and sometimes giant-cells at once removes the growth from the class of pure fibroma and places it either in doubt or in that of the sarcoma.

**Point of Origin.**—Fibroma may originate from any part of the fibrous tissue of the nasopharynx. The most common origin is from the periosteum of the anterior part of the base of the occipital bone and the sphenoid bone,

behind the roof of the nasal cavities, and from the superior cervical vertebræ. It may also arise from the sphenothmoidal, the pterygomaxillary, or the tubal regions. In my own experience those of the basilar type have largely preponderated. The base of these growths is usually small, but it may have broad attachments, and if not treated early the tumor may extend itself by secondary insertions which may invade neighboring cavities and by pressure seriously injure or even destroy their walls, although malignant infiltration of adjacent parts characteristic of sarcoma does not take place nor is there any associated enlargement of the neighboring lymph-glands, or any metastasis.

The typical nasopharyngeal fibroma is a rare neoplasm. It occurs chiefly in males, rarely in females, between the ages of ten and twenty-four years, which period marks the time of the maximum activity of the development of the growth. After the eighteenth year the activity may decline and the growth retrogress to the extent, in some cases, of its entire disappearance. Spontaneous cure, however, is rare and by no means to be depended upon, especially since in many cases long before a retrogression can be hoped for the conditions may become very serious or even fatal. Tumors of the nasopharynx occurring in younger children are apt to be adenoid in character.

**Symptoms and Diagnosis.**—In its early stages nasopharyngeal fibroma may not make itself evident until it has attained sufficient size to cause obstruction to nasal respiration, either by its presence or by the excess of secretion which it excites, and at the same time to occasion characteristic changes in the quality of the voice. Rhinoscopic examination or direct inspection by elevating the soft palate may not distinguish it clearly from other forms of growth, although as contrasted with lymphoid hypertrophy its surface is usually uniform and smooth. Occasionally it is slightly lobulated. Palpation instantly discloses a marked difference in that the fibroma is hard to the touch, while the adenoid is a soft mass which yields readily to pressure. Palpation, however, cannot differentiate it from sarcomatous forms of growth. Sometimes a small fibroma has been mistaken for an adenoid and its true character only recognized at the time of attempted operation.

Bleeding may occur early in the history of the fibroma; later, it is almost certain to be present—an important feature—becoming more frequent and more abundant with the growth of the tumor. Surface ulceration may occur early. Both bleeding and ulceration may be present with sarcoma. Unlike sarcoma, pain is not usually an early symptom. “Not malignant in fact, but practically so in effect,” the growth may invade adjacent parts and cavities of the skull, including the nasal sinuses, the orbit, and even in some cases it may penetrate into the brain cavity itself, and by pressure cause serious injury and death. Invading the nasal cavities and their sinuses, it may create separation of the nasal and the superior maxillary bones, thus causing the deformity known as “frog-face”; or it may invade the orbital region, causing exophthalmos, limitation of the field of vision, or even irremediable atrophy of the optic nerve. By pressure upon the eustachian orifices it may cause deafness. The effect upon the nutrition and general vitality of the patient may be pronounced on account of the obstruction to respiration and the consequent deficiency of oxygen, the loss of blood, the difficulty of deglutition, the presence of excessive secretion, and the general discomfort produced.

When the growth has attained sufficient size to cause pressure upon nerve-trunks or ganglia, pain, often of an intense character, may follow. Meanwhile, the attacks of bleeding may become more frequent and more profuse, so that between the neuralgic pain and the oft-recurring loss of blood, together with the other conditions mentioned above, the patient's strength is rapidly exhausted. In such a case relief is imperative. The longer delayed, the less favorable the case for treatment.

The diagnosis of nasopharyngeal fibroma may be difficult; other forms of growth, especially of the sarcomatous varieties, sometimes closely resembling it even under the microscope, where the lines of demarcation between them may be obscurely defined. Bleeding and ulceration of the surface may occur in both; but while sarcoma may develop in childhood it is far more common in middle life, contrary to what is true of fibroma, while the tendency of sarcoma to infiltrate neighboring tissues is absent in fibroma. The more rapid growth of sarcoma and the consequent relatively early development of its characteristics would generally render the diagnosis less obscure as time passes, were not time an important factor in cases demanding such prompt recognition and aid.

**Treatment.**—The story of the early methods of treatment of nasopharyngeal fibroma, begun nearly two centuries ago, is one of the tragedies of surgical history. To the adventurous surgeon such a tumor offered an irresistible temptation. Within the time of the last generation such cases were treated by methods involving extensive resections of the bony structures anterior to the pharynx, including resections of the nose, of the hard palate, and of the superior maxilla. These resections, "preliminary operations," as they were called, followed by attempts to remove the growth by excision, avulsion, or the cold *écraseur*, were often attended with terrific hemorrhages and severe shock, the patient not infrequently dying upon the table or within two or three days after the operation. I have seen many such instances in the hands of distinguished operators.

Many modifications of the surgical procedures were introduced, until finally the method of electrolysis was suggested by the French surgeon Nélaton. Acting upon this suggestion, Voltolini and Michel experimented with electrolysis and found that by its use, particularly when applied near the base of the growth, the volume of the tumor could be materially reduced through restriction of the blood-supply, so that in tumors even of large size and great vascularity the conditions could be improved to such an extent as to permit the successful application of the galvanocautic loop. The method of Voltolini was adopted by Lincoln of New York, who improved the technic and demonstrated the value of the procedure upon a considerable number of carefully studied successful cases.<sup>2</sup>

In the application of the electrolytic current either of two methods, the unipolar or the bipolar, has been employed. By the former, progress is slow and the effect of the current apt to be painful. The patient sometimes suffered severely from it. The bipolar method is less painful and is more rapid and positive in its destructive effects. The current should be of decided strength, namely, from 80 milliamperes to a degree limited by the tolerance of the patient—in my experience considerably less than has been recommended by some European authorities. For the reduction of the tumor many applications of the treatment may be necessary, the number of them depending on the size and histological characteristics of the

growth. The disparity between certain cases is remarkable. As long as the applications were evidently causing shrinkage, the electrolytic treatment was continued, at least to a point where a maximum result was secured. Following this, the growth was removed by the incandescent loop.

The advantage of the cautery loop over the cold *écraseur* is evident, since notwithstanding the extraordinary toughness of the fibrous tissue through which the wire must pass it can be made to progress at the will of the operator. If the proper degree of heat is applied, bleeding is almost sure to be prevented. Remembering this, the loop should be kept at such a temperature that it will make its way slowly and steadily through the extraordinarily dense tissues. Upon the skilful and accurate adjustment of the loop to the base of the growth much of the success of the operation may depend. With single pear-shaped tumors having bases easily reached little trouble should be experienced; where the base is large and excessive prolongations have taken place into surrounding cavities, encircling the pedicle may be almost impossible. Even in such cases the exercise of great judgment and skill has produced brilliant results.

Fletcher Ingals of Chicago modified the Voltolini method by substituting for electrolysis deep-injections of lactic acid into the substance of the tumor. The use of monochloroacetic acid has also been suggested for this purpose.<sup>3</sup> Following the subsequent shrinkage of the growth, its removal is effected by the incandescent snare. For the prevention of hemorrhage at the time of the removal of the growth, injections of adrenaline have been used.

The success of the Voltolini method and its modifications was most gratifying. Hemorrhage and shock were abolished, the base of the growth was profoundly affected by the caustic action of the loop, and recovery was generally rapid and satisfactory, the patient being kept under observation, and any remaining tissue or sign of recurrence carefully recognized and treated.

A comparison of the three methods of treatment mentioned leaves no doubt as to which is to be preferred. Thus, of 27 cases removed after severe preliminary operation, in which the end-result was stated, 59 per cent. were cured and 26 per cent. died. Of 41 cases surgically treated through the natural passages, 95 per cent. were cured and 5 per cent. died. Of 66 cases treated through the natural passages by electrical methods, 100 per cent. were cured or improved. None died.

But the electrolytic method requires especial skill, knowledge, and judgment on the part of the operator. With the general surgeon it was unpopular, notwithstanding its proved advantages over the harsh surgery of the past. Doyen, an eminent surgeon of Paris, entirely discarding it, reverted to the old method of avulsion through the natural passages. His views were widely adopted and the method of Voltolini was promptly forgotten. The progress of the really scientific treatment of fibroma was thus retarded for many years.

The use of radium for the cure of nasopharyngeal fibroma was suggested a number of years ago.<sup>4</sup> The idea has been slow of acceptance doubtless because of the scarcity of material to work upon. Few cases were reported, even from institutions having large supplies of radium and treating many patients for other forms of new growth. Silence on the subject has at last been broken by the reports of several successful cases,<sup>5</sup> notably a series of 32 treated at the Mayo Clinic.<sup>6</sup> The radium was applied by

three methods. In the first and early cases a T-shaped lead applicator with a 50-mgm. tube of radium in the trough of the T was held in various positions against the tumor in the nasopharynx for ten to fifteen hours. It was difficult to apply the radium accurately in this manner, and there was severe reaction in the structures around the tumor, especially in the soft palate. Such complications may be prevented by protecting the posterior surface of the palate with a lead retractor, made of 2 mm. of lead covered with a finger-cot, and by more accurate dosage. In the second group of cases steel points containing the radium emanation or the element were inserted directly into the tumor. Three or four points are usually inserted, depending on the size of the tumor, about the same amount of the radium being used as in the initial dose. In the third group, emanation seeds averaging 5/10 to 1 millicurie each were planted directly into the tumor, the number depending on its size. Such treatments are repeated within six weeks to two or three months, according to the reaction and the result of the previous treatment. The dosage of the secondary treatment varies greatly, depending on the progress of the case. The number of applications required in the finished cases varied from two to nine, averaging between five and six. The average length of time was about fourteen months. Several patients had been operated upon elsewhere, but with much bleeding; the radium seemed to have controlled this. The crusting and scabbing secondary to radium treatment may be cared for symptomatically by the use of oil sprays, potassium iodide internally, etc. Eleven of these patients (73 per cent.) were less than twenty-three years old when cured; and 4 (6½ per cent.) were over twenty-three.

At the Memorial Hospital, New York, radium has been used successfully in a number of cases. The fibroma, slowly progressive, permits of rather slow, prolonged radiation. It is recommended to treat them with heavily filtered radium in rather small amounts over a considerable period of time. This results in a gradual shrinkage without necrosis of tissue, and seems to offer the best form of treatment for the majority of this type of growth.

The effect of radium treatment seems ideal.<sup>7, 8</sup> The shrinkage of the growth is quietly and successfully effected without the various disadvantages of the other methods of treatment, these including the dangers and disfigurements of the old surgery, the danger to important blood-vessels outside of the tumor, the severe hemorrhage from the growth itself common to both the older and the newer surgical procedures, and the pain and tediousness of the many applications of electrolysis which were often required.

Even if in some cases the growth is not completely eradicated by it, the holding of it in check until the advancing age of the patient shall have played its part in causing atrophy will be an advantage.

The application of the Roentgen ray has been tested in a number of recorded instances with satisfactory results.<sup>10, 11</sup> Schempp reports 16 cases, 9 of which were cured and 7 improved.<sup>12</sup>

Surgical diathermy or, as more accurately described, endothermy<sup>13</sup> was originally applied by Cook<sup>14</sup> of New York and has recently been advocated and used with success in various types of growth in the upper air-passages. It is deserving of serious attention, especially where radiation is not available, since it is easily applied, causes little if any pain, and is bloodless.<sup>15</sup>

Evidence of the value of radium and of endothermy in nasal fibroma is slowly accumulating. In its treatment and in that of growths of the less malignant type they are valuable resources. The most experienced observers express unqualified approval of them, as compared with other methods.

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### CHRONIC GRANULOMATA OF THE NOSE AND NASAL ACCESSORY SINUSES

**Definition.**—Tumors or tumor-like nodules made up of granulation tissue.

**Synonyms.**—Bleeding tumors; proud flesh; chronic infectious inflammations; specific granulomata; chronic specific inflammatory processes; infectious granulomata.

**Etiology.**—The cause depends upon the underlying disease, the infecting organism or organisms. The primary organism may be and often is difficult to determine. Secondary infections of granulomata of the nose and nasal accessory sinuses are common. Mixed infections are frequent. The causes of granulomata are probably easiest to determine in the first six of the following:

1. *Syphilis*; 2, *tuberculosis*; 3, *lupus*; 4, *actinomycosis*; 5, *carcinoma*; 6, *sarcoma*; 7, *trauma*; 8, *pyogenic infections*; 9, *mixed infections*; 10, *glanders*; 11, *foreign bodies*; 12, *leprosy*; 13, *rhinoscleroma*.

Kyle<sup>1</sup> believed syphilis, tuberculosis, actinomycosis, glanders, leprosy, and rhinoscleroma to be the causes of granulomata. Other important causes should be added, as the preceding classification shows.

*Syphilis*.—The finding of the *Spirochæta pallida* in bits of tissue removed from granulomata readily determines the etiology. Evidence of lues in other parts of the body, a positive blood or spinal fluid Wassermann test, even when the spirochete cannot be found in the granuloma, helps to determine the cause. The possibility of a granuloma from other causes, even in a syphilitic patient, must always be borne in mind. In the absence of other confirmatory evidence the therapeutic test may help.

*Tuberculosis*.—Tubercle bacilli are rarely recovered from the surface of granulomata, but may be found in bits of tissue removed for microscopic examination. Giant-cells may be due to the irritation of the tumor masses by picking the nose and are not positive proof of tuberculosis as a cause. Granulomata in the anterior nasal fossæ are subjected to irritation oftener than in the sinuses and may contain giant-cells even when the new growths are not due to tuberculosis. Pearlman<sup>2</sup> and Pilot found numerous giant-cells clustered about strands of gauze in a nasal granuloma due to a severe nasal injury two years before. The patient was operated upon soon after the injury, but did not recover completely until after the granuloma was removed.

A large number of epithelioid cells is also suggestive of tuberculosis. Patients suffering from pulmonary tuberculosis may pick the nose with the finger or a handkerchief contaminated by sputum. Occasionally the nasal lesion may be primary.

*Lupus*.—The comparative infrequency of this disease in America makes this cause less likely. However, with lupus of the face and the alæ of the nose, the invasion of the nasal fossæ and sinuses, and particularly the vestibule, should be anticipated.

*Actinomycosis*.—The ray-fungus or actinomyces is a rather common cause of granulomata of the nose and sinuses, especially in farmers, ranchmen, or buyers and shippers of horses and cattle. Here again the finding of the ray-fungus in bits of tissue removed from the granulomata is the surest way to determine the etiology.

On one occasion I found an actinomycotic granuloma in an Italian coal miner, who took care of the mules and one or two milk cows inside of the stockade at a coal mine between Boulder and Denver. At first his occupation of coal mining did not make me think of actinomycosis. A patient's history, and especially his occupation, may be either very helpful or very misleading.

*Carcinoma*.—Microscopic examination of as large a piece of the tumor as can be removed will reveal the cause. Large granulomata either of the nose, of the accessory sinuses, or of both, in older patients should arouse our suspicion.

*Sarcoma*.—Biopsy is the surest method of determining the cause. Fibrosarcoma of the nose and accessory sinuses has very little tendency to metastasis, so there is very little risk to the patient from biopsy of this type of sarcoma. Fibrosarcoma is the type found most frequently in the nose and nasal accessory sinuses.

*Trauma*.—Granulomata are prone to grow at the point of fracture of the nasal septum or of the nasal bridge. The growth may take place subsequent to operations for the correction of fractures, deformities, or obstructions. With the annual increase in the number of automobile accidents, with facial injuries, granulomata of the nose from trauma may be seen more

frequently. Thomson<sup>3</sup> mentions that nasal granulomata may be traumatic in origin and often immature histologically.

Pearlman<sup>2</sup> and Pilot have reported a granuloma in the right side of the nose following a severe nasal injury and an operation in a man thirty-one years old. The granuloma was attached to the right inferior turbinate.

*Pyogenic and Mixed Infections.*—These are often secondary infections, but may be primary. The primary organism may not be found. Microscopic examination of tissue is best. Swabs from the nasal fossæ are of very little value on account of surface contamination from inhaled dust.

*Glanders.*—The finding of the *Bacillus mallei* readily determines the cause. The disease usually occurs in farmers, ranchmen, buyers and shippers of horses and cattle, butchers, packers, and tanners. The thorough manner in which well-trained veterinary surgeons eradicate this disease in stock prevents its occurrence in man. It is so rare in man that I almost never see a patient with glanders in any part of the body.

*Foreign Bodies.*—Rhinoliths stimulate the formation of granulomata. Almost any foreign substance remaining for any length of time may do the same. Recently my associate, Dr. C. L. LaRue, removed a large rhinolith weighing 74 grains from the left side of the nose. There were several areas of granulomata resulting from the irritation produced by the prolonged presence of the stone.

Pearlman<sup>2</sup> and Pilot emphasize the importance of foreign body irritation leading to the formation of granulation tissue which may appear as a polypoid growth in the nose.

*Leprosy.*—The extreme rarity of this disease in America makes this cause very unlikely. The finding of the so-called lepra bacillus requires a special technic. Evidence of the disease elsewhere in the body is very important. I have seen a very few patients with leprosy, so that I speak from a very limited experience with this disease.

*Rhinoscleroma.*—The infrequency of this disease in America makes this cause also unlikely. The disease is thought to be due to the *Bacillus rhinoscleromatis*. The disease is common in Austria, Russia, and Central America. In North America the disease is almost never encountered except in foreigners from the European countries where it is prevalent.

*Prophylaxis.*—The prevention of granulomata is naturally the prevention of the causative disease or diseases. For detailed information as to the best methods of prevention of the diseases enumerated under etiology the reader should consult any of the standard text-books of internal medicine and other sections of this book. Picking the nose with soiled fingers and handkerchiefs is a frequent source of nasal infections of all kinds, which may readily spread to the sinuses. Any of the foregoing causes or diseases may start about the teeth or mouth. If they begin in the upper alveolus on either side, it is easy for the disease, if not promptly checked, to extend to the nasal fossæ and sinuses. The maxillary sinus is usually the first one involved from the alveolar process. Proper oral hygiene is very important. It should not be necessary to mention infections from contaminated dental and surgical instruments with our present knowledge of this danger. Modern sterilization, with the use of sterile rubber gloves by the operator, should make the raising of this question unnecessary.

*Pathology.*—Granulomata are usually pale red or pink when viewed macroscopically in the living patient and have slightly roughened or

granular surfaces. They bleed rather easily when touched with a probe or cotton-wound applicator. Following their removal they are much paler and more friable.

*Microscopically* the epithelium is greatly thickened and desquamating with many goblet cells. Baum<sup>4</sup> says, "The granulation tissue masses are simply collections of rapidly growing blood-vessels; their walls formed by a single layer of endothelium and supported by a stroma of young connective tissue containing many cells." The epithelium is found denuded in parts where ulceration has taken place or new epithelial cells may be seen attempting to bridge the defect. The granulation tissue proper is the result of the regenerative activity in the subepithelial layer. Numerous young blood-vessels consisting of a single layer of endothelial cells frequently enclosing leukocytes form the center of the picture. They are surrounded by cells of various shapes and origin, chiefly round cells of the lymphocyte and leukocyte variety, and fibroblasts. As the submucous layer of the nose and accessory sinuses consists of a loose connective tissue, the edema is quite pronounced as a result of the exudative factors in the inflammatory process. The lobules of the mucous glands are surrounded by zones of round-cell infiltration. The granulation tissue tends to rise at first above the level of the healthy mucosa, but on the completion of the reparative process and the formation of fibrous tissue a contraction ensues, so that a sinking of the level or puckering may take place in the process of cicatrization. Such processes may be observed in chronic suppurative rhinitis, suppuration of the nasal sinuses, in ulcers due to suppuration, etc.

The *primary lesion of syphilis* is rare. *Congenital lues* is characterized by catarrh and round-cell infiltration of the mucosa, later by the formation of ulcers. The *principal lesion* of interest is the gumma. It is almost always periosteal or perichondrial and is usually on the septum. It may appear also on the floor and the roof of the nose or in the sinuses. Microscopically there is a central zone of caseation in which the original structure of the tissue may be faintly discernible. A few giant-cells, some epithelioid cells, and a surrounding zone of round cells are present. *Syphilis* is characterized microscopically by an obliterating endarteritis, with fewer giant-cells, and a cutting off of the blood-supply to the surface with necrosis and ulceration. As healing takes place there is a more extensive cicatrix, which contracts, producing the stellate scar, which may be seen macroscopically. A luetic granuloma of the septum may and often does extend to the hard and soft palate. The well-known perforation of the palate, as seen through the oral cavity, is almost pathognomonic of the tertiary stage. Generally speaking, syphilitic granulomata are most frequently found to involve the nasal bridge or the posterior portion of the vomer in the septum. Other types of granulomata are found most frequently involving the quadrangular cartilage or turbinates. Luetic granulomata of the sinuses may invade adjacent sinuses, the orbit, cranial or oral cavities.

*Tuberculous and lupoid granulomata* may, for practical purposes, be considered under the same heading. They have a broad base, are grayish red, and usually ulcerated on the surface. Tuberculous ulcers of the nose or sinuses are seen in patients with pulmonary tuberculosis. Microscopically these lesions are characterized by the presence of tubercles, consisting of aggregations of round cells, epithelioid cells, giant-cells of the Langhans' type, and foci of caseation necrosis. In granulomata caused by

*tubercle bacilli* there are many more giant-cells usually. Steward<sup>5</sup> mentions the importance of a larger number of epithelioid cells in tuberculous granulomata. Small tubercles may be found in the submucosa as further evidence of tuberculosis. The tubercles are homogeneous, non-vascular masses developing about the blood-vessels in the submucosa. As these enlarge the blood-supply to the surface is gradually shut off with subsequent ulceration, necrosis, and even abscess formation. Fibrous tissue forms about the tubercle and, if healing takes place, there is subsequent contraction of the fibrous tissue with stenosis of the nasal fossa, sinus ostium, or sinus cavity. The tuberculous invasion may extend well beyond the granuloma and involve the septum or turbinates. Perforation of the quadrangular cartilage or even the perpendicular plate of the ethmoid is not unusual. If the granuloma is in a sinus, the ostium may be enlarged by the destruction of bone or adjoining sinuses, and cavities may be invaded. The orbit and cranial cavity do not always escape. Beck<sup>6</sup> shows a microphotograph of a "pyogenic membrane lining the sphenoid in chronic suppurative sinusitis." Lincoln<sup>7</sup> reports 2 cases: One caused by syphilis and one caused by tuberculosis in which the granulomata were "exactly similar." Herzog<sup>8</sup> believes the "growths are alike whether tuberculous or syphilitic." While I believe this may be true macroscopically, it is not so microscopically.

The granulomata caused by the *ray-fungus* resemble a tubercle both macroscopically and microscopically. The proliferating tissue about the nodule resembles a sarcoma microscopically. These nodules are prone to mixed infections with the formation of an abscess and the discharge of pus. The *ray-fungus* is rather easily found in the pus.

Although *malignant tumors* do not strictly belong to the granulomata, they are considered in this connection on account of their being mistaken for granulations of inflammatory or infectious origin. A *cancer* of the *adenocarcinomatous* type may originate in the sinuses and grow outward. Usually carcinomata arise from the vestibule in the form of squamous-cell carcinomata. Masses and nests of epithelial cells are seen growing atypically and invading the subepithelial layers. In some there is a formation of horny matter. A basal-cell carcinoma arising at the ala may extend into the nasal passages or sinuses. If caused by *carcinomata* the invasion of the submucosa and deeper tissues by invaginations of epithelium is evident. In fact, epithelial pearls or nests may be found isolated below the surface epithelium. The submucosa is infiltrated with small round-cells, epithelioid, and giant-cells.

*Sarcomata* are quite rare. They may arise in any portion of the nasal passages or sinuses and be made up of round or spindle-cells.

The granulomata caused by *trauma* and *pyogenic infections* usually show more pus on the surface than do the others. This is particularly true if there is a pure culture of staphylococci found. The *Streptococcus*, *Staphylococcus*, *Pneumococcus*, *Micrococcus catarrhalis*, and rarely the influenza or colon bacillus may all be found in a series of patients. *Mixed infections* in the nose and sinuses are far more frequent in the chronic cases. A pure culture of one organism is the exception. The presence of a *foreign body* does not necessarily alter the pathology of the granuloma, but it gives additional information concerning the etiology and pathology.

The rare lesions of *glanders*, *leprosy*, and *rhinoscleroma* are briefly con-

sidered. In glanders numerous miliary nodules are seen in the reddened mucosa. These coalesce, break down, and form ulcers with bacony surface. Leprosy is manifested by nodular granulations tending to ulcerate in which lepra bacilli are found in large numbers. Rhinoscleroma is characterized by the appearance of flat or raised granulations of cartilaginous hardness and indolent growth. Microscopically there are bands of dense fibrous tissue infiltrated by plasma cells and so-called Mikulicz's cells.

I desire to express my appreciation to Doctor Phillip Hillockwitz, of Denver, for his valuable assistance in helping me to write the section upon the pathology. His many years of experience as a clinical pathologist give his opinion the weight of authority.

**Symptomatology.**—The *subjective nasal symptoms* are often those of more or less obstruction to breathing with a feeling of fulness, increased mucopurulent discharge, headache, smarting, burning, itching, sneezing, lacrimation, more frequent colds, and frequent epistaxis. If these symptoms are present on one side only, they will attract the attention early of an intelligent, observing patient. One patient told me that she was able to feel a soft swollen area on the left side of the septum just superior and posterior to the vestibule. Examination showed that she was correct.

*Nasal obstruction* is the most constant, frequent, and pronounced subjective symptom. *Pain* is a rare symptom early, but may be present with large granulomata of the nasal fossæ or sinuses. *Pain* is a frequent symptom if the granulomata are caused by malignancy with pressure from the large size of the tumor masses.

The *subjective sinus symptoms* are not always those of increased discharge, because they are present after the acute and subacute symptoms have subsided. They are part of the sinus disease, but are not responsible for all of the symptoms, such as headache, increased nasal discharge, smarting, burning, itching, sneezing, lacrimation, frequent head colds, slight fever, toxemia, pains in the joints, lassitude, inaptitude for work, anosmia, cacosmia, etc.

The *objective nasal symptoms* are those of a pale red or pink edematous, slightly rough, tumefaction involving the septum, roof, floor, turbinates, or meatus. Most of the granulomata occur on the cartilaginous septum. As a rule they are sessile and not pedunculated. Pegler<sup>9</sup> reports a lobulated, tuberculous granuloma, in a woman of fifty, occupying a depression in the left side of the quadrangular, septal cartilage. There was obstruction on the left side and a deflection of the septum to the right with a small perforation. The granulomata bleed rather easily, but the bleeding is not profuse and soon stops, which is in contrast to angiomas. If they are caused by a sarcoma or a carcinoma, bleeding is more profuse. Sometimes minute yellow tubercles can be faintly seen when the granulomata are tuberculous. If they are caused by syphilis, small gummata may be recognizable, although usually not. Granulomata are frequently ulcerated regardless of the cause.

In Steward's<sup>5</sup> series of 100 cases the tuberculous granulomata were distributed as follows: On the septum, 70 (mostly cartilaginous portion); inferior turbinate, 5; septum and inferior turbinates, 6; septum, floor, and inferior turbinates, 6; septum and floor, 5; septum and ala, 2; roof, ethmoid, middle turbinate, and nasal mucosa, each 1.

Mackenzie<sup>10</sup> and Cummings have reported a case of nasal tuberculous

granuloma, which was in the form of a reddish-brown polyp about 2 inches long before removal, and which completely blocked the right side. It had been growing for almost a year; was freely movable, vascular, and attached to the middle turbinate; it bled freely upon removal. They believe tuberculous and malignant granulomata bleed more freely than other types of nasal tumors except angiomata. Their patient did not show any other evidence of tuberculosis and they think the nasal lesion may occur as a primary tuberculous granuloma.

Lincoln<sup>11</sup> reported a granuloma of the right side of the cartilaginous septum, measuring 2.5 by 3 cm., caused by tuberculosis in a woman forty-five years of age. The granuloma was embedded in the nasal mucous membrane by a broad, sessile base and was not ulcerated. Following its removal it recurred on the other side of the septum one year later. Lincoln was unable to find other evidence of tuberculosis. However, he emphasizes the importance of a thorough physical and bacteriological examination and the importance of excluding syphilis.

The *objective sinus symptoms* are necessarily those observed at the time of operation when the sinus is opened for the thorough removal of granulations, polypi, pus, etc. It is rather unusual to find granulomata covering all of the walls of a given sinus except in neglected cases of long standing. However, many of the chronically infected sinuses contain granulomata as an important part of the pathology for which the operation has been performed. Very rarely have I found granulomata so large that they protrude from a sinus except in cases of long standing or in which malignancy was the cause. Granulomata in the sinuses are more frequently coated with pus than in the nose. They are more edematous, more papillated or lobulated, and more often tend to become pedunculated. Necrosis at the surface and later of the deeper structures with subsequent involvement of the underlying bone is more frequent from the pressure of confined pus, mucus, blood, polypi, etc. The granulomata also bleed more readily.

**Diagnosis.**—A soft, pale red or pink tumefaction, with a slightly roughened or mammillated surface, which is sessile and bleeds rather easily, is usually sufficient for a tentative diagnosis of granuloma. The surface may be, and often is, ulcerated, with pus coating the ulcerated areas. *Syphilitic* granulomata are more frequent in the nose and sinuses than tuberculous. This is partly due to the relative immunity of the nose and sinuses to tuberculous lesions of all kinds. If caused by *tuberculosis* the edges of the ulcers are often "mouse-nibbled." Granulomata caused by *lupus* are often only a part of the disease. Myers<sup>12</sup> has reported a case in a girl fourteen which he believes was due to lupus. Nasal stenosis and hypersecretion, produced by a granular neoplasm which filled both nasal fossæ, were the most noticeable symptoms. Pain was absent. The growths were attached to the septum, floor, and turbinates. Recurrences followed each removal. The involvement of the skin of the face and nose helps in making the diagnosis. In the nose or sinuses the disease is as sluggish as it is on the skin of the face, so that it spreads very slowly. Lockard<sup>13</sup> believes the nasal granuloma is more "indicative of a lupoid than of a true tuberculous infection." *Actinomyotic* granulomata are often more nodular, firmer, redder, and develop faster. The patient's occupation may help in making the diagnosis.

If caused by *malignancy*—either carcinoma or sarcoma—ulceration and

hemorrhage are usually more pronounced and the extension is rapid as a rule. Woods<sup>14</sup> believes the term "malignant granulomata" should be used for those due to malignancy. He reports 2 cases. One had extensive nasal involvement, in a man sixty-eight; there was a sunken bridge, invasion of the right lacimal sac with an external fistula, fetid crusts, and finally extension posteriorly with ulceration, infiltration, and death after four years. The second case was similar to the first, in a man sixty-seven, but a brilliant result was finally obtained by using radium. Syphilis was excluded in each case and antiluetic treatment was of no avail. Douglas<sup>15</sup> reports a case in which the granuloma was first noticed in the nasopharynx involving the right sphenoid.

If caused by *trauma* a history of former injury, operation, or both, will help in making the diagnosis. The evidence of trauma may also be apparent. When caused by *pyogenic* or *mixed infections* the granulomata are more frequently pus coated. This is especially true in the sinuses. In men who handle cattle *glanders* should always be thought of. *Foreign bodies* should always be watched for in small children and the insane because of the well-known tendency of such patients to poke all kinds of small foreign bodies into the nose. *Leprosy* is so rare in this country that for practical purposes it scarcely need be considered. The white appearance of the nodules in *rhinoscleroma*, so far as I know, is not found in any other type of granuloma.

*Microscopic examinations* of sections of granulomata and *animal inoculations* are more certain means of diagnosis, especially if tuberculosis, glanders, actinomycosis, mixed or pyogenic infections, etc., are suspected. The diagnosis of granulomata is not complete until the cause has been determined, as a few illustrative cases will show:

Mrs. A., age twenty-five years, married, mother of two healthy children, consulted me June 1, 1917, complaining of a feeling of fullness, obstruction, headache, bleeding, and mucopurulent discharge from the left side of the nose for several weeks past. Examination of the nose showed a granuloma about 3 cm. in size on the left side of the septum well forward over the quadrangular cartilage and above the vestibule. A general physical examination was made by a very competent internist and was negative, except that a short time previously the patient had had a miscarriage and had lost enough blood to produce a secondary anemia. Several blood tests were made and one spinal fluid Wassermann test and all were negative. Biopsy showed only a low-grade mixed infection.

The granuloma was thoroughly excised and the base cauterized with the galvanocautery, but in a few months it had returned. By this time the patient was visiting her parents in a large city and consulted an otolaryngologist whose reputation for thoroughness is well established and in whom I have the greatest confidence. He too suspected lues, but was unable to demonstrate the disease. He also excised the granuloma and cauterized the base.

By the time the granuloma had recurred I was serving in the Medical Corps of the Army and Mrs. A. was a patient of my associate, Dr. C. L. LaRue. While under his care she developed a very small perforation of the palate, and antiluetic treatment was started immediately. Soon after my discharge from the service I examined the patient. The perforation had entirely closed, leaving only a small dimple on the oral surface of the palate and a cicatrix 2 cm. in diameter on the septum. Vigorous antiluetic treatment has prevented subsequent symptoms to date.

Mrs. B., age fifty years, married, mother of several grown children, was seen in consultation April 27, 1920. She complained of very severe, excruciating pain in the right upper jaw for the past three months. She had not been able to sleep for eight weeks, except with the relief which morphine gave, and she had lost so much weight her condition was pitiful. A dentist had extracted the first and second molar above on the right. A large sequestrum of bone came away with the teeth.

Examination showed a large opening, 2 cm. in diameter, leading up through the right upper alveolar process into the right maxillary sinus. Granulomata were present in the naso-antral wall on the right and in the opening in the right alveolar process. Very little pus was found, but the odor was terrible. With a probe a large firm tumor mass was found

in the right maxillary sinus which bled easily. By transillumination this same sinus was very dark. Skillern<sup>16</sup> says, "The tumor itself remains usually unrecognized until tumefaction sets in." He makes this statement in connection with carcinoma of the sinuses.

In this case, without waiting for biopsy, Roentgen-ray examinations, and other laboratory tests, a diagnosis of advanced malignancy, *probably carcinoma*, was made. The condition was inoperable and the prognosis very serious. In spite of all reasoning Mrs. B. demanded an operation, but I refused to do one. In about ten days she discharged her regular physician and called another, who did advise an operation, but she died the day before the one set for the operation.

Master C., age sixteen years, schoolboy, seen December 2, 1919, in consultation with his uncle, who is a physician. The patient complained of complete obstruction on the right side of the nose with frequent and severe epistaxis on this side and mouth breathing. He had recently been under treatment with radium and Roentgen rays in a large eastern city by a very competent roentgenologist.

Examination revealed a rather extensive superficial desquamative dermatitis of each side of the face, and a large, dark red, cyanotic tumor mass in the right nasal fossa springing from the right inferior turbinate. The right side of the nose was completely blocked with the tumor, which was covered with granulatoma. These bled very profusely and very easily. The right maxillary sinus was extremely dark by transillumination. The clinical diagnosis was sarcoma. Biopsy showed fibrosarcoma. The roentgenologist reported extensive involvement of the right superior maxilla, sphenoid, and ethmoid. The prognosis was serious and the sarcoma probably inoperable because of its extension into the ethmoid and sphenoid. A follow-up letter to the patient's uncle says that subsequently the tumor mass, with the granulatoma, sloughed off with very little hemorrhage. The patient is living and enjoying apparently good health.

Fibrosarcoma of the nose has very little tendency to metastasis. Spontaneous healing of nasal sarcoma was not uncommon even before the days of Roentgen rays and radium.

Mrs. D., age seventy years, widow, complained of a very full feeling in the left side of the face with prominence of the left eyeball and a moderate amount of constant dull pain for several weeks past. Three months before she had had polypi removed from her nose by an otolaryngologist in her home city. Examination showed complete obstruction of the left nasal fossa, with granulatoma covering the turbinates and the latter forced over against the septum. The anterior wall of the left maxillary sinus and the floor of the left orbit were bulging with a firm, smooth, non-yielding swelling. The left eyeball was divergent and exophthalmic. By transillumination the left maxillary sinus was very dark. As the patient was on her way home from Estes Park she was advised to consult her specialist at once upon her return home, because malignancy seemed almost certain. Her otolaryngologist at her home reported that biopsy showed a sarcoma of the left maxillary. The sarcoma developed rapidly and terminated fatally according to a letter from her physician.

**Differential Diagnosis.**—Granulatoma must be differentiated from angiomata, polypi, osteomata, fibromata, myxomata, epitheliomata, sarcomata, and carcinomata. Angiomata are brighter red or bluer red and bleed very easily. Polypi are paler, smoother, and pedunculated. Hyperplasia of the ethmoid usually precedes and accompanies polypi. Osteomata are very firm, pale, and non-yielding. Fibromata and myxomata cannot always be determined clinically. The final word in diagnosis should always be with the aid of microscopy in case of suspected malignancy. Granulatoma which develop rapidly, especially if they recur following removal, should arouse a suspicion of malignancy. If there is bulging of a sinus wall, or of a cavity adjacent to a sinus, such as the orbit, malignancy should be considered at once. The laboratory tests and diagnoses are extremely valuable, but mature surgical judgment is most essential. Consultation with a colleague of wide experience is very helpful to the attending physician and to his patient.

**Prognosis.**—The prognosis necessarily depends upon the cause. If the granulatoma are caused by syphilis, antiluetic treatment will often produce

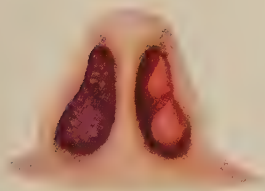
# PLATE I



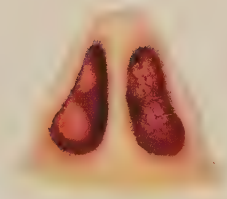
1. Mrs. A.—Nasal granu- loma, of approximately 3 cm. in size, on the left side of the septum, well forward over the quadrangular cartilage and above the vestibule. Surface irregular and quite pale.



2. Mrs. B.—Granulomata over the naso-antral wall on the right side. Rather pale in color with a very irregular, un- even surface.



3. Master C.—A dark red, cyanotic granulomatous mass, in the right nasal fossa spring- ing from the right inferior turbinate and completely blocking the right side of the nose.



4. Mrs. D.—A large gran- uloma covering the turbinates and resting against the septum, so that the left nasal fossa was almost completely obstructed by the tumor mass.



very gratifying results. Granulomata due to primary nasal tuberculosis, lupus, actinomycosis, trauma, pyogenic and mixed infections, and foreign bodies usually offer the best prognosis. Those produced by malignancy, except in the very earliest stages, offer a very serious prognosis. The outlook in leprosy is poor, but I have not had any experience with the treatment of rhinoscleroma.

**Treatment.**—*Palliative treatment* can easily be carried out with mild remedies such as 5 to 10 per cent. aqueous solutions of argyrol, protargol, or silver nitrate for nasal granulomata. Two per cent. mercurochrome in water is just now quite popular for rhinologic treatment. Mild ointments of camphor, menthol, oil of eucalyptus, yellow oxide, zinc sulphate, etc., are often more pleasing than beneficial if used alone. Davis<sup>17</sup> recommends "exposure to the sun or electric light as an efficient method" of treatment. Heliotherapy and the different forms of electric treatment should be useful. I have not had any experience with heliotherapy, the Finsen light, quartz lamp, fulguration, etc., in the treatment of granulomata.

*Curative Treatment.*—The best *curative remedies* for small granulomata are the galvanocautery, 95 per cent. phenol, 95 per cent. aqueous solution of trichloroacetic acid, or a bead of chromic acid fused on a probe if the tumors are in the nasal fossæ. Phenol should always be applied with great caution on a cotton-wound applicator, and should always be removed after a few minutes with alcohol on an applicator. The excess of phenol should not be allowed to run over the normal mucous membrane of the nose or over the skin of the alæ and upper lip. Before trichloroacetic acid is applied the skin of the alæ and upper lip must be well coated with zinc oxide ointment. Several minutes after the application has been made on a cotton-wound applicator, all excess may be neutralized with a saturated aqueous solution of sodium bicarbonate applied on a cotton-wound applicator, although this is not necessary. Chromic acid should also be neutralized with a saturated aqueous solution of sodium bicarbonate after a few minutes. Granulomata respond well to treatment with radium and Roentgen rays. If they are produced by malignancy, radium and Roentgen rays are indicated following the surgical removal. Barnes<sup>18</sup> has reported such a series successfully treated.

*Indications for Operation.*—If the granulomata are large or are in the sinuses, *excision* should be considered. If the granulomata are caused by syphilis, antiluetic treatment should always be tried first or in conjunction with mild local measures. When the tuberculous lesion in the nasal granuloma seems to be primary, cauterization is usually better than excision. Granulomata in the sinuses are not so accessible to treatment, and if producing annoying symptoms usually require operation. If they are caused by glands, sarcoma, or carcinoma early radical removal should be resorted to.

Granulomata caused by trauma, pyogenic infections, mixed infections, glands, and foreign bodies usually require excision followed by cauterization. Glands in particular requires early, thorough, radical removal followed by cauterization with the galvanocautery if anything is to be accomplished. Temporizing measures are worse than useless with this disease. The treatment for rhinoscleroma must be carried out in much the same manner as for glands. With leprosy the story is very different because the nasal or sinus lesion is almost invariably a small part of the

clinical picture, so that removal of the granulomata will have very little if any influence upon the disease in other parts of the body.

*Preparation of Patient.*—For a few days prior to operation a 4 per cent. aqueous solution of protargol should be dropped into the nose three times a day. The night before the operation the patient should have a hot tub-bath and some laxative. The morning of the operation breakfast should be omitted.

*Anesthesia.*—*Local anesthesia* can best be produced by applying 1 or 2 grains of powdered cocaine on a cotton-wound applicator to the granuloma if it is in the nasal fossa, or to the area of entrance to the sinus if it is in the sinus. A solution of  $\frac{1}{2}$  to 1 per cent. novocaine, procaine, or apothesine in a 1 : 200,000 adrenaline chloride solution in water may be injected if so desired. The committee on local anesthetics has shown, by its investigations, that combinations of two or more local anesthetics are more dangerous than one. If two or more are used together, therefore, much smaller quantities of each are safer.

*General anesthesia*, when preferred, can best be produced by gas and oxygen, ether, or all three. Chloroform is usually not suitable for nasal surgery. So able a rhinologist as Dr. Harris P. Mosher tells me he prefers general to local anesthesia for almost all of his nasal operations. His anesthesiologist uses a Rocky inhaler, to good advantage, with the patient sitting upright.

*Technic.*—If the granulomata are in the nasal fossæ they should be excised with a small scalpel or scissors, and with the minimum loss of mucous membrane, followed by cauterization with the galvanocautery. If they are pedunculated the snare may be the best. Curettes are better for the removal of granulomata from the sinuses than scalpels or scissors. If syphilitic, the removal should not be too extensive on account of the subsequent cicatricial contraction which is so apt to take place. Antiluetic treatment can usually be depended upon to cure syphilitic granulomata and often without any surgical intervention. If the granulomata are tuberculous, the galvanocautery, not preceded by excision, is probably the best. If they are caused by lupus Roentgen rays or radium have been very useful except with erythematous lupus. Actinomyces yields best to the internal administration of potassium iodide, the local application of heat and radium, without surgical removal. If malignancy is suspected an erythematous dose of Roentgen rays should be administered a few days before biopsy is resorted to, in order to seal the lymphatics, with the frank understanding that radical surgical removal is to follow immediately if the pathologist reports malignancy. Most modern hospitals are equipped with a freezing microtome, so that a pathological diagnosis can be obtained in a few minutes while the patient is on the table. Too much emphasis cannot be placed upon the importance of saving as much of the normal mucous membrane of the nasal fossæ and sinuses as possible, except in cases of malignancy, glanders, and rhinoscleroma. For the best method of operating upon the sinuses the reader should consult other sections of this book or Skillern's<sup>16</sup> book on "Accessory Sinuses of the Nose."

*After-care.*—The after-care should consist of proper cleansing of the wound with some mild alkaline antiseptic solution followed by the application of 2 per cent. mercurochrome, 10 per cent. protargol, argyrol, silver nitrate, or iodine cum petrox. If the wound is infected, tincture of iodine

or 50 per cent. silver nitrate may be required. The sinuses may require irrigation with Carrel-Dakin solution or with dichloramine-T solution. Clean wounds usually require very little after-care other than careful observation.

FRANK ROBERT SPENCER.

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#### NEURALGIA—NON-SUPPURATIVE NASAL HEADACHE

**Definition.**—Neuralgia or non-suppurative nasal headache includes those pains about the head, face, or neck which are the subjective symptoms of non-suppurative pathological changes in the walls of the nose or paranasal cells. These symptoms may be recurrent or constant, of varying severity and duration. The objective change in the affected tissues is usually characterized by hyperplasia. Included in this group are the following rather distinct entities: (a) Vacuum frontal headache; (b) the syndrome of nasal ganglion neurosis of Sluder; (c) the headaches of hyperplastic post-ethmoiditis and sphenoiditis; (d) nasociliary neuralgia.

**Etiology and Prophylaxis.**—This disease is not rare, being found wherever man is subject to repeated attacks of "cold in the head." The picture is

well known—coryza, acute rhinitis with nasal engorgement, intumescence, hypersecretion and subepithelial cellular infiltration, with more or less injury to the nasal epithelium. Various deficiencies in the caliber of the nasal meatus do much to increase the effect of the changes incident to acute rhinitis and to retard the return to normal. A single attack of head cold produces no noticeable permanent changes in the nasal structures, but the summation of the effect of numerous attacks is very evident and microscopically these changes are found to affect the deeper soft parts and even the underlying bone. Occasional intercurrent attacks of nasal suppuration or purulent sinusitis certainly hasten the hyperplastic process.

This hyperplasia, narrowing of the natural ostia of the paranasal cells or encroaching upon the adjacent nervous structures, seems to be the basic pathologic factor in the etiology of nasal headache. To what extent heredity influences the occurrence of this neuralgia is difficult to estimate. That it does play a part is strongly suggested by the observations of this complaint in two and sometimes three generations.

Means of prevention are general and are not specific. The condition of the nose is important from babyhood up. "Colds in the head" are not to be considered negligible, especially if recovery is slow. Much might be done to lessen the severity of the changes in the nasal mucosa accompanying the exanthemata of childhood by more care of the nose during these diseases. Adenoids or any obstruction to proper nasal ventilation should not be overlooked.

The further consideration of nasal neuralgias must be taken up under the subdivisions already noted: vacuum frontal headaches, the syndrome of nasal ganglion neurosis, and the headache of hyperplastic postethmoiditis and sphenoiditis.

**Vacuum Frontal Headache.**—*Definition.*—"A low-grade unending headache," established by closure of the frontal sinus, without nasal symptoms or signs, *i. e.*, obstruction or secretion, made worse by the use of the eyes.

*Etiology.*—The etiology is based upon the variations in the anatomic form of the nasofrontal duct. That form which is least liable to obstruction, which may be called the normal, is a funnel-shaped tube, the infundibulum, connecting the frontal sinus with the hiatus semilunaris. This normal duct may be encroached upon by an ethmoid cell bulging into its posterior, lateral, or anterior aspect, or by several cells in various combinations. The superior opening of the infundibulum, the large end of the funnel, may not open directly into the floor of the frontal, but may be covered by a roof of bone. When this condition exists the drainage of the frontal sinus may be into the infundibulum by a small ostium, or it may be through a separate narrow tube into the hiatus semilunaris. At a slightly lower level the lumen of the nasofrontal duct may be limited by a hypertrophied uncinate process; a large bulla ethmoidalis, or a middle turbinate closely applied to the lateral nasal wall, due to its own hypertrophy, to a deviated septum, to a large septal tubercle, or to a narrow nose. There may be various combinations of these limiting factors upon the patency of the duct.

These diverse factors limiting the size of the duct may not be sufficient in themselves to cause frontal headache, but the smaller the duct in any part of it, the more vulnerable is its patency to any pathological swelling.

*Pathology.*—A narrow nasofrontal duct may be temporarily closed by any acute swelling. This will cause a partial vacuum in the sinus. Varying

directly with the duration of the swelling and vacuum there will be more or less hyperemia of the sinus walls, pain, and tenderness on pressure. The swelling may become chronic hyperplasia, which may be limited to the soft parts, or may involve the underlying bone also. When the bone has undergone a hyperplastic change the condition is, of course, more permanent, and the closure of the duct chronic and less amenable to treatment.

*Symptomatology and Diagnosis.*—The complaint is of a dull frontal headache, less intense than in suppurative frontal sinusitis. It may be present on waking, becoming worse on use of the eyes for close work; or it may be inaugurated by ocular effort. It may be sufficiently severe to prohibit any sustained use of the eyes. There may be no nasal symptoms whatever. On palpation of the region of the sinus there will be found some general tenderness, but the greatest tenderness will be found on pressing over Ewing's point. This point is under the inner end of the eyebrow, medial and posterior to the fovea trochlearis. This tenderness will be greater than the usual tenderness of the frontal nerve as it crosses the superciliary ridge. This is Ewing's sign, and may be the only indication that the cause of the trouble is nasal. It is not found in any other headache of nasal or other origin except in suppurative frontal sinusitis.

*Prognosis.*—The prognosis must be guarded. Ordinary treatment may at times give permanent relief. Other cases may be successfully handled by operative measures. Still others may find only temporary relief from any treatment or operation. A reclosure of the nasofrontal duct is more likely to take place where the hyperplastic changes involve the periosteum or bone, also in noses in which any simple rhinitis is prone to cause polypoid changes.

*Treatment.*—(a) NON-OPERATIVE.—Non-operative treatment should be directed toward the reduction of the hyperplastic changes in the anterior and superior limits of the middle meatus: silver nitrate, mercurochrome, zinc salts, methylene-blue, and other astringents are useful. These efforts should be continued for several weeks before any surgical procedure is undertaken.

(b) OPERATIVE.—The most satisfactory operation for the relief of persistent closure of the nasofrontal ducts is the removal of the anterior two-thirds of the middle turbinate. This should include not only the pendulous or full portion of the middle turbinate, but its attached portion up to 2 mm. from the cribriform plate. This should include the internal covering of the hiatus semilunaris and even of the infundibulum. Care should be exercised to leave uninjured the external wall of the upper end of the hiatus semilunaris and infundibulum in order to minimize the possibility of closure by scar tissue. For this reason a curette is to be avoided. Sluder's knife is admirably adapted to cut away the parts desired, leaving the remaining parts intact and uninjured. It may be necessary to remove an encroaching uncinate process and bulla ethmoidalis. Relief is usually obtained by one operation, and if a return of symptoms requires further operative interference the procedure is much more simple than the original, unless the mucosa of the frontal duct has been destroyed and replaced by scar tissue. Under such conditions the external operation may be necessary.

**The Syndrome of Nasal (Sphenopalatine) Ganglion Neurosis.**—The syndrome of nasal (sphenopalatine) ganglion neurosis is a group of nervous phenomena caused by an inflammation of the nasal ganglion. It is usually

manifested by a "lower half" headache, involving the root of the nose, the eye, the temple, the zygoma, the ear, the mastoid, and the occiput.

*Anatomy.*—The nasal ganglion belongs to the involuntary nervous system. It is a small triangular, reddish-gray body, with its apex pointing posteriorly, lying in the upper part of the fossa pterygopalatina, just in front of the forward end of the canalis pterygoideus vidii, just lateral to the foramen sphenopalatinum, a few millimeters beneath and slightly medianward to the nervus maxillaris. It consists of an interlacement of nerve-fibers and stellate "sympathetic" nerve-cells. There are two roots, the sensory root and the motor and sympathetic root. The sensory root consists of the nervi sphenopalatini, usually two or three. These give off a few fibers to the nasal ganglion which are chiefly dendrites of the cells in the semilunar ganglion (gasseri), but includes a few sympathetic cell axones. The motor and sympathetic root is the nervus canalis pterygoidei (vidii) which enters the ganglion at its posterior apex. It is formed by the union in the canalis pterygoideus of the nervus petrosus superficialis major and the nervus petrosus profundus. The former arises in the ganglion geniculi of the nervus facialis and anastomoses with the nervus petrosus superficialis minor just after that nerve leaves the plexus tympanicus and runs forward through the foramen cæcum to the posterior opening of the canalis pterygoideus (vidii). The nervus petrosus profundus arises from the plexus caroticus internus, woven about the arteria carotia interna. In its passage through the canalis pterygoidei the nerve gives off a few small nasal branches composed of sensory and sympathetic fibers which supply the ostium pharyngeum tubæ auditivæ, the posterior part of the roof of the nose and of the nasal septum. It also receives a filament from the ganglion otici while in the canal.

The branches of the nasal ganglion, really a continuation of the nervi sphenopalatini with the addition of some nervous threads from the ganglion, are named for the region which they supply. As these regions are important, they alone will be mentioned here: viz., the mucosa of the posterior ethmoidal and sphenoidal cells; the mucosa of the turbinates, the fornix pharyngis, and the ostium pharyngeum tubæ auditivæ; the nasal septum; the mucosa of the hard and soft palate and gums, and the region of the tonsil. The anatomical relations of the nasal ganglion to the nose and the paranasal cells is important. They are given in full in Dr. Sluder's book,<sup>1</sup> though not mentioned in the usual text-books of anatomy.

"The nasal ganglion lies close to the lateral bony wall of the nose, in which the sphenopalatine foramen occurs as a small deficiency at its upper posterior part." By actual measurement the distance of the ganglion from the nasal mucosa varies from 1 to 9 mm.

The fossa pterygopalatina is bounded above by the walls of the sphenoidal sinus, including the sphenoidal process of the palate bone, closing it, and the separating wall is a thin one; in front by the wall of the maxillary sinus and this wall, too, is of thin bone. One or more of the postethmoidal cells may form part or all of the superior and anterior boundary. It is bounded behind by the anterior surface of the great wing of the sphenoid, and this wall also is, in some cases, only a thin plate separating the fossa from a downward prolongation of the sphenoid cell into the pterygoid process and into the great wing. The outer limit of the fossa is the only one not in intimate association with the cavity of the nose or the paranasal cells. Variations in the sphenoid cell may bring it into even more extended appo-

sition to the fossa. The wall of the nose may curve so sharply outward as to form a part of the anterior boundary of the fossa. The nasal ganglion lies close to the top of the pterygopalatine fossa. It is apparently prolonged backward into the vidian nerve, n. canalis pterygoidei. In front the ganglion is in relation with the arteria palatina descendens and the arteria sphenopalatina and with their associated veins. These vessels with some surrounding connective tissue usually form a separation of 3 or 4 mm. from the wall of the maxillary sinus, the anterior boundary of the fossa.

The sphenopalatine foramen is accurately placed at a point just posterior to and immediately above the posterior end of the middle turbinate. The ganglion usually lies close to the plane of this foramen, though it may be 7 or 8 mm. external to it. The variations in the position of the ganglion whether higher or lower are very slight. The pterygopalatine fossa is seen to resemble a paranasal cell, however, not closed externally by nasal tissue and filled by the before-mentioned structures instead of air. With such intimate anatomical association clinical manifestations from the extension of inflammation in the nasal fossa or its products would seem of almost necessary occurrence. Characteristic disturbances have followed post-ethmoidal and sphenoidal suppurative inflammations which cannot be explained otherwise than by assuming that the nasal ganglion has become involved by extension. In other cases extension has been from the nose proper.

*Symptomatology.*—The symptoms of nasal ganglion neurosis may be divided into the neuralgic and the sympathetic syndromes. The prodromal symptoms are any degree of rhinitis, a single slight coryza, recurrent attacks, an acute inflammation, swelling of posterior naris or of both, or a post-ethmoidalsphenoid suppuration. A short time later pain at the root of the nose, in and about the eye, the upper jaw and teeth, sometimes also the lower jaw and teeth, and extending backward to the temple and about the zygoma to the ear, making earache, emphasized at the mastoid, but always *severest at a point 5 cm. back of that*; thence reaching backward by way of occiput and neck, it may extend to the shoulder-blade and shoulder (less often to the axilla and breast) and in severe attacks to arm, forearm, hand, and even the finger-tips. This is the most frequent picture, but at times there may be also a sense of "stiff" or "aching" throat. There are numerous other variations in the distributions of the painful or disturbed sensations. The ganglion is accessible for cocainization which relieves the pain. The neuralgic symptoms may be the only ones. The sympathetic syndrome may not be manifested. It is more apt to occur if the prodromal symptoms are severe. There may be a "vasomotor-secretory phenomenon." There may be "severe and protracted sneezing accompanied by much nasal congestion and thin secretion." In addition to the intranasal disturbance redness and swelling of the external nose may occur. The eyes may be greatly reddened, and dilatation of the pupils and photophobia may be present. Sometimes these symptoms are accompanied by dyspnea with dry râles (asthma).

Cocainization of the nasal ganglion will control these sympathetic disturbances as well as the pain. Diagnosis of the nasal ganglion neurosis of Sluder may usually be made tentatively by the symptoms, though it is unusual for a case to include the whole list. It should be confirmed by

noting the effect of cocainizing the nasal ganglion. If the induction of anesthesia of the nasal ganglion controls the symptoms, the diagnosis is established. It is well, however, not to rely upon the result of a single test.

The failure of anesthesia of the nasal ganglion to control the symptoms may indicate that the lesion responsible for them is located at a point central to the ganglion, the root of the nervus maxillaris or the n. canalis pterygoidei (vidii). Such a condition under favorable circumstances may be proved by anesthetizing these nerves through the sphenoidal cells. Of course, this may not be possible because of the difficulty of entering the sphenoid cell or due to the inaccessibility of the nerves because they are separated from the cell by too great a thickness of bone. The observation by postnasal examination of congestion and hyperplastic thickening of the mucosa over the sphenopalatine foramen is usually an indication that the nasal ganglion is the seat of the lesion.

*Treatment.*—Relief of the nasal ganglion syndrome may be accomplished by local applications to the region of the sphenopalatine foramen. The remedies include 2 per cent. solution of silver nitrate, 1 per cent. solution of formalin, 2 to 20 per cent. solution of mercurochrome, 10 per cent. solution of phenol in glycerine,  $\frac{1}{2}$  per cent. phenol combined with 1/10 per cent. iodine as a wash. The more irritating medicaments should be preceded by a mild cocainization of the nasal path to the ganglion, using 4 to 5 per cent. solution of cocaine. This application may require some ingenuity; a straight applicator will sometimes do, but usually the end must be bent so that it will curve around the middle turbinate, or the inferior turbinate if that obstructs, or around some irregularity of the septum. Postnasal inspection may be needed to determine whether the desired position has been reached. If the local applications are unsuccessful, the ganglion should be injected. Sluder<sup>2</sup> recommends a 5 per cent. phenol solution in 95 per cent. alcohol; 0.5 c.c. of this solution is used. The injection requires a special knowledge of the anatomy and a special needle.

*Special Anatomy.*—The sphenomaxillary fossa is reached at about its center, 0.33 cm. back of the posterior tip of the middle turbinate. The posterior tip of the middle turbinate always marks the anterior limit of the sphenopalatine foramen. These relations being borne in mind, it must appear that a straight needle introduced into the nose from the nostril to pass under the posterior tip of the middle turbinate at its origin from the lateral wall, in a direction backward, upward, and slightly outward must pass out of the nasal fossa and enter the nasal ganglion or its immediate vicinity. The distance from the point of entrance of the needle so placed and the ganglion tissue is almost invariably 0.66 cm. The lateral wall of the nose plays an important part in this technic and its variations should be borne in mind, to wit:

1. In the middle meatus the lateral wall may curve outward and slightly forward from the attachment of the posterior tip of the middle turbinate so that the needle may be inserted into the sphenomaxillary fossa lateral to the ganglion, unless the point of attachment of the turbinate be carefully determined.

2. The lateral wall of the middle meatus is sometimes straight and smooth and of very hard bone. The point of the needle placed under the tip of the turbinate attached to such a wall may often be pushed backward without penetrating the wall. The tip of the turbinate will be punctured

and the point of the needle will pass submucously backward and upward to slip into the opening of the sphenopalatine foramen, and if carried farther backward will enter the vidian canal which is on a line internal to the ganglion. It may be impossible to reach the ganglion with a straight needle under this condition, and the curved needle may have to be used.

3. Very rarely the lateral wall is convex. This condition also requires the curved needle.

4. The lateral wall of the superior meatus is usually but not always on a plane internal to that of the middle meatus. It sometimes dips sharply outward, leaving the middle turbinate—a very prominently marked thin film of bone; posteriorly, however, it proceeds inward, finally to be on the same plane as the middle meatus, one meatus above and the other below the crista ethmoidalis as it ends on the anterior semicircle of the sphenopalatine foramen. This relation of the crista ethmoidalis, the posterior attachment of the middle turbinate to the foramen sphenopalatinum, is almost invariable.

**Instrumentarium:** Two needles are needed: (a) A straight steel needle 1 mm. in diameter with a strong cross bar handle; a flange 0.5 to 0.66 cm. from the point may be added to prevent too deep penetration; (b) a similar needle, but with a curved tip.

A syringe which may be attached by a slip-joint.

The nasopharyngoscope is helpful in properly placing the curved needle.

The injection fluid is 5 per cent. phenol in 95 per cent. alcohol.

The anesthesia for the injection may be nitrous oxide or local. For the latter, a drop of saturated (90 per cent.) cocaine is applied on a small cotton-tipped applicator first under the posterior end of the middle turbinate for five minutes and then the same applicator is moved to the sphenopalatine foramen for another five minutes. The injection under a successful local anesthesia is easily borne, but it is followed by pains which may last for an hour or for several days. Though at first it may be very severe, it gradually decreases. The result may be the permanent relief of the neuralgia. In more chronic cases several injections may be necessary. After each there should be relief, and the injections should be made after increasing intervals of two to three weeks, four to six weeks, and then at intervals of six weeks until the series are finished.

The neuralgia may recur as a result of fresh attacks of inflammation in the nasal fossa or neighboring nasal sinuses. The recurrences will require a repetition of the original treatment, and as subsequent postethmoid sphenoiditis may originate fresh attacks of nasal ganglion neurosis. The final relief may be obtained only after the operation on these sinuses.

**The Headache of Hyperplastic Postethmoid Sphenoiditis.**—*Definition.*

—This is pain in the distribution of the nervus trigeminus (n. ophthalmicus and n. maxillaris), and of the n. canalis pterygoidei (vidii) similar to that caused by empyema of the postethmoid or sphenoid, but caused by a characteristic hyperplastic change in postethmoid and sphenoid.

*Etiology.*—Repeated attacks of inflammation of those cells as a part of a general nasal infection or of those cells alone, the remainder of the nose being unaffected. This may have been a suppurative condition with slow recovery, the secretion passing from pus to mucopus to a thin watery almost indistinguishable serous flow. There may, however, have been no evidence of discharge, merely repeated attacks of swelling and hyperemia.

*Pathology.*—The changes to be observed in hyperplastic postethmoid sphenoiditis are a thickening of the mucosa, which has a cloudy appearance, may be dry or wet, and lacks the normal smooth texture. There are frequently small dilated blood-vessels distributed over the face of the sphenoid or laterally. This hyperplasia may be general, but if the postethmoid is involved the posterior end of the middle turbinate is apt to be especially hyperplastic. If the sphenoid is involved the plica septi is usually the seat of marked hyperplasia. It may be so hyperplastic as to stand out, a hemispherical mass touching the lateral nasal wall. The changes are rarely unilateral, though they may be more marked on one side. At operation the cells may be found to contain polypi or there may be only a thickening of the lining mucous membrane. The hyperplasia may, however, involve the submucosa and underlying periosteum and bone. The mucosa and submucosa may return to an apparently normal condition while the bony hyperplasia continues.

*Symptomatology.*—The symptomatology of hyperplastic postethmoid sphenoiditis is a frequently recurring headache. This may be of a dull heavy character situated in the occiput, when the anatomic relations of the sphenoid are such that the neighboring nerves are separated from it by thick walls. Usually the headache is much more extensive and varied, depending upon the involvement of the neighboring nerve-trunks in the disease process. It may be indistinguishable from "migraine." The optic nerve is frequently affected with the well-known result upon vision. The first or second divisions of the nervus trigeminus when involved may cause pain simulating that caused by disease of the frontal sinus and of the maxillary antrum. When the n. canalis pterygoidei (vidii) is involved in the hyperplasia of the sphenoid as well as the n. maxillaris the whole nerve-supply of the nasal ganglion is included in the disease and headache typical of neuralgia of the nasal ganglion may be produced. There is this difference, however, the pain is not controlled by cocainizing the nasal ganglion, as the involvement is central to the ganglion. If it be possible to apply cocaine to these nerves within the sphenoid cell the pain may be controlled in that way.

*Diagnosis.*—Diagnosis of hyperplastic postethmoid sphenoiditis should be attempted only by proper illumination. Inasmuch as all the light available in postnasal examination is that which is reflected from a mirror of about 1 cm. in diameter, the ordinary office light is not sufficiently intense. The Lilliput arc lamp made by Leitz is ideal. The sun itself, properly screened so as not to be too hot or too dazzling, may be used, but the sun is variable in position and not always available. The nasopharyngoscope is another valuable means of examining the postethmoid sphenoid region. The combined picture by means of the postnasal mirror and the nasopharyngoscope is often helpful in solving a doubtful problem of diagnosis.

In determining any pathological condition a knowledge of the normal is essential. Sluder's description<sup>3</sup> of this region should be borne in mind: "The color of the normal membrane is pink and it fits close to the bone. It gives the impression of thin pink silk velvet. The epithelium is smooth and transparent and the effect is given that the membrane is translucent. It appears moist, but not wet. If it is wet it will glisten. No vessels are recognizable clinically. The posterior tip of the middle turbinate is smooth and pink, but gives the impression that the membrane is less closely at-

tached to the underlying bone. The plica septi is only slightly developed and is of pearl pink appearance, that is to say, it is slightly less translucent than the surrounding membrane." This picture is unusual and is rarely found in the noses of patients who consult a rhinologist. Children and adults who have not been subject to colds and headache may afford an opportunity to study the normal.

Hyperplasia of this region will be observed as thickening of the membrane, which becomes more opaque or rough, and small vessels are apt to be visible. A more advanced condition will be shown by greater thickening, greater roughness, greater opacity, and more marked dilation of the vessels. Inasmuch as a serious intracellular hyperplasia, as shown by polypi at time of operation, may be accompanied by relatively slight visible changes, careful consideration should be given to apparently small departures from the normal.

The distribution of the hyperplasia is important. It may be part of a general nasal affection, the whole nasal mucosa being thickened with polypi in the middle and superior meati. It may be wholly limited to the postethmoid sphenoid region. The anterior face of the sphenoid may be the seat of marked mucosal thickening. The posterior tip of the superior and middle turbinates and the plicæ septi may be markedly involved. Usually postethmoidal disease is indicated by hyperplasia of the posterior tip of the middle turbinate, while hyperplasia of the plica septi indicates sphenoidal disease. There may be no secretion, the mucosa being dry, though usually there is increased moisture and careful search may detect a discharge. While the condition may be unilateral, it is usually present on both sides, and this should be borne in mind when examination reveals the disease only on one side while the symptoms are located on the other side.

**Anterior Ethmoidal (Nasociliary) Neuralgia.**—This specific type of pain, localized in the small area bounded by the supraciliary ridge above, the supra-orbital notch laterally, and the nasal bones below, was described by Sluder in 1922. As a rule the localization of the painful spot by the patient is quite definite and falls within this area described above. However, in severe cases the painful sensations may extend beyond this limit. Patients will occasionally say that it is impossible for them to wear their glasses because of the pressure of the bridge of the glasses upon this painful area at the root of the nose.

*Special Anatomy* (Quain).—The nervus nasociliaris enters the orbit between the heads of the external rectus muscle and between the two divisions of the third nerve. It then inclines inward over the optic nerve, passing beneath the superior rectus and superior oblique muscles, to the inner side of the orbit, and leaves that cavity by the anterior internal orbital canal. In this part of its course it furnishes a slender branch to the ciliary ganglion, one or two filaments (long ciliary) directly to the eyeball, and a considerable infratrochlear branch, which arises just before the nerve enters its canal on the inner side of the orbit.

Arrived in the cranial cavity, the nerve is directed forward in a groove at the outer edge of the cribriform plate of the ethmoid bone to a small canal between the forepart of the plate and the frontal bone, through which it descends to the nasal fossa. Here it gives off internal or septal and external branches to the mucous membrane of the forepart of the nasal fossa, and is then continued downward in the groove on the back of the nasal

bone, to terminate as the anterior or superficial branch in the integument of the lower part of the dorsum of the nose.

The branch to the ciliary ganglion, very slender and from  $\frac{1}{4}$  to  $\frac{1}{2}$  inch in length, arises generally between the heads of the external rectus. It lies on the outer side of the optic nerve, and enters the upper and back part of the ganglion, constituting its long root.

The long ciliary nerves are situated on the inner side of the optic nerve; they join one or more of the short branches from the ciliary ganglion, and, after perforating the sclerotic coat of the eye, are distributed in the same manner as those nerves.

The nervus infratrochlearis runs forward along the inner side of the orbit, below the superior oblique muscles, and parallel to the supratrochlear nerve, from which it receives, near the pulley of the oblique muscle, a filament of connection. The nerve is then continued below the pulley to the inner angle of the eye, and ends in filaments which supply the conjunctiva, the caruncle, and the lacrimal sac as well as the integument of the upper eyelid and root of the nose.

The internal or septal branch supplies the pituitary membrane over the forepart of the septum, extending downward nearly as far as the opening of the nostril.

The external branch, often represented by two or three filaments, is distributed to the mucous membrane of the forepart of the outer wall of the nasal fossa, including the anterior ends of middle and lower turbinate bones.

The anterior or superficial branch issues between the nasal bone and the upper lateral nose and runs downward under cover of the compressor naris muscle to the tip of the nose, supplying the skin of the lower part of the organ.

*Etiology.*—It is to be seen that the nasociliary nerve as it enters the nose in its uppermost anterior limit is quite near the surface of the mucosa. So it is easily understandable that an inflammation of the mucous membrane in this part may irritate or inflame the nerve and produce pain. In older cases, with general hyperplasia of the soft parts and the bone, it is also easily understandable that the ethmoid slit may be encroached upon so as to narrow its caliber and place the nerve in a more vulnerable position.

*Differential Diagnosis.*—This pain must be differentiated from the pain of both vacuum and suppurative frontal sinusitis, the pain of ethmoiditis, supra-orbital neuralgia, and the pain originating in the nasal (sphenopalatine) ganglion. The most important differential point is that in true nasociliary neuralgia the pain can be controlled by the application of cocaine to the mucosa of the nose covering the point of exit of the nervus nasociliaris. The pain of vacuum frontal sinusitis is almost always made more intense by use of the eyes, and Ewing's sign is present. Suppurative pathology in the frontal or anterior ethmoidal sinuses is usually shown by the radiograms and, as a rule, is made quite evident by the presence of pus in the middle meatus.

Pain due to the nasal ganglion can be relieved by the application of cocaine to the mucosa covering the sphenopalatine foramen and is not affected by cocainization of the nervus nasociliaris, while in nasociliary neuralgia the converse is true.

*Treatment.*—The pain can be relieved by an application of cocaine to the area of exit of the nerve into the nose. For this purpose a small cotton-tipped applicator carrying 20 per cent. cocaine usually suffices. It is passed upward on the inside of the nose, being held forward in contact with the anterior limit of the nasal fossa until it reaches the roof of the nose. By this procedure it arrives automatically in the apex of the angle formed by the cribriform plate above and the anterior limit of the nasal fossa. It is at this point that the nasociliary nerve enters the nose.

The relief from this treatment lasts from a few hours to several days. In the more intractible type of pain it may be necessary to inject a few drops of 5 per cent. phenol in 95 per cent. alcohol into the nerve. This is done following the technic first described by Otto Stein. For this purpose the straight needle of Sluder which he uses in the injection of the nasal ganglion is to be preferred. After cocainization of the mucosa covering the exit of the nerve the needle is passed upward under direct vision and good illumination. Just below the roof of the nose the point of the needle is passed under the mucosa and a few drops of the solution injected. This will usually be sufficient to infiltrate around the nerve and produce the desired result and is much safer than the injection of the nerve itself at its exit, for here there is danger of perforation of the thin cribriform plate of the ethmoid.

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ARTHUR M. ALDEN.

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## BRONCHIAL ASTHMA

*Symptoms.*—An attack of typical or true bronchial asthma consists of the following cycle of events: Some type of foreign protein, acting either centrally or peripherally as an irritant on the nerves that innervate the smooth muscular tissue lining the bronchi, causes a spasm or constriction of the bronchial musculature. The muscles of inspiration are equal to the task of drawing air through the constricted bronchi into the air-cells of the lungs, but the elasticity of the lungs, together with the muscles of expiration, are not sufficient to expel the inspired air in the normal time, so that expiration becomes prolonged and is finally interrupted by an inspiration before the normal amount of air has left the lungs. Consequently, as the attack progresses the lungs become overdistended with residual air, and sooner or later this overfilling of the lungs with air causes labored inspiration, although expiration remains more labored and more prolonged than inspiration. The attack is now at its maximum and it may continue for only a few minutes or for a few hours. During the attack the patient develops a dry cough which in a short time may become productive in raising a more or less characteristic type of sputum. This sputum is thin, clear, slightly tenacious, and in it are suspended small white tapioca-like masses of mucus called Laennec's pearls. Microscopically eosinophils, Charcot-Leyden crystals, Curschman's spirals, and small bronchial casts

may be found; however, none of these elements is of clinical importance. The attack of asthma begins to subside when sputum is raised. A normal or subnormal temperature and only a slight elevation of the pulse-rate accompany the attack. After the attack has subsided the patient may be more or less fatigued, but is otherwise normal and free from symptoms until another attack is suddenly precipitated hours, days, or months later, depending upon when some foreign irritant is again encountered.

**Physical Examination.**—On physical examination, during an attack of typical or true bronchial asthma, inspection verifies what has already been described, and in addition there may be some cyanosis. Percussion of the lungs during the height of the attack reveals a high-pitched resonance. On auscultation expiration is prolonged and feeble, and inspiration is wheezing and accompanied by dry râles; after expectoration has developed there may be moist râles. Fluoroscopy of the chest at the height of the attack reveals a motionless diaphragm which seems to be fixed in a depressed position, and the lungs expand very slightly on inspiration.

**Specific Causes and Treatment.**—*Animal Emanations.*—The inhalation of the proteins contained in the hair, dandruff, and skin dust of the horse, dog, cat, of fur-bearing animals such as pets, and fur-wearing apparel, and the protein in the feathers of chicken and goose are frequent causes of asthma. When these are the causes of asthma, it is best and usually satisfactory to dispense with the sources of the protein; that is, discard the feather pillows, get rid of the cat, dog, rabbit, or parrot, and discontinue wearing the fur neck-piece or coat, as the case may be. Very often, when horses are the cause, the patient is too sensitive to the protein to be able to live near a stable; furthermore, he may wish to be near or to drive horses. In such instances the patient may be treated in the same manner as that outlined for hay-fever by substituting the animal hair protein for the pollen.

*Food proteins* often cause asthma through inhalation of the flour of the cereal grains. Such instances are confined to bakers, housewives, cooks, or grain merchants, and keepers, all of whom handle the various types of flour and ground-up grain. The best and most satisfactory way of treating these cases is to have the patient avoid the flour dust, even though a change of occupation is necessary.

The most usual manner in which patients have asthma from foods is by the ingestion or eating of them. Cereal grain flour (chiefly wheat), eggs, and milk are the most common foods to cause asthma. In the case of wheat flour, the patient may eat shredded wheat biscuit, puffed wheat, and thoroughly toasted bread because the exposure of the flour to the extremely high temperature destroys the anaphylactic or poisonous element. Other foods containing white flour should be omitted from the diet and it is often necessary to remind the patient that macaroni, spaghetti, thickened gravies, dark breads, crackers, and the like contain white flour and consequently should be avoided. In testing with milk it is essential to use two proteins, namely, casein and lactalbumin, because when only the lactalbumin reacts positively the milk may be heated until the lactalbumin coagulates in the form of a scum on the surface of the milk and this coagulated lactalbumin or scum may be removed and the remaining milk may be taken. When casein reacts positively, milk should be avoided. In the case of eggs the white and the yolk may be tested separately since

occasionally only one part of the egg may be positive and the part failing to react may be eaten. The patient may eat baked potato when boiled potatoes cause trouble.

Although any food protein may cause trouble, it is the food that is frequently or constantly eaten that causes asthma for which the patient seeks relief. Therefore, in addition to what has already been mentioned, each patient should be tested with the foods that he is accustomed to eat frequently, namely, the cereals, the meats, the common vegetables, common fruits, and fish, and treatment should consist of avoiding the foods that cause a cutaneous reaction. Occasionally the patient may eat small amounts of the offending proteins, whereas larger amounts cause symptoms. Nursing infants should be tested with a similar list of food proteins since it is now known that sufficient food protein may be present in mother's milk to cause asthma in the nursing infant.

Although absolute omission of the offending protein is entirely satisfactory and not nearly as difficult as might be anticipated, there are methods of treating or desensitizing for foods. Capsules containing minute amounts of the offending protein may be given in gradually increasing doses, or if the food protein is in a liquid form, begin with drop doses and gradually increase the number of drops.

*Bacterial Proteins.*—As a rule bacterial proteins do not cause asthma and cutaneous tests with these are of little avail. In case bacterial proteins are desired for testing it is the protein of *Streptococcus hemolyans* and *viridans*, *Staphylococcus aureus* and *albus*, and *Pneumococcus* Type IV that should be employed. Treatment should consist of giving a vaccine of the organism that caused a reaction. It should not be understood that bacteria do not cause asthma because they very often do cause it; however, it is their infectious element rather than the protein element that causes symptoms. This bacterial cause of asthma will be discussed later under Vaccine Treatment.

*Organic Dust.*—The inhalation of dust from the cereal grains has been discussed under foods. Room dust and street dust may cause asthma because of the presence of animal emanations; this part of dust has already been sufficiently described in the paragraph entitled Animal Emanation Causative of Asthma. Face powders containing orris root and rice sometimes cause asthma, and these may be detected by doing cutaneous tests with orris root and rice protein; treatment consists of elimination. Sifters of green coffee beans, jewel polishers, and fur dyers have been known to become sensitive to the dust of their occupations. If avoidance of these dusts is impossible, treatment with subcutaneous inoculations, depending upon tests with various dilutions of these proteins, is curative. These and other organic dusts cause asthma because of the sensitization of the patient to them and this condition should not be confounded with the fact that inorganic dust which does not sensitize often causes asthma on account of mechanical irritation. Examples of inorganic dust irritation are chalk dust and ordinary dirt which is a part of house and street dust; these naturally do not cause sensitization.

*Pollens.*—To save space reference may be made to the chapter on Hay-fever and especially to the paragraph dealing with Perennial Hay-fever.

*Vaccine Treatment.*—Frequently the sensitive patient whose asthma is primarily caused by animal emanations, food, pollens, or what not, may

need autogenous vaccine in addition to the specific protein treatment. Vaccine treatment in these patients may be necessary in order to benefit an accompanying or a resultant bronchitis; furthermore, the condition of frequent colds, which is often associated with true bronchial asthma and which does precipitate attacks, is benefited by vaccine treatment.

*Climate.*—Change of climate benefits the bronchial asthmatic only when the offending protein is avoided. This holds true more especially for pollens in that a change may be made to a locality where the particular pollen does not exist. In a similar way a patient may move from close proximity to a stable or chicken farm to a more distant locality. After all, it is not the change of climate, but the omission of the offending protein.

*Drug Treatment.*—The most reliable and harmless drug that temporarily relieves the acute attacks of asthma is epinephrine. This is obtained as adrenaline chloride in a 1 : 1000 dilution and should be administered subcutaneously in  $\frac{1}{2}$  c.c. doses for adults, repeated as often as necessary. Benzyl benzoate by mouth sometimes seems to benefit children. When the causative agent is determined and removed, there is no need for drug therapy, unless there may be a complicating bronchitis, in which case expectorants or sedatives may be indicated. The Chinese drug ephedrine gives temporary relief in some cases, but it is not as efficacious as adrenaline chloride.

I. CHANDLER WALKER.

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### ASTHMATIC BRONCHITIS

An attack of asthmatic bronchitis is really an atypical attack of bronchial asthma and is usually associated with respiratory infections such as colds and bronchitis, chronic bronchitis, catarrhal conditions of the nose and throat, and occasionally with infections of the teeth, tonsils, and sinuses, and rarely with infections located in any part of the body. The primary cause is bacterial infection rather than protein sensitization.

**Symptomatology.**—Patients with this type of asthma usually develop their attacks in one of two usual ways. The most common manner is as follows: The patient has been subject to bronchitis for a period of months or even years. During this time the symptoms of bronchitis have progressed and have become more and more severe. At first possibly there may be only a slight unproductive cough which may have followed a neglected cold; later the cough is more annoying and may become productive of expectoration. There may or may not be slight fever and the patient, since physical signs are practically negative, may be suspected of having tuberculosis. After a time there is some difficulty in breathing, especially on exertion. Later still respiration becomes wheezy and dry rhonchi are heard on auscultation. If these symptoms progress no further the condition is called bronchitis. If, however, the patient develops attacks of dyspnea (it is inspiratory in type) and suffocation, with or without exertion, the condition is called asthmatic bronchitis. In reality the condition is a severe type of bronchitis.

The manner, next most common to the above, in which patients develop

this kind of asthma is as follows: As in the above case, the patient becomes subject to chronic bronchitis, and although he is more or less troubled with it during the time he is awake, he is usually free from attacks of marked dyspnea and suffocation, but during his sleep the attacks appear and usually awake him in the early morning hours; this type of asthma most commonly develops during or past middle life.

The sequence of events which takes place in these two types of attacks of asthma or asthmatic bronchitis is as follows: The bacterial infection in the bronchi causes the usual type of bronchitic sputum which may be thick, but it is not very tenacious or jelly-like, and it is raised with little difficulty ordinarily when the patient is not sleeping. At times, however, the sputum becomes very tenacious and jelly-like and it clings so tenaciously to the lumen of the bronchi that repeated coughs may fail to remove it. The stimulus to coughing, however, is so great that the patient coughs repeatedly, and the more he coughs the more dyspneic he becomes, until finally the tenacious secretion is raised, after which the patient rapidly becomes free from dyspnea. There is probably a slight constriction of the bronchial muscles, since the inhalation of fumes from antispasmodic remedies is followed by the raising of sputum and, consequently, relief from dyspnea. These drugs release the muscular constriction, thus leaving the secretion unattached. This muscular constriction, however, is not as marked as it is in bronchial asthma, neither is it caused by protein irritation of the nerves supplying these bronchial muscles. The cause of this slight muscular constriction probably results from local irritation due to the protracted spell of coughing or less likely it is due directly to the irritation of the tenacious sputum. The dyspnea in these attacks is chiefly inspiratory in type and is due partly to the unproductive cough, and partly to the narrowed lumen of the bronchi; this narrowed lumen is due partly to slight muscular constriction and partly to the coating of tenacious mucus superimposed upon the constricted mucous membrane of the bronchi. After the acute attack has subsided, the patient is not entirely free from symptoms; he still has more or less cough and expectoration until another attack occurs; this may be a few hours later or not until the early morning hours of the next night. The duration of the attack may be a few minutes, but more commonly it lasts an hour or two, and frequently the patient may continue in a more or less acute attack for several days. These attacks are frequently accompanied by a little fever and a slightly elevated pulse-rate.

**Physical examination** of patients afflicted with this type of asthma reveals, during the interval between attacks, signs of chronic bronchitis and emphysema. During the attacks the dyspnea is chiefly inspiratory in type, although both inspiration and expiration are prolonged, but the patient manifests the greater effort on inspiration, and, in addition, to the wheezing and dry rhonchi there may be heard coarse bubbling râles in the bronchi. The patient himself describes the dry râles as whistling and the wet râles as rattles. Fluoroscopy of the chest during an attack reveals the diaphragm fixed in about the normal position, midway in its greatest excursion, thus indicating no great amount of distention of the lungs. The lung vital capacity is low in these cases between the attacks at a time when the patient is most free from symptoms; this indicates a state of permanent emphysema. Pathology and Roentgen ray reveal a peribronchial thickening.

**Treatment.**—*Vaccine Treatment.*—Since patients with asthmatic bronchitis usually fail to give positive protein tests and since the cause nearly always is a bacterial infection, treatment consists mainly of vaccines. The bacterial infection is chiefly present in the bronchial tubes in which the patient's thick sputum contains the causative bacteria. Occasionally when the patient has little chest sputum, a catarrhal secretion of the nose or throat or an infected sinus harbors the causative bacteria, and rarely infected teeth are the source of the bacteria. The causative organisms are usually the Streptococcus group, although the Staphylococcus pyogenes aureus, diphtheroids, and pneumococci cause asthma, and rarely any respiratory tract organism may be the cause.

If stock vaccines must be used, those containing chiefly streptococci are the choice; however, in each case the use of a stock vaccine is merely a guess at the causative bacteria. Autogenous vaccines are by far the best since they offer the best chance of obtaining the causative bacteria. In making autogenous vaccines, thick masses of sputum, which are raised at the end of an attack or come from the smaller bronchi, are washed in sterile saline, shaken in bouillon, and plated on blood-agar. From the blood-agar plates the predominating organism may be selected. Equally good results follow from inoculating and growing the washed sputum in dextrose bouillon, and from this the vaccine is made. In a similar manner vaccines may be made from nasal secretions, or from the pus from an infected sinus or tooth.

Vaccine treatment should be given preferably at weekly intervals and never oftener than five-day intervals. The first dose of vaccine for adults should approximate two or three hundred million and each succeeding dose should be increased one hundred million until at least one thousand million is given at one time. If the patient is improving under such treatment it is best to continue increasing the dose up to two thousand million or until relief is obtained; if no benefit has resulted it may be best to make a new vaccine. Any dose that causes much local or any systemic reaction should be repeated once before the next increase of dosage is given.

The permanency of relief from vaccine treatment depends on the individual's resistance to the bacteria in question; therefore, the duration of relief from vaccine varies. Some patients continue free from asthma for many months or even years after vaccines are discontinued, others are free for only a month or two, and some patients require the constant use of vaccines to be free from asthma. Succeeding courses of vaccine treatment seem to relieve more promptly than the first course of vaccine treatment; when a relapse is not relieved promptly by a second course of vaccines a new vaccine should be made.

*Non-specific Protein Treatment.*—Since in most chronic infections intravenous foreign-protein treatment may be of benefit, the same may apply to asthmatic bronchitis. Good results have been reported from the intravenous injection of peptone; before trying this, however, the patient should be tested with peptone to be sure that he is not sensitive to it. In a similar manner typhoid vaccine has been used intravenously. Giving peptone in capsules by mouth an hour before each meal has yielded favorable results. The author has had little or no success with non-specific treatment.

*Climate.*—With asthmatic bronchitis a change of climate occasionally benefits or relieves attacks; even moving a short distance, as from low ground to high ground and vice versa, may relieve; but such instances are

not common. Florida is a suitable location for an occasional case, Arizona for still another, California for a third, and so on, but no one of these states or climates is suitable for all three; it is an expensive experiment and usually a bad investment.

*Drug Treatment.*—In the asthmatic bronchitis type of asthma potassium iodide in 0.6 gram (10 grain) doses three times a day is of considerable service. This drug thins the secretion in the bronchi, thus enabling the discharge of an otherwise thick, tenacious sputum, which, when not easily raised, causes choking up, severe coughing spells, and asthmatic attacks. In other words, potassium iodide favors free drainage from the bronchi with slight effort—a bronchial cathartic. The incorporation of small amounts of codeine with the potassium iodide is serviceable in allaying undue irritation. Benzyl benzoate by mouth sometimes seems to benefit children, but it is of little value in adults. Intravenous treatment with sodium iodide in 1-gram doses sometimes benefits; atropine subcutaneously in large doses and aspirin by mouth occasionally give temporary relief. The most reliable and yet the most harmless drug that temporarily relieves the acute attacks is epinephrine. This is obtained as adrenaline chloride 1 : 1000 and should be administered subcutaneously in  $\frac{1}{2}$ -cm. doses for adults, repeated as often as necessary. This drug should not be given intravenously or intramuscularly, and large doses should be avoided in children, with whom 0.2 to 0.3 c.c. suffices as a rule. Since the patient himself cannot use hypodermic medication, he tends to rely upon patent medicines and so-called asthma cures. The most serviceable among these seem to be the ones that contain stramonium leaves and saltpeter in the form of a powder, the fumes of which are burned and inhaled for the relief of the paroxysm. These fumes seem to be antispasmodic in action and following their inhalation thick sputum is raised and temporary relief results.

*Surgery.*—Operations for the removal of growths and obstructions to the upper respiratory tract are beneficial. Diseased sinuses and tonsils often harbor the causative bacteria in asthmatic bronchitis, and even though the causative agent of asthma may not be the same as that of sinusitis and tonsillitis, the eradication of the latter conditions is still more important since they lower the patient's resistance to other bacteria which may be the cause of the asthma. When complete cures of sinusitis are not possible by operation, autogenous vaccines made from the bacteria present in the pus are valuable after treatment. Polypoid and adenoid growths seem to play more or less of a part in the cause of asthma either directly or indirectly. Infected teeth may indirectly cause asthmatic bronchitis, but they do not play a sufficient part to justify wholesale extraction. Operations for the removal of infection and growth in any part of the body have been known to benefit asthma, but not frequently enough to justify such a prognosis.

*Bronchoscopy* may afford a means of local treatment; the presence of tenacious mucus in the bronchi is often the immediate mechanical cause of the patient's distress. In such cases bronchoscopic aspiration gives immediate relief and repeated bronchoscopic aspirations added to other measures will afford permanent relief. (See "Asthma" under the heading of Bronchoscopy for Disease—EDITOR.)

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### ALLERGY

The word "allergy" is generally used to designate all conditions in the human that are caused by protein sensitization or anaphylaxis. The mechanism of anaphylaxis is as follows: The first dose of protein, given to an animal, may be followed by a condition of markedly greater susceptibility to that protein. This phenomenon is called anaphylaxis; the animal is sensitized by the first dose of protein and is shocked by a properly spaced second dose of that protein. The anaphylactic shock is due to the meeting of a specific antigen (the second dose of protein) with its antibody (produced by the first dose), and the resulting reaction gives rise to a toxic product which causes the characteristic symptoms. Anaphylaxis, therefore, consists simply in the cellular reaction due to the fixation of antigen by cellular antibody. Anaphylaxis is, then, the reverse of vaccination or immunization, since the anaphylactic animal reacts to the second injection much more strongly than to the first. With the human the word "allergy" is often used for protein sensitization. The best examples of allergy are seasonal hay-fever and bronchial asthma, both of which conditions are described elsewhere in this book. Other conditions which may be due to allergy and which concern the nose, throat, and ear are, namely, angioneurotic edema and eczema.

#### ANGIONEUROTIC EDEMA

**Symptoms.**—In this condition there are local edematous swellings of transient duration and sudden onset. Sudden edema of the larynx may prove fatal.

**Cause and Specific Treatment.**—Angioneurotic edema is frequently caused by food proteins. Not only are the common or frequently eaten foods the cause, but also foods, such as strawberry and shell-fish, that are eaten less often or at definite seasons. Therefore, cutaneous tests should be done with a wide range of food proteins. Usually omission of the offending protein brings relief; however, occasionally an angioneurotic edema that is definitely caused by a food will persist for unknown reasons over a long period of time or will recur at intervals even though the causative protein has not been eaten. Similar instances are frequent following the injection of therapeutic serums. Angioneurotic edema is occasionally a complication in the treatment of hay-fever and asthma; the injection of too large doses of the pollen or animal emanation protein may cause the condition for a few hours' duration, but it does not become chronic or recurrent.

**Non-specific Treatment.**—Frequently angioneurotic edema, from the history of the patient or for some other reason, seems to be caused by foods when the protein tests are negative. In this case the condition may be digestive or alimentary in cause and may be treated by feeding capsules of peptone in 0.1-gram doses one-half to one hour before each meal. This treatment is based on the theory that peptone is an early decomposition product of all proteins, and by giving it prior to the ingestion of food the patient is made temporarily anti-anaphylactic or non-sensitive to any food protein so that the eating of the causative food, whatever it may be, will not produce angioneurotic edema. The author has obtained better results by giving *Bacillus acidophilus* with milk-sugar in milk prior to each

meal than by giving peptone. Small doses of milk of magnesia prior to each meal seems to be of considerable benefit. *B. acidophilus* and milk of magnesia seem to speed up the gastro-intestinal tract, thereby diminishing the chance of absorption of undigested proteins which probably cause this type of angioneurotic edema. Free evacuation of the intestinal tract is desirable.

**Emergency Treatment.**—Epinephrine (adrenaline chloride) 1 : 1000 given subcutaneously usually relieves.

#### ECZEMA

In infants chronic eczema, exclusive of the scalp alone, is very frequently due to some food protein even while the infant is breast fed. Rarely do breast-fed infants show sensitization to human milk, but when this is the case, goats' milk may be substituted. Usually the nursing infant is sensitive to some protein that it has never eaten, but that the mother is eating in large quantities, such as cows' milk, egg, cocoa, etc. The human milk in such instances contains the food protein which the child has ingested and absorbed. Even though the mother is not sensitive to these proteins, the nursing infant may be; therefore the nursing infant should be tested with the proteins which the mother is eating. Treatment naturally consists in the omission of the protein that affects the child from the mother's diet.

In children eczema is frequently caused by some food protein that the child is eating. When eczema begins at the period of weaning, as is very often the case, the cause is usually the proteins of milk, egg, white flour, oat or potato, since these are the first to be eaten in much quantity and are, so to speak, foreign proteins to the child. When eczema develops in older children the above-mentioned food proteins are less often the cause, and other foods, such as tomatoes, strawberries, and in fact any food that the child eats, may be the cause. Therefore, the older the child when eczema begins, the less frequently are foods the cause and the larger must be the list of food proteins for determining the causative one by the cutaneous test. Treatment consists of omission of the offending protein as determined by the cutaneous test.

In adults food proteins are less often the cause of eczema; however, if the eczema is not amenable to the usual treatment and no cause is demonstrable by other means, food protein tests are worth trying.

**Miscellaneous Causes.**—Since this section concerns only anaphylactic foods, other causes of eczema should not be mentioned; however, since fats and carbohydrates are foods and at times cause eczema in children and adults, even though they are not anaphylactic because they are proteins, it may not be amiss to mention fats and carbohydrates as causes of eczema. Furthermore, it may be mentioned that the author has occasionally found eczema to be due to bacteria (*Staphylococcus pyogenes aureus*), to pollens (grass and ragweed), and to animal emanations. Locally crude coal-tar is efficient medication.

I. CHANDLER WALKER.

### HAY-FEVER

The symptoms of hay-fever are watering and itching of the eyes and nose, sneezing, and itching of the throat. When these symptoms are present in an individual only at a definite season each year, the condition is defined as seasonal hay-fever, and the cause is nearly always some pollen which has been inhaled. The alkaline secretions of the mucous membranes of the eyes, nose, and throat dissolve the protein out of the pollen grains and this protein acts as an irritant to the mucous membranes, causing the nasal symptoms. Only those who are susceptible to the pollen protein are affected, or, in other words, only those who have become sensitized for some unknown reason are affected. When this condition is present in an individual more or less continually throughout the entire year, the term "perennial hay-fever" is used and the causes are not only the proteins of pollens but also of foods, animal hairs, and, in fact, any protein that is inhaled or ingested. There is also a condition so closely resembling hay-fever that it is called pseudohay-fever, the causes of which are mechanical, chemical, odorific, or thermal; these causes are direct irritants to the mucous membranes and do not concern proteins.

**Seasonal Hay-fever.**—Nature divides hay-fever into various seasons, in that different plants pollinate at different times. The season of pollination varies with the locality; the warmer the climate, the longer and earlier is each season. North of Mason and Dixon's line there are three seasons, namely, spring, early summer, and fall. Further south the seasons overlap and in the extreme south there are really only two seasons, namely, early and late. Therefore, in order to determine the cause of pollen hay-fever, it is very essential to learn from the patient at what time of the year he has his symptoms. Having obtained this information it is just as essential to know what plants are in pollination at that definite time. Since only the wind-borne pollens cause symptoms and, furthermore, since those pollens which are produced in great abundance are the chief causes, it is essential to have this knowledge.

**Causative Pollens.**—As a rule the trees pollinate first, and those that have the greatest abundance of pollen are the maples, elms, ash, box-elder, birch, black walnut, poplars, cottonwoods, willows, and pines; these trees usually begin pollination in February and the last is finished by late May. The grasses are next to be considered in importance. They may pollinate along with the trees in the south, but in the north they have a definite season extending from early May to August. The same grasses do not grow universally, but wherever the following grasses are present they should be considered as chief causes of hay-fever: timothy grass, June grass, blue grass, alfalfa, Bermuda grass, Johnson grass, poverty grass, broncho grass, quaking grass. Although this type of hay-fever is usually called rose cold, roses rarely are a cause. A third season of pollination concerns the *Compositæ* which pollinate as a rule from the first of August to the first frost. The chief causative pollen is the ragweed; dwarf or small ragweed in the east and giant or large ragweed in the south and west. Other plants that must be considered wherever they grow in abundance are sunflower, aster, sagebrush, rabbit brush, pigweed, marsh elder, Russian thistle, careless weed, lamb's quarters, salt bush, and sagewort. In some southern localities there is a second crop and pollination of Ber-

muda grass and Johnson grass in the fall. Therefore these must be considered among the causes of fall hay-fever as well as the earlier type of hay-fever. For a detailed discussion of the chief pollens in various localities reference may be made to Hygeia.<sup>1</sup>

**Pollen Tests.**—After it has been determined at what time of the year the patient is affected and also what pollens are present at that time, tests with these pollens may be made to determine more definitely what particular pollen chiefly affects the patient. Small scratches are made with a sharp scalpel on the flexor surface of the forearm, each scratch is about  $\frac{1}{8}$  inch long and not deep enough to draw blood, but still penetrating the skin. Upon each scratch a small amount of pollen is placed and a drop of tenth normal sodium hydrate is added to dissolve the pollen protein, or if the pollen protein is already in solution a drop of this is put on the scratch. In a few minutes a definite wheal will appear, thus demonstrating that the patient is sensitive to that pollen protein. Since the same patient is apt to be sensitive to several closely related pollens, it is advisable to test with various dilutions of the pollen proteins and to select as the chief cause that pollen which reacts in the greatest dilution or in the smallest amounts.

**Pollen Treatment.**—There are so many different satisfactory ways of treating the pollen to get a protein extract for treatment and so many commercial preparations may be obtained, all of which seem to give good results, that it is folly to state that any particular method or preparation is best. On the whole the best results seem to be obtained by starting treatment early enough to insure the completion of treatment just previous to the usual onset of symptoms. Since most treatments consist of ten to fifteen inoculations and it is best to give only one treatment each week, it is advisable to start the series of inoculations ten to fifteen weeks previous to the onset of symptoms. Less favorable results are obtained by starting treatment later, thereby necessitating treatment during the season of symptoms. No matter what method of treatment or what preparation is used it is very essential to test each patient with the various treatment solutions in order to learn with what strength of solution treatment should be begun. This is obvious since different preparations may vary in strength and patients do vary in the degree of sensitivity. In other words, without these tests the preliminary dose may be too strong for one patient, thereby causing unpleasant symptoms, and another patient may be able to begin treatment with a stronger dilution than usual. The first treatment should be given from the dilution which fails to give a reaction by skin test and each succeeding treatment should be an increased amount, although succeeding doses should never be doubled, that is, never double the previous dose. Any treatment that causes any general or much local reaction should be repeated once before an increase is given. A period of five days seems to be the shortest interval between treatments that is advisable, and by using the five- or seven-day interval the writer, using his own preparations, seems to have permanently cured a number of patients; at any rate, following such treatment many patients have given negative pollen tests in succeeding years and these patients have been free from hay-fever without treatment for a period of three or more years. Using a shorter interval between treatments has not given such permanent relief.

**Causes Other Than Pollens.**—Occasionally, when pollen treatment

fails, treatment during the season with an autogenous nasal vaccine will benefit. It is quite possible that pollen may so irritate the mucous membranes that the ever-present bacteria, either alone or together with pollen, may be a cause of hay-fever symptoms. Rarely the inhalation of some animal hair protein or the ingestion of some seasonal food protein such as fruit or green corn or a seasonal vegetable may be the cause, and treatment consists in avoiding the causative protein.

**Miscellaneous Treatment.**—For those hay-fever patients who cannot be treated as already outlined or in whom the pollen treatment fails, a change of locality to a place where the causative pollen does not grow is advisable. High altitudes are usually free from causative pollens and, naturally, ocean trips will avoid pollens. When avoidance of the pollens is out of the question as well as desirable treatment, dark glasses, a boric acid eye-wash, adrenaline nasal sprays, and saline nasal douches alleviate somewhat the acute symptoms. Operations on the nose and throat are of no benefit in the protein sensitive hay-fever cases unless the excessive irritation has predisposed to or permitted infection to complicate the condition.

**Perennial Hay-fever.**—As already stated, this condition is present more or less continuously throughout the year and the cause may be the protein in food, pollen, animal emanation, a dust, or even bacterial infection. Cutaneous tests should be done with these proteins and the offending cause may be omitted. The inhalation of plant pollens at a definite season may predispose to a perennial hay-fever and a typical seasonal hay-fever caused by pollens may become a perennial hay-fever due to other superimposed causes. Therefore pollen treatment is essential in such cases. Patients that fail to give a positive cutaneous test are often benefited or relieved by treatment with an autogenous vaccine made from the nasal secretion. In this type of case the nose and throat specialist often finds some source of infection or some abnormal growth which may be the cause of symptoms. Since it is difficult clinically to distinguish non-sensitive cases of perennial hay-fever from vasomotor rhinitis, the nose and throat specialist should be consulted after the cutaneous tests have failed to demonstrate the cause.

**Pseudohay-fever.**—The causative agents of pseudohay-fever are mechanical, chemical, odorific, and thermal. Among the mechanical causes any kind of dust is the most frequent one, more especially sweeping dust and hay dust; fine powder, such as talcum and the like, is also a frequent cause. Among the chemical irritants, soap powder, lye, and ammoniacal fumes, are very frequent causes. Among the odorific irritants, heavily scented perfumes, face powders, musty air, and stable odors are frequent causes. Thermal irritants concern sudden changes of temperature, as going from warm air to extreme cold, from moist air to very dry air, and exposure to drafts; a very frequent history is that of a paroxysm of sneezing with or without running of the nose on retiring and arising. The mechanism of the latter seems to be a reflex due to the sudden exposure of the warm and protected skin of the body to cold air, as in getting out of bed and undressing, during which acts the warm body surface is suddenly and momentarily exposed to cool air; in other words, there is a mild chilling of the body surface. The same mechanism holds for many who take cold easily. Occasionally pseudohay-fever patients are sensitive to some type of protein which may have rendered their nasal mucous membranes

sensitive to those irritants. Appropriate protein treatment for those who are sensitive usually relieves these symptoms, no matter what the irritant may be, and occasionally autogenous nasal vaccines will benefit or relieve the non-sensitive individual. The usual causative irritant may be avoided.

I. CHANDLER WALKER.

#### REFERENCE

1. Walker, I. Chandler: Hay-fever; Early Spring, Summer, and Fall Types, Hygeia, 1923, i, 69, 169, 291.

### EXTERNAL DEFORMITIES OF THE NOSE

External deformities of the nose may be due to loss of substance or disarrangement of normal elements. It may consist of loss of skin, cartilage or bone, or may be the result of pressure internally or the contraction of scar tissue within.

It may, further, be due to a deflected septum which, through pressure or interference with respiration, interferes with development.

Depression of bone or cartilage may give a unilateral deformity, although in the case of bone the opposite side is usually forced outward.

**Classification.**—1, Congenital and developmental; 2, traumatic; 3, infections; 4, neoplasms.



Fig. 115.—Bifid nose, also showing deformity of eyelid.

The most frequent deformity of the congenital variety is the flattened nose, either wholly or on one side, produced by cleft lip. The degree of deformity depends largely on the extent of the cleft in the lip. By this we do not necessarily mean that we may not have a great deformity with only a slight notch.

Depression, due to removal of too large a portion of cartilaginous septum anteriorly, may take place when septal resection is not carefully done.

Infection which interferes with the external appearance must destroy the cutaneous covering or the bony and cartilaginous framework and may be due to local infection or to local manifestation of constitutional disease. That injury may play a part must be admitted, but that infection in these

cases overshadows the injury, as injury overshadows infection in Class 2, must be evident.

Abscess of septum, causing depression at the junction of nasal bones and lateral nasal cartilage, is rarely seen.

Broadening of nose, due to periostitis and later osteomyelitis of the nasal bones and nasal process of maxilla, is seen either following sinus infection or due to injury followed by infection.



Fig. 116.—Cleft lip, showing flattening of alæ.



Fig. 117.—Same as Fig. 116, after operation.

Tumors may cause deformity because of pressure from within or on account of external growth. We have also a tumor produced partially by some foreign body having been introduced and this producing the true tumor growth. This is seen in the so-called paraffinoma.

Deformity from polypoid growth in the nose, producing widening in the ethmoidal region, is frequently seen. Malignant tumors of this region are



Fig. 118.—Nose destroyed by syphilis.



Fig. 119.—Same as Fig. 118, nose partially restored by rib-graft placed in forehead and flap turned downward.

somewhat rare, although epithelioma on the external surface is not uncommon.

Traumatic, as the second class, must be considered from the nature of the injury, whether recent or of long standing, whether complicated by other injury or by manifestations of local or general infection or disease. Furthermore, we must take into account whether the injury has caused loss of tissue or whether the tissue may have been so injured that it will be

eventually lost and whether this be soft tissue or the bony or cartilaginous framework, or all three. The deformity after healing will depend so much upon the careful consideration and judgment of these facts, and the ultimate result from the standpoint of appearance and function of the parts as it affects the patient's welfare, mental attitude and economic life, that they should not be looked upon lightly.

**Etiology.**—Blows, falls, industrial, auto, and railroad accidents with infrequently a gunshot wound, in civil life, may be said to cover, in the main; but we are not so far removed from the late war to forget the deformities which followed many of the facial wounds.

Complete removal of nose or, as more frequently happens, the tip may be met with even today. This was seen in ancient times when this organ was removed as a punishment for certain crimes. Today this calamity is most frequently caused by an accident. One case, however, came under our care in which the nose had been bitten off in a fight.



Fig. 120.—Depression due to abscess of septum.

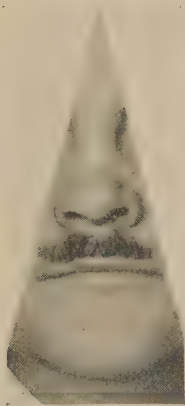


Fig. 121.—Depression due to abscess of septum.

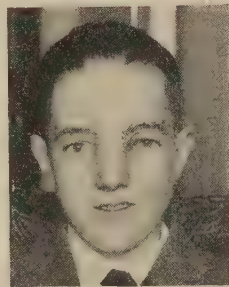


Fig. 122.—Nose broad at base, due to growth of nasal polypi in ethmoid and frontal regions.

With incomplete cleft of lip, incomplete fibers of orbicularis oris in this region may allow the nostril to flatten. Deep clefts may show only slight nasal deformity in some cases, although this is contrary to the rule.

Other congenital nasal deformities found are stenosis and notching of the tip, but they are rare.

Congenital absence has been reported and an operation devised by Maisonneuve for its correction.

Bifid nose is a congenital condition in which a groove is found between the two halves of the nose where they have not united during fetal life. It is generally associated with a meningocele or encephalocele, deformities of eyes, lids, etc., but may be found as a single deformity.

The deformed nose, due to family characteristics or to deformities of the maxilla, is rather developmental than congenital, but nevertheless belongs in Class 2, unless as it may occur when development has been interfered with by accident or infection. It may be difficult at times to decide under which class a given deformity should be considered.

The long nose, the short upturned nose, the hump-nose, the flat-wide nose with large nostrils, and the pointed narrow nose with collapsed nos-

trils may be called either congenital or developmental. With these may be included the saddle-nose which is not traceable to syphilis.

Treatment of these deformities is necessarily surgical and depends upon the individual case.



Fig. 123.—Paraffinoma.

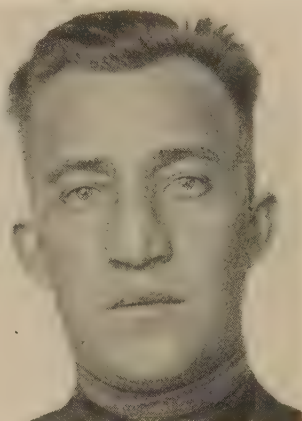


Fig. 124.—Paraffinoma. Front view of Fig. 123.

The interest in plastic surgery, especially of the face, has increased in late years. This is due not only to demands by the laity, but to improvements in technic of operations of this kind.

The difficulties of performing operations of this character under general anesthesia and the fear which the patient manifests toward ether has been a deterrent both to the patient and operator. Improvements in methods of administering local anesthetics has also added to the momentum in this direction. The older methods of infiltration interfered with healing as the stretching of the tissues interfered with nutrition. More care is exercised now and nerve blocking does not interfere with nutrient vessels. There has been marked advance in after-treatment and care, which makes for better and more certain results in these conditions.



Fig. 125.—Same as Figs. 123, 124 after operation.

Congenital deformities, if very evident, should be corrected early; but if of minor importance may be left until later in life. The difficulties of performing operations on very small children, on account of the

size of the parts, makes for greater liability of infection and the possibilities of changes in the course of development must be considered. The shape of the nostril in the newborn is almost an equilateral triangle, while in the adult it becomes elliptical, and this must be taken into account. Clefts of the lip which affect the nose must, of necessity, be corrected early, while deformities due to septal deviations should be left if possible until puberty.

Correction of the flattened nostril is not an easy task and requires loosening of the soft tissue of the ala from the superior maxilla and anchoring to the columna with the formation of the floor of the nostril which is either completely or partially absent. The scar formed has a tendency to shorten the lip, and if the cleft in the maxilla is not closed recurrence of the flattening results.

Defects due to loss of tissue must be corrected by replacing with either the portion lost or some substance that will be tolerated by the remaining tissues. Replacements of the completely separated nose with subsequent healing has been reported and should be tried, for, if successful, it will be much better than a result obtained by any other method. Transplantation, however, at the present time is impracticable because of the impossibility of finding a donor.

Considerable controversy has taken place as to whether cartilage, bone, or some foreign substance should be used to replace lost tissue. At present



Fig. 126.—Result of automobile accident. Crushing of nasal bones, nasal processes of superior maxilla, and superior nasal spine.

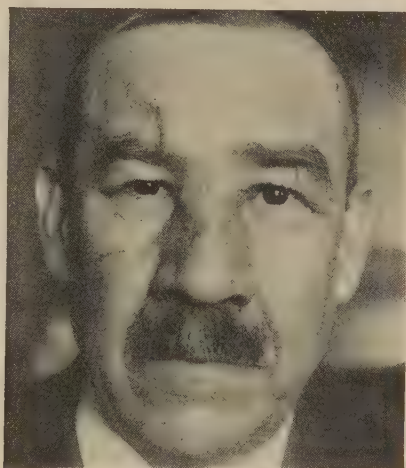


Fig. 127.—Same as Fig. 126 one month later.

it cannot be definitely stated that any one substance is the correct and only one.

Paraffin, which was introduced by Gersuny, met with favor for a time, but very bad results are attributed to it and, consequently, it has been largely replaced by other substances.

Carter, Cohen, and many others use bone and cartilage, New and Lewis use celluloid, while Risdon and others use ivory.

It must be stated that cartilage is best to replace cartilage, but that many other substances besides cartilage may be used to replace bone. Our preference is for autogenous grafts, whether the bone be taken from rib or tibia (Iglauer prefers the former), and whether cartilage is costal or from the ear. Free grafts may be taken or the tissue may be attached to another part of the body first, as the hand, and after uniting with this, is transferred to desired location. While this is an old method it has lately been ingeniously applied by Dr. Arce of Buenos Aires in transferring a portion of the car-

tilage of the external ear to replace a destroyed ala. Incision is made in the ear and a linear incision made on the inner surface of the thumb. After union has taken place, the portion of the external ear desired is



Fig. 128.—Depressed nose following submucous operation.



Fig. 129.—Same as Fig. 128 after transference of cartilage from ear.

severed from the remaining ear and transferred to the nose, which, when union takes place, is severed from the thumb.

Incision in the nose for removal of the hump was formerly made at the junction of nasal and frontal bones, for the reason that the scar would not



Fig. 130.—Old fracture with depression.



Fig. 131.—Same as Fig. 130 after rib-graft. Incision made within ala.

be noticeable or in the line of the eyebrow. The objection to this was lack of drainage. Later incision was made within the vestibule of the nose on one side, which had the advantage of drainage, but if great care was not taken, infection occurred.

Recently it has been suggested that incision be made in the columella

which apparently overcomes the objections to the other incisions, but the possibility of separation of the alar cartilages must be considered. The removal of the hump, which is usually bone but may be partially cartilaginous, is easily done with rasp or forceps and the wound closed with sutures, preferably horse-hair.

Next to the hump-nose, the so-called "saddle-nose" requires attention. Repair of this requires the same incision which should be one of the above mentioned for hump, but requires something to fill the space. After incision is made, separation of the soft parts from the bone should be thoroughly carried out. This may be by blunt dissection or with the knife—our preference being a long thin-bladed scissors—carrying the separation to the nasofrontal suture or above and well down both sides to the inner canthus if the deformity is large. For small depressions we prefer small pieces of cartilage from the ear as suggested by New, but for large, the seventh, eighth, or ninth rib with cartilage for the lower end.



Fig. 132.—Blastomycosis of nose.



Fig. 133.—Destruction of tip and right ala from lupus.

It is probably unnecessary to state that in removing a section of the rib care should be taken not to tear the parietal pleura, but as this accident has been reported a number of times, and as the result is likely to be more disastrous than the deformity which is being corrected, warning is not out of place.

If the rib is removed subperiosteally the above accident is not so likely to happen and regeneration of rib apparently occurs very soon. Periosteum and subcutaneous tissue are sutured with catgut and skin closed with Michel clips or other suitable sutures. Shaping of graft requires some skill and care must be taken not to have graft leave sterile field. At times the whole rib may be required, at others, slabs may be made by splitting the rib. The wound is closed with suitable sutures and dressing applied. The copper splint of Cohen or some other light pliable material may be used to support and protect part.

Many deformities may be prevented if recent fractures are promptly treated. Depressed fractures can readily be replaced by manipulation with instruments or the gloved finger. Bernay's sponge splints (sometimes spoken of as Simpson's, but originally Bernay's) will frequently assist replacement and act as retainers. It has been found advisable in some cases to perform a submucous resection of the septum before correction of fracture, if septum is injured.



Fig. 134.—Carcinoma.

If external wounds are present they should be cleansed and sutured, the latter tied loosely to allow for the possibility of swelling. Dressings should not be applied as maceration is likely to take place. However, if infection does occur a wet boric or magnesium sulphate dressing will be found efficacious.

THOMAS E. CARMODY.

## PART II—PHARYNX AND NASOPHARYNX

### SIMPLE ACUTE PHARYNGITIS

**Definition.**—An acute inflammation of the pharyngeal mucosa characterized by redness, swelling, submucous infiltration, hypersecretion, excessive discharge of mucus with discomfort or actual pain.

**Etiology.**—Exposure to cold. Extension from adjacent inflammations of adenoids, tonsils, sinuses or nasal mucosa. Disturbance of the digestive tract which is especially frequent in children. Constitutional diathesis. Bad hygienic conditions and sedentary occupations. Intemperance in the use of alcohol, tobacco or any other stimulant. The disease is more prevalent in youth and middle age.

Atmospheric conditions, dust, irritating fumes, draughts, and sudden changes in temperature. Certain drugs such as iodine, bromine, and phosphorus which are eliminated through the mucous membrane.

Certain fruits have a curious effect upon mucous membranes. For example, strawberries not only bring out a rash on the skin, resembling scarlet fever, but may produce erythema of the mouth and pharynx, simulating an acute infectious disease. Grapes, apples, and pears produce similar effects in those susceptible to fruit acids. Among vegetables, tomatoes and rhubarb likewise produce congestion of the pharyngeal mucous membrane. This, of course, does not mean that such fruits and vegetables are harmful to the average person, but only that in certain cases there is an idiosyncrasy to these foods which should always be borne in mind when making a diagnosis.

**Symptomatology.**—There is first a dry throat followed by a watery discharge which later becomes mucopurulent. There is a fulness or constriction in the pharynx associated with hawking or spitting. An uncomfortable sensation as of a “lump” in the back of the throat is often complained of. Pain may also be present and referred to the ears or eustachian tubes.

**Pathology.**—The same as in acute catarrhal inflammation of other mucous membranes. There is excessive exudation from the swollen overactive mucous glands. It contains fibrin, mucin, and desquamated epithelium. The fibrin depends on the peculiarities of the chemical constituents of the blood for a given person. It passes originally from the capillaries with the white and the red corpuscles of the blood. Occasionally the exudate is so highly fibrinous as to dry and form a “membrane” of nonbacteriogenic nature on the mucosa.

**Diagnosis.**—The chief thing is to know whether it is of bacterial origin. Therefore a culture should always be taken.

**Prognosis.**—Recovery is certain unless some complication such as middle ear or mastoid disease supervenes. Repeated attacks, however, lead to chronicity and incurability.

**Treatment.**—Cracked ice, ice-bag, or ice compresses should be applied.

Hot saline irrigations should be administered during the day in the first stage. An effervescent tablet of pilocarpine (1/100 gr.). Tincture of guaiac, 1 dram to the ounce of hot water or milk, t. i. d., given as a gargle is very effective.

℞. Oil of sandalwood  
 Oil of sassafras..... āā gr. ii  
 Liq. petrolatum..... 3j

May be given as a spray and often gives relief.

If of gastro-intestinal origin, give calomel and follow by a saline, *e. g.*, effervescing sodium phosphate.

HARMON SMITH.

### SIMPLE CHRONIC PHARYNGITIS

**Definition.**—A chronic inflammation involving the pharyngeal mucous membrane, with permanent alterations in the gland structure or in the submucous connective tissue or both.

**Etiology.**—It may be the result of an uncured acute pharyngitis or it may be due to constitutional disorders, with alteration of blood-supply, venous stasis, or passive congestion due to circulatory disturbance in the liver, kidneys, lungs, or heart. Sometimes there is an underlying neurosis involving the peripheral nerves. Digestive disturbances exert an influence also, and continued use of alcohol and tobacco are common causes. Sexual excesses are said to exert a marked influence. Gouty or uric acid diatheses are underlying causes at times, as are also tuberculosis and syphilis. Post-nasal discharge from a chronic sinus condition produces chronic inflammatory changes in the pharyngeal mucous membrane. Bad use of the voice, such as is indulged in by outdoor speakers and street hawkers, will produce a traumatic pharyngitis. An obstructed nose, from whatever cause, produces mouth breathing, and the inhalation of dust and germ-laden air are conducive.

**Pathology.**—There is chronic progressive inflammation of the submucous connective tissue which eventually encroaches upon the gland cells, altering the quantity and quality of their secretion. Hypersecretion is the rule until the condition becomes far advanced, when atrophy and dryness of the mucous membrane occur.

**Symptoms.**—The pharynx is congested and frequently shows surface capillaries. The mucous secretion is frequently very tenacious, making it necessary to "scrape and hawk" in order to clear the membrane. The voice is often hoarse, and it is frequently necessary to stop in the middle of a sentence to clear the throat. The voice tires easily, even though the speaking effort be not prolonged. There is a desire to swallow but, owing to the stickiness of the secretion, this gives rise to the sensation of a foreign body in the throat and to a false diagnosis of "globus hystericus." Frequently there is an accompanying laryngitis, either from extension of the inflammation or from the unhealthy discharge dropping on the cords from above.

**Diagnosis.**—The history of the case and adequate examination make the diagnosis easy.

**Prognosis.**—Persistent daily treatment at home, combined with office treatment by the physician, often relieves the condition but when marked pathological change has taken place a cure is out of the question.

**Treatment.**—This is, of course, based upon an accurate knowledge of an underlying systemic condition. As for local treatment, the mucous membrane must be thoroughly cleansed by daily irrigation with a solution containing 2 drams of salt and sodium bicarbonate each to the quart of water. At home after irrigation, the patient should use some form of colloidal silver, preferably by the drop method, from 5 to 10 drops being introduced into the nostril morning and night with the head elevated. Some solution of iodine or Mandle's solution should be applied locally by means of a cotton applicator.

HARMON SMITH.

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## INFECTIVE PHARYNGITIS

**Definition.**—Inflammation, swelling, ulceration, or suppuration of the pharyngeal mucous membrane due to bacterial infection.

**Etiology.**—Any infectious organism which can grow on mucous membrane, chiefly Streptococci, Staphylococci, Pneumococci, and Klebs-Löffler will produce this type of pharyngitis.

**Symptoms.**—The same as in simple acute inflammation of the pharynx, but more severe and associated with slight rise in temperature, headache, malaise, and anorexia. There may be pain on swallowing which is reflected into the ears and sometimes into the neck muscles.

**Pathology.**—There is ulceration and necrosis of discrete areas due to bacterial invasion which may extend deep below the mucous membrane into the muscle and form abscesses, single or multiple, and ultimately retropharyngeal abscess.

**Diagnosis.**—Not infrequently little white spots are seen in the "granular" tissue of the pharynx, sometimes ulcerative, and located behind or upon the posterior pillars.

**Prognosis.**—Usually good, but there may be toxic absorption resulting in metastatic foci in joints, muscles, or vital organs.

**Treatment.**—Antiseptic medication applied so as to be as nearly continuous as possible. Recently mercurochrome, 1 or 2 per cent., dropped through the nose into the nasopharynx and swabbed or sprayed generously over the entire upper respiratory mucous membrane has been very successful. The mucous membrane must first be cleansed with an alkaline wash to permit contact with the antiseptic. Orthoform can be blown in upon the affected areas or used in solution. Ulcers may be touched lightly with pure phenol, which relieves pain.

HARMON SMITH.

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## MEMBRANOUS PHARYNGITIS

**Definition.**—An inflammation of the pharyngeal mucous membrane from whatever cause leading to the formation of a false membrane because of the fibrinous exudate present.

**Etiology.**—It is in reality a non-specific infectious disease caused by micro-organisms other than the Klebs-Löffler bacillus. The formation of the exudate is due more to the reaction of the patient's tissues than to the type of organism present.

*Streptococcus pyogenes* is frequently found, also various strains of *Staphylococci*.

**Symptoms.**—There is the same dryness, soreness, malaise, fever, etc., which we find in the other types of pharyngitis.

**Pathology.**—In appearance and general behavior the disease resembles diphtheria of a mild type. There is no ulceration or destruction of mucous membrane but when the false tenacious membrane is pulled off, there is a slightly bleeding surface underneath due to capillary oozing.

**Diagnosis.**—Can be made only through bacterial cultures. These should be taken in every case.

**Prognosis.**—Good if there are no complications.

**Treatment.**—Cleansing with alkaline spray or douche followed by diluted hydrogen peroxide. Then apply Mandle's solution of iodine, or guaiacol, mercurochrome, acri-violet, or gentian-violet. The false membrane must first be removed to permit the action of the bactericides.

HARMON SMITH.

## ATROPHIC PHARYNGITIS

**Definition.**—A disease of the mucous membrane of the pharynx in which permanent pathological changes have caused dryness of the mucosa and a replacement of the mucous glands by fibrous tissue.

**Etiology.**—In many cases this condition, which is also known as pharyngitis sicca, is associated with sinus disease and in every case the sinuses should be investigated. It is most often secondary to atrophic rhinitis. The inhalation of irritating fumes or vapors and the presence of trophic lesions are potent causes. In many cases the condition is associated with nasal obstruction.

**Pathology.**—In most cases the normal glandular elements in the mucosa have been replaced by connective tissue so that the glands are eventually obliterated. Not infrequently the surface is coated with a thin, dried, mucofibrinous crust which it is difficult to remove.

**Diagnosis.**—Simple inspection diagnoses the condition at a glance.

**Prognosis.**—The outlook is, of course, far from favorable, as little can be done to relieve the intense dryness and discomfort.

**Treatment.**—All nasal obstructions should be corrected and any sinus infection cleared up in so far as is possible. It is very difficult for the patient to cleanse the mucous membrane at home and, therefore, frequent office treatments become necessary. Swabbing the surface with hydrogen peroxide and cinnamon water has been recommended. One drop of oil of mustard to 2 drops of oil of cassia in an ounce of neutral oil, applied

every other day, by swabbing or dropping has been recommended. Crude petroleum may be applied in the same manner. Potassium iodide given internally is excreted through the mucous membrane and may be of some advantage, but must be given in large doses. Attention to the nasal condition is paramount and the many forms of treatment indicated and applied to the nasal mucosa apply to the pharynx.

HARMON SMITH.

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### FOLLICULAR PHARYNGITIS

**Definition.**—A chronic inflammation of the pharyngeal mucous membrane, involving especially the mucous glands, which stand out as small red areas, or islands of tissue between which is a more or less normal mucous membrane.

**Etiology.**—It is not well understood why all cases of pharyngeal inflammation do not show this discrete glandular involvement but it probably depends upon the atonic condition of the lymphatics. The same underlying causes are at work here as in other forms of pharyngitis; as in repeated attacks of acute pharyngitis, influenced by occupational abuse of the voice. The habitual use of hot, pungent foods, results in overstimulation of the glandular structures and undoubtedly plays a part in the causation of this condition. It has not been disproved that there is some bacterial cause which affects the glands chiefly, to the exclusion of the entire mucosa.

**Symptoms.**—There is irritation of the pharynx and frequently a sharp hacking cough. The voice is altered, slightly hoarse, and the secretion is increased as well as altered.

**Pathology.**—There is submucous infiltration, with proliferation of cells. The blood-vessels are relaxed and distended, especially the veins. In certain areas there is a thickening with the formation of fibrous tissue. In the glandular areas there may be retention of secretion, which probably accounts for the swollen appearance.

**Treatment.**—As in other pharyngeal conditions, the general health of the patient must be studied and brought to normal in so far as is possible. For internal administration phosphorus and iodine have been recommended in varying combinations. The local treatment is directed chiefly to the follicles to which may be applied a 20 per cent. chromic acid solution, care being taken that the acid does not spread to surrounding tissue. At home the use of hot gargles gives some relief. The use of condiments, tobacco, and alcohol should be interdicted.

HARMON SMITH.

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### RHEUMATIC PHARYNGITIS

**Definition.**—An inflammatory process, usually acute, caused by the excretion of acid urates from the blood.

**Etiology.**—The vascularity of the pharynx favors the excretion of waste products from the blood and produces the same irritation as is commonly seen in the mucous membrane of the kidney. Not infrequently there is a

history of gouty and rheumatic manifestation elsewhere in the body, consisting of painful "twitches" in the muscles and joints, especially in the region of the neck.

**Pathology.**—There is no specific pathology and a congested appearance of the mucous membrane may be quite similar to that seen in other types of pharyngitis.

**Symptoms.**—The first symptom is a sensation of fulness and accumulation in the throat, with slight pain or discomfort on swallowing. There may be a rigid, stiff feeling and sometimes a feeling as if the membrane were being pricked.

**Diagnosis.**—Both the blood and the urine should be examined, in order to determine the amount of uric acid and the percentage of urea.

**Prognosis.**—Prompt and efficient treatment may cure the condition if it has not progressed too far.

**Treatment.**—Local treatment is merely palliative. Gargling the throat with a hot alkaline solution brings relief. A 5-grain aspirin tablet, dissolved in a half glass of hot water, is a satisfactory method of relieving the discomfort. The constitutional management of the case should be left in the hands of the general practitioner.

HARMON SMITH.

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### HEMORRHAGIC PHARYNGITIS

**Definition.**—An extravasation of blood into localized areas beneath the mucous membrane of the pharynx.

**Etiology.**—Usually associated with eruptive fever, *e. g.*, typhoid and typhus. It is seen in purpura hæmorrhagica, and as a result of trauma. Vomiting incident to an alcoholic debauch is a very frequent cause.

**Symptoms.**—Expectoration of blood-tinged sputum may be the only symptom. There is little or no pain.

**Pathology.**—Small, dull red, edematous areas in the mucous membrane with little or no inflammatory reaction are found on the soft palate and the uvula as well as in the pharynx.

**Diagnosis.**—Made by inspection, but it may be difficult to determine the underlying cause of the condition. In every case the general constitutional elements must be studied, including blood examination.

**Treatment.**—Depends on the cause. Locally, hot alkaline gargles, and astringents, such as alumol and sulphocarbolate of zinc are useful.

HARMON SMITH.

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### ACUTE INFLAMMATIONS OF THE VELUM PALATI AND UVULA

Among the simpler forms of affections of the soft palate are edema, secondary invasions from other adjacent areas, such as the tonsils, pemphigus, leptothrix, aphthæ, lupus, herpes, actinomycosis, and conditions associated with palatal involvement, such as stomatitis and exanthemata. One must not overlook mucous patches found in secondary syphilis and occasionally Vincent's angina.

**Edema.**—Infiltration of the uvula is a very common affection and when it occurs it is described by the laity in the phrase, "My palate is down." It occurs from the ingestion of foods that are too hot or poisonous to the individual, such as is shell-fish to some, from trauma, alcohol, particularly from too much champagne, when too cold, and in association with tonsillar infection and following tonsillectomy.

**Treatment.**—The uvula after anesthetization with a 10 per cent. cocaine solution may be pierced in several places with a sharp pointed bistoury to evacuate the accumulated fluid. This usually suffices to bring about prompt relief from the mechanical interference. There is little if any pain in this condition, but the discomfort is annoying and in some instances alarming.

**Pemphigus.**—This is a disease caused by the formation of bullæ, which after absorption, or eruption leave pigmented spots or an area of coalesced membranous exudate. It may be attended by itching, burning, and even severe pain and, therefore, becomes sometimes unbearable to the patient. It is occasionally associated with a skin manifestation and, therefore, presents itself to the dermatologist as frequently as to the laryngologist. The disease is progressive and ultimately results in a gastro-intestinal involvement which is fatal.

**Treatment.**—No medication is abortive, but hot saline irrigations followed by insufflations of orthoform or the local application of anesthesin lessen the discomfort.

**Leptothrix.**—A parasitic disease associated with tartar of the teeth or gangrene of the lung, typhus fever, etc. Microscopically it is made up of bundles of filamentous rods, often forming pseudo threads or strands. It is difficult to recognize clinically and usually the diagnosis must be made by the microscope.

**Treatment.**—Caustic or the actual cautery reduces the formations, and lessens the irritation which they occasion. Change of diet and location have aided, in many cases, in the disappearance of the growth.

**Aphthæ.**—This condition is also known as *thrush*, or *aphthous stomatitis*. It is a disease found chiefly in infants and is characterized by the formation of whitish spots in the mouth, due to the presence of a fungus, *Oidium albicans*. The aphthæ are followed by shallow ulcers, often attended with fever and gastro-intestinal irritation.

**Treatment.**—The spots may be touched with 60 gr. to the ounce of silver nitrate. The old-fashioned method was to swab the spots with borax and honey. Mild aperients, followed by a general tonic and sustaining treatment, are often useful.

**Lupus.**—Lupus vulgaris is a tubercular disease of the mucous membrane and skin, marked by the formation of brownish nodules. The lesions appear in various forms and are named according to their character, such as lupus vegetans, lupus serpiginosus, pustular lupus.

**Symptoms.**—Subjectively the symptoms of lupus are not marked. Pain is practically absent and often considerable destruction of the palate has taken place before the patient consults a physician. Small light-colored points are observed, resembling miliary nodules, which are followed usually by ulceration. There is a necrotic focus, covered by grayish secretion. These isolated areas tend to become confluent, producing large areas of ulceration and destruction.

The velum palati may undergo swelling, ulceration, contraction and scar formation, interfering with deglutition, closing the posterior nares and giving the voice a nasal "twang." At this stage it may be mistaken for rhinoscleroma and the diagnosis may have to be determined by taking a section for microscopic study.

*Prognosis.*—The outlook for a cure is unfavorable. Many cases die from tubercular conditions of the lungs, probably secondary to the dissemination of the tubercle bacilli from the lupetic area.

*Treatment.*—Surgical removal, curetting and the use of the galvanocautery are the most useful means of combating the malady. D. Braden Kyle has recommended cleansing the surface and insufflating 5 per cent. pyoktanin in stearate of zinc.

If the disease extends to the larynx and causes obstruction to breathing, tracheotomy is indicated. Roentgen ray and radium have been successful in a measure when the lesion has been readily accessible.

*Herpes.*—The affection is probably due to circumscribed inflammation in the terminal nerve filaments. There is always a certain amount of discomfort, sometimes pain and occasionally intolerable itching of the fauces. Small papules, purplish red in color, are contrasted markedly with the surrounding normal mucosa. The condition is usually unilateral and disappears in from five to ten days; but may recur after being absent for months.

*Treatment.*—Locally orthoform tablets or anesthesin may be used for the pain and discomfort. The general system should not be neglected and it is well to employ cod-liver oil, hypophosphites and the lactophosphates of lime, iron, and arsenic.

*Elongation of the Uvula.*—Relaxation of the muscle fibers composing the uvula as well as of the mucous membrane and the connective tissue brings about lengthening, or elongation, of the uvula. This condition may not be noticed by the patient and may give rise to no symptoms whatever. Not infrequently, however, it causes a chronic, irritating cough, because it touches the epiglottis and sets up reflex activity in the larynx, thus producing fits of coughing.

*Treatment.*—Astringent sprays, gargles, etc., are useless in this condition. Amputation is the only measure of value. An expedient method of removal is by means of the lingual tonsillotome, used in the inverted position. It is important that not more than the lower third be removed, as too great a removal causes voice impairment, particularly in singers in whom the high tones are chiefly affected.

HARMON SMITH.

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## EXANTHEMATA

They all have their beginning upon the mucous membrane.

*Scarlet Fever.*—*Definition.*—An acute infectious disease of the mucous membrane of the pharynx, mouth or tongue, characterized by a deep bluish-red infection and highly swollen papillæ of the posterior part of the tongue giving rise to the so-called "strawberry" tongue.

*Etiology.*—It is thought to be due to a special kind of streptococcus, and by some ascribed to Class's bacillus, the *Diplococcus scarlatinæ*.

*Symptoms.*—Sore throat, malaise, anorexia, and fever for one or two days followed by the characteristic skin eruption in one to two weeks.

*Pathology.*—Osler classifies the intra-oral and pharyngeal lesions into three groups:

First: Slight redness with swelling of the follicles of the tonsils.

Second: A more intense grade of swelling and induration of the parts with follicular tonsillitis.

Third: Membranous angina with intense inflammation of all the pharynx structure accompanied by cervical adenitis. The process tends to spread to the eustachian tubes giving rise to middle ear and sometimes to mastoid complications. It is undeniable that the pharyngeal tonsil harbors the organisms for a long time after, if not before manifestations of the disease.

*Diagnosis.*—Chiefly by inspection, but likewise by history of exposure and characteristic rise of temperature. Undoubtedly there are many "missed" cases.

*Prognosis.*—Uncertain at best. There are likely to be several complications which add to the gravity of the outcome.

*Treatment.*—Ice-bags or ice compresses externally. Occasionally heat is better. Hydrogen peroxide diluted or in full strength should be employed as a mouth-wash and gargle. To local areas there should be applied some antiseptic according to the preference of the attending physician, as Lugol's solution, Zonite in half strength or some alkaloidal silver preparation.

*Measles.*—*Definition.*—A local infection of the pharyngeal and buccal mucous membrane, characterized by the so-called Koplik spots.

*Etiology.*—Unknown, probably microbic.

*Pathology.*—Has not been adequately elucidated because of the short duration of the condition and its fleeting evasive nature.

*Treatment.*—Antiseptic sprays, gargles, and applications to mouth and throat. Particular attention should be paid to the rhinitis and bronchitis always associated with this disease. The bronchitis often goes on to a fatal bronchopneumonia. Nasopharyngeal irrigations with the postnasal syringe of 20 per cent. argyrol, 50 per cent. Collene or 50 per cent. hydrogen peroxide solution have lessened the eustachian tube complications.

HARMON SMITH.

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## ACTINOMYCOSIS

*Definition.*—A disease of the mucous membrane in man transmitted to him from cattle by the ray-fungus, or *Actinomyces bovis*.

*Etiology.*—In animals the disease is commonly known as "lumpy-jaw" and it is thought to be due to infected barley or rye. It is transmitted to man through any means whereby the fungus can reach the mucous-membrane surface.

*Pathology.*—A granulating tumor develops at the site of the fungus and forms a nodule of small round cells, surrounded by inflammatory reaction,

not unlike a sarcomatous growth in appearance. Infection is transferred both through the lymphatics and blood-vessels and is essentially chronic in nature.

**Symptoms.**—There may or may not be considerable pain but continuous aching is the rule. The breath is foul. Suppuration from irregular deep-seated sinuses is commonly seen.

**Diagnosis.**—It is important to differentiate it from sarcoma. This can be done only by laboratory methods.

**Prognosis.**—This is a chronic disease which slowly depletes the vital resistance of the patient throughout many months until death ensues.

**Treatment.**—Thorough surgical removal or destruction by cauterization of the infected area offers the only hope of relief. Medical treatment and local applications are practically useless.

HARMON SMITH.

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### RETROPHARYNGEAL ABSCESS

**Definition.**—An abscess located between the posterior wall of the pharynx and the vertebral column.

**Etiology.**—It is commonly seen in marasmic and undeveloped children and not uncommonly in the offspring of tuberculous or syphilitic parents. It is probably secondary to infection of the lymphatic glands, and most frequently to infection of the lymphoid tissue in the nasopharynx.

**Pathology.**—The disease has an insidious onset. The exciting agent, whether a parasite or germ, develops beneath the mucous membrane, may go on for many weeks until it burrows deeply into the neck, extending downward, if unchecked, into the mediastinum.

**Symptoms.**—There may be slight rigidity of the neck; the head may be held to one side and the child may not be able to swallow or breathe normally through the nose. There may be a slight cough and a peculiar alteration of the voice. The pain, if any, is deep-seated and constant, increasing until the abscess ruptures or is opened.

**Diagnosis.**—The condition may be limited to the nasopharynx and, therefore, will not be readily seen upon inspection, or to the oropharynx, but is often noted even as far down as the laryngopharynx. Palpation with the finger elicits a sense of fluctuation and if detected makes the diagnosis certain. The condition may be mistaken for croup, bronchitis or edema of the glottis and in rare cases must be differentiated from aneurysm.

**Prognosis.**—Recovery is usually uneventful if prompt diagnosis is arrived at, and an incision is made. Not infrequently there may be a recurrence, therefore the history of the case is very important.

**Treatment.**—Incision should be made whether pus has formed or not, as depletion of the infected area will often give relief. Usually, however, an extensive opening can be made with the child's head in a dependent position, suction being used to withdraw the pus the moment it is released. In cases of tuberculous origin the mere opening of the abscess is insufficient and the real cause of the disease is not found until necrosed bone is removed. General anesthesia is contraindicated, but the patient may be wrapped in

a sheet and held upright until the incision is made, when it is then inverted so that no pus is inhaled into the lungs. A second opening is almost always necessary on the following day.

HARMON SMITH.

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## PHLEGMONS

**Definition.**—Inflammation of connective tissue, usually ending in ulceration or abscess. It is thought to be associated with the penetration of bacteria through capillary spaces.

**Historical Notes.**—Stuart Mudd and Emily B. H. Mudd have described this phenomenon under the heading, "Transport Through Berkefeld Filters by Electro-endosmotic Streaming." These authors were able to drive a culture-medium containing bacteria through an artificial membrane by means of electricity. In their experiments they used mixed growths of *Erythrobaillus prodigiosus* and *Vibrio percolans*. The first of these is the conventional organism used in experiments with filters to prove the tightness of the filter for ordinary bacteria. *Vibrio percolans* passes readily through Berkefeld candles.

Many years ago Jonathan Wright (1896) found tubercle bacilli were able to pass through the intact mucosa of a patient having laryngeal tuberculosis, and from this he has proceeded to his present theory of biochemical surface changes which also control the progress of bacteria through capillary spaces.

J. E. Goodale, in 1897, dusted carmine on the surface of tonsils shortly before removal and found carmine particles had passed in between the tonsillar epithelial cells and in the direction of the efferent lymph channels. Jonathan Wright subsequently confirmed and extended these observations by showing that the foreign particles were taken in between the cells and that the cryptal bacteria were kept out.

G. B. Wood, in 1914, infected hogs with tubercle bacilli and anthrax by swabbing cultures of the specific organisms lightly on the animals' tonsils and demonstrated that the anthrax bacilli penetrate between the cells of the tonsillar epithelium. The crypt bacteria were kept out until the epithelium had been devitalized by the invading anthrax organism.

A. S. Warthin, in 1923, confirmed both the selective transfer through tonsillar epithelium and the intercellular course of the particles admitted. Jonathan Wright has especially urged the importance of seeking physico-chemical mechanisms associated with the reactions of electrodynamic forces of the lipoids and the proteins of the substance of the mucosæ. These are capable of explaining such selective action by the first line of defense against infectious diseases. He seeks to apply this theory to bacterial invasion in general. The alteration in the surface tension of surface epithelial cells is the first step, the bacterial invasion the second step, to infection. Thus the possibility that electro-endosmotic streaming and cataphoretic movement are at work here must be considered in relation to the penetration of bacteria through epithelial tissue at the surface.

Having these facts adequately demonstrated, we can more reasonably explain the sudden onset, the disproportionate systemic involvement and

the profound sepsis attendant upon what we may call phlegmonous pharyngitis. The ordinary quinsy, retropharyngeal abscess, and Ludwig's angina are not to be classed under this heading.

**Etiology.**—Profound shock to the sympathetic nervous system which may result from an alcoholic debauch, great mental or physical fatigue, improper or insufficient food, sudden exposure to cold, diabetes, Bright's or uric acid diathesis is a predisposing cause of this condition.

**Pathology.**—A molecular change in the ganglia of the sympathetic nervous system whereby some impulse is conveyed from which a dialysis is produced, alters the biochemical resistance at the distal extremity of the nerve. There is a change in surface tension of the mucous membrane which permits the formerly innocuous *Streptococcus*, often present for years in the tonsillar crypts or other organism, to gain entrance beneath the surface. The phagocyte and its biochemical equipment within the tissue fails to function. The antitoxin which should counterbalance the toxin of the invading organism is inadequate for the demand made upon it. Surface resistance has already collapsed, phagocytic juice is inhibited, antitoxin is annihilated, and the invading organism is left to its destructive tendencies.

Ferments now form which assimilate and digest the unresisting tissues which are destroyed, and the infection is extended even to the circulation. The type of organism varies, but the *Streptococcus hemolyticus* is most frequently found.

**Symptoms.**—Lowered resistance from mental or body fatigue; underlying dyscrasia as Bright's, lithemia, or diabetes, and recurrent attacks of tonsillitis or pharyngitis. A mild sore throat precedes the severer symptoms and the patient complains of undue malaise and fatigue. Suddenly a feeling of fullness or discomfort upon one or both sides may be complained of, followed by a sharp pain on swallowing. This is succeeded by difficulty in breathing, malaise, and possibly a chill. The suddenness of attack, with the severer constitutional symptoms, immediately conveys to the patient an apprehension which makes him dispense with home remedies and consult his doctor.

The physician finds the patient breathing with difficulty, swallowing often and with discomfort; there may be some dyspnea; the pulse is rapid and the temperature relatively low. The face is bathed in sweat, and the expression anxious. The lips are blue and the general picture alarming. Ordinarily one or both tonsils are protruding from their fossæ and extend beyond the middle line of the pharynx. The uvula is edematous; the saliva abundant and the voice modified as in a quinsy. The picture is one of a peritonsillar abscess, but no one spot of hard protuberance can be palpated. The whole pharynx and lateral pharynx is soft, mushy, or waterlogged. An endeavor to locate a pus pocket is usually ineffectual, though possibly a number of small abscesses are present throughout or around the tonsil. The epiglottis and even the larynx may be edematous due to extravasation of serum.

**Treatment.**—Ice externally by means of the ice-collar or Leiter coil; hot antiseptic irrigations of the pharynx followed by an astringent gargle; free catharsis with colonic irrigations; ample injection of fluids, and frequent pricking of the edematous areas over the tonsil will, in the majority of cases, result in a favorable outcome. A tracheotomy set should be in constant readiness for an emergency. Slight astringent sprays of adrenalin

(1 : 8000) or alumnol, gr. xx to ʒj of water, may be of help. Aggressive surgical measures are of but little avail in controlling these cases as no concentrated pus pocket exists, and the more cutting and puncturing that is done, the greater the tendency to infiltration and edema not only of the pharynx, but of the epiglottis and arytenoids.

HARMON SMITH.

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### LUDWIG'S ANGINA

**Definition.**—This condition, first adequately described by Ludwig<sup>1</sup> of Stuttgart, in 1836, is an acute septic inflammatory process involving the cellular tissues of the floor of the mouth and the submaxillary region of one or both sides of the neck.

**Synonym.**—Angina ludovici.

**Etiology.**—The origin of infection is usually within the lower gingival borders, especially around the teeth. Experimental studies of the lymphatic drainage have fairly well established the fact that such infections do not come from the region of the faucial tonsils. Bacteria may enter through abrasions, wounds, ulcerations, or carious teeth. The condition may result from delayed development of a third molar, with necrosis of the tooth and alveolar process, and abscess formation. It may follow tooth extraction. Bacteriologically we are dealing most commonly with a pure streptococcus infection, though very often staphylococci and pneumococci are also found. In a few cases pneumococci or staphylococci have been found alone. In a few cases also, *Bacillus septicus* has been isolated.

**Pathology.**—The process is essentially a cellulitis; the submaxillary gland and lymph-nodes may be found more or less intact in the center of necrotic cellular tissue. The fact that pus is not found in many cases on incision does not prove that there is phlegmonous swelling without pus formation. Cases have been reported in which autopsy demonstrated the presence of pus which had been missed by incisions made prior to death. Much of course depends on the stage of the process at the time of incision.

**Symptomatology.**—The onset is marked by difficulty in talking and swallowing, pain in the floor of the mouth, and salivation. Elevation of the tongue, with redness and edema of the mucous membrane over the involved area, is characteristic. A painful swelling appears beneath the jaw, rapidly extending into the neck, in some cases even reaching the level of the sternum. This swelling has usually a board-like hardness. The skin may be pale or dusky red. There may or may not be marked tenderness. Dyspnea from edema of the glottis may prove alarming and necessitate a tracheotomy. The temperature is not high in most cases, ranging from 99° to 103° F. A moderate leukocytosis is to be expected.

**Diagnosis.**—The symptoms pathognomonic of Ludwig's angina are those referable to an infectious process under the tongue and confined within the floor of the mouth, namely, elevation of the tongue, with redness and edema of the mucous membrane over the area involved.

Conditions from which it is to be distinguished are quinsy and retro-pharyngeal abscess.

**Prognosis.**—Mortality is high, especially if treatment is not instituted

early. Various authors have placed it at about 50 per cent. Death is usually due, according to Da Costa,<sup>3</sup> to edema of the larynx or bronchopneumonia. Thomas, who has made an extensive study of the subject, believes that most deaths are due to edema of the glottis.

**Treatment.**—Incise promptly through the swelling in the submaxillary region, using local anesthesia. “The finger should be passed upward into the wound until only mucous membrane intervenes between it and the mouth” (T. Turner Thomas<sup>4</sup>). Drainage-tubes are inserted and the part dressed with antiseptic fomentations. Irrigations with 5 per cent. mercurochrome-220 soluble may be of value. Stimulants and supportive measures such as the Murphy drip should be used when indicated.

Tracheotomy is of life-saving value when there is edema of the glottis, and it should be done without delay at the first sign of obstructive laryngeal dyspnea, which is usually indrawing at the suprasternal notch. The fact that most deaths are due to edema of the larynx emphasizes the danger of delaying tracheotomy.

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### CHRONIC GRANULOMATA OF THE THROAT (PHARYNX, FAUCES, AND MOUTH)

**Histology, Bacteriology, and Pathology.**—According to the definition of Virchow and other pathologists, a granuloma is a neoplasm composed largely of granulation tissue. This definition admits of some variations depending upon the underlying diseased conditions responsible for the development of the granuloma.

A chronic granuloma if it returns promptly after removal is nearly always the result of some pathological condition in the underlying structures, and the nature of this condition will cause some variations in its histological structure. It may at times be the cause of such pathological conditions. Some prominent pathologists believe that granulomata should not be considered true neoplasms; that the specific varieties, particularly the tuberculous and syphilitic, should be considered simply localized manifestations of those conditions.

Some light has recently been thrown on the bacteriology of granulomata of the respiratory tract by the investigations of Small and Julianelle.<sup>3</sup>

Cultures of strains of encapsulated bacilli from inguinal granulomata indicated that they belong to the mucosus group, and this was determined also in cultures from the respiratory tract.

A study was made of strains of granulomata of respiratory origin and of strains of inguinal origin. Non-motile Gram-negative bacteria and en-

capsulated bacilli, exhibiting in culture the characteristic growth of the mucosus group were discovered.

The usual types of granulomata, and it is understood that chronic granulomata is always meant, observed in the mouth, fauces, and pharynx are the non-specific (granuloma simplex), the tuberculous, syphilitic (Fig. 135), the granuloma pyogenicum (a variety of non-specific granuloma), the dental granuloma, the lupus granuloma, occasionally the polypoid form of granuloma not necessarily associated with any pyogenic or specific infection, and the malignant granuloma.

One case of an extremely rare form of granuloma of the pharynx in Hodgkin's disease has been observed by the writer. In this case there were several masses of granulation tissue in the form of neoplasms on both sides of the lateral walls of the pharynx, which probably developed in conjunction with the involvement of the cervical lymph-glands.

Granulomata occurring with lupus are extremely rare. The writer has seen a few cases on the tonsils. Very rarely the bleeding polypoid granuloma of pregnancy is seen in the pharynx.

Histologically, the non-specific granuloma consists of lymphocytes held together by a matrix of globulin and collagen, which after hardening appear as a delicate meshwork. Some polymorphonuclear leukocytes are also found microscopically. This form of granuloma may be associated with any form of bacterial infection or it may occur with chronic inflammatory processes.

The polypoid form of granuloma of the pharynx often takes on a rapid increase in size caused mainly by an increased development of the blood-vessels, with a serous or mucous edema. It is at times the result of localized circulatory disturbances. Plasma-cells are numerous.

The prompt recurrence or persistence of granulomata after removal is sometimes associated with or may be the forerunner of malignant disease, and if only a small piece of the growth is removed for microscopical examination and diagnosis the real nature of the process may be overlooked.

In the typical tuberculous granuloma there are centrally located giant-cells not very different from the giant-cells of other neoplasms, except that in this type of granuloma they are more sharply defined like the giant-cells of Langhans. Large and small lymphocytes and some leukocytes are also found microscopically. The syphilitic granuloma may also contain giant-cells, and is sometimes difficult to differentiate microscop-

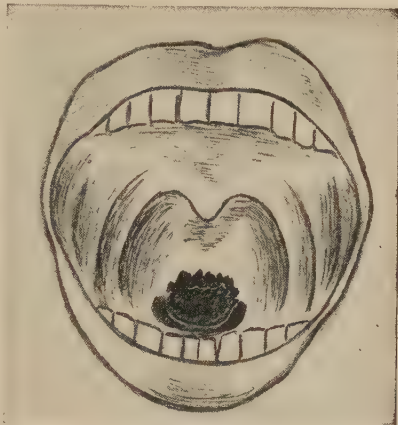


Fig. 135.—Author's case and drawing. Syphilitic granuloma on the posterior wall of the pharynx showing stage of beginning ulceration. Granulomata of this type, unless vigorous antisyphilitic treatment is promptly started, break down rapidly, causing deep ulceration of the pharyngeal wall followed by the typical radiating cicatrices. They practically always occur as a tertiary syphilitic lesion. In the nasopharynx the cicatricial tissue resulting from the ulceration of the granuloma and surrounding tissues sometimes causes occlusion of the posterior nares and nasopharynx. The drawing shows the inflammation and thickening of the surrounding mucosa.

ically from the tuberculous. Changes in the blood-vessels often cause coagulation necrosis.

Histologically the differential diagnosis between a sarcoma and some varieties of chronic non-specific pharyngeal granulomata is not always easy. The sarcoma, however, contains elements of one variety while the granuloma contains protoblasts, plasma-cells, etc.

The dental granuloma is a small mass of granulation tissue in the root of the tooth and contains bacterial deposits.

The granuloma pyogenicum is a fungating, usually pedunculated, growth containing masses of Staphylococci. This has occasionally been observed on the tonsil after attacks of peritonsillar abscess and Vincent's angina, and will persist unless the tonsil is removed.

The granuloma pyogenicum occurs at times on the mucous surface of the lips. The name was first suggested by Hartzell.

Histologically this form of granuloma is composed of connective and granulation tissue with many thin-walled blood-vessels. In the stroma and around the vessels, there is a marked round-cell infiltration and Staphylococci are found implanted in the intima of the blood-vessels.

In the lupus granuloma, giant-cells are present but tubercle bacilli are not found. It shows a tendency to sclerotic changes.

The writer has used the term "malignant granuloma" as referring particularly to cases of chronic granuloma of the sides and base of the tongue and sometimes the tonsil, that at times take on a malignant change. This is more liable to happen in cases in which the granuloma assumes a papillary type. Microscopically this is shown by a dense cellular infiltration of the growth which increases during the transition period. Granulomata along the side of the tongue that are subjected to constant irritation as from the sharp edge of a tooth, are particularly prone to become malignant in patients beyond middle life.

In the nasopharynx, in granulomata of this type, granulation tissue may invade the soft and hard palate which can be demonstrated microscopically. Many polymorphonuclear leukocytes are found in the tissues.

The invasion of the hard and soft palate with granulation tissue may result in the development of necrotic areas which sometimes spread rapidly.

The syphilitic granuloma in the nasopharynx, always a tertiary manifestation, when it breaks down results in ulceration and the formation of cicatricial tissue. The ulcerative process may extend into the posterior nares causing by the development of cicatricial adhesions complete stenosis. The bleeding polypoid granuloma of pregnancy is an extremely rare variety, but has been seen in 2 cases by the writer. In both the growth was situated below the tonsil on the lateral pharyngeal wall.

Microscopically plasma-cells are found with evidence of a serous or mucous edema.

The non-specific granuloma develops fairly frequently after the ulcerative stage of Vincent's angina and becomes chronic often recurring after removal. It occurs in the parts of the pharynx and mouth where Vincent's angina is usually observed, the tonsils, base of the tongue, mucous surface of the cheeks near the last molars, or on the gums.

This form of granuloma is also occasionally seen on the posterior wall of the pharynx coming on after a Vincent's, and springing from the ulcerated surface before it has fully healed.

Syphilitic granulomata of the pharynx frequently develop on the sites of old healed secondary lesions, probably as a result of *Spirochætæ pallidæ* remaining from the old infection. They are most frequently observed on the tonsils, the posterior wall of the pharynx, and high up in the nasopharynx.

**Etiology.**—The etiology of granulomata of the mouth, fauces, and pharynx will be considered together, as many of the same etiological factors apply to all. Some of the main facts have already been mentioned in the part of this article devoted to a consideration of the histology and pathology.

Non-specific granulomata of the tonsil (Fig. 136) that are sometimes observed after attacks of Vincent's angina, particularly in cases in which there has been extensive ulceration, are, as a rule, caused by associated infections, usually streptococcic, and not by the spirillum. They also occur after peritonsillar abscesses, but this is very unusual.

The granulomata that develop after attacks of Vincent's angina are really exuberant masses of granulation tissue caused by the ulceration of Vincent's, and while they are usually, as has been stated, caused by associated infections, the fusiform bacillus and spirillum with the spirocheta have been found in scrapings from the ulcerated surface when these granulomata break down.

A non-specific granuloma in inveterate pipe-smokers may develop on the mucous surface of the upper or lower lips, and may be caused by the constant pressure of the pipe-stem. This also at times becomes papillary and takes on a malignant change.

The etiological factor in cases of granuloma pyogenicum which sometimes occurs on the mucous surface

of the lips as a small pedunculated tumor, dark red in color, is not a fungus, but probably the *Staphylococcus aureus*. Staphylococci first become implanted in the intima of the blood-vessels as a result of which granulation tissue forms. This fact explains why recurrence promptly follows incomplete excision of the growth without thorough cauterization of the seat of attachment.

In a general way it may be said that all non-specific granulomata of the fauces, mouth, and pharynx are the result of some form of bacterial infection or of a non-pyogenic inflammatory process.

According to Sir Robert Woods,<sup>2</sup> "granulation tissue is usually the result of that attempt at repair that follows tissue destruction, or it may



Fig. 136.—Author's case and drawing. Non-specific granuloma of the right tonsil following Vincent's angina. Patient stated that it started to develop three weeks after the Vincent's angina cleared up. When seen by the writer it had been in existence for six months. Microscopically, the growth was found to consist largely of granulation tissue. The numerous plasma-cells with a suggestion of edema rather indicated the polypoid form of granuloma. Drawing shows a beginning ulceration of the growth. It returned after it was removed for histological examination, after which a tonsillectomy was performed.

in itself be the primary cause of such destruction. When the cause of such destruction is bacterial, there is usually little difficulty in isolating the organism."

The non-specific granulomata that occur on the tonsil after peritonsillar abscess are rarely observed, and are usually caused by a streptococcus or *Staphylococcus aureus* infection. They develop near the seat of the incision for draining the abscess. The discharge, irritation, and inflammation of the tissues attending the peritonsillar or tonsillar infection are important etiological factors in causing the development of the granuloma. The term "granuloma pyogenicum" may be used here also, as this particular form of granuloma is nearly always associated with *Staphylococcus aureus* or streptococcus infections.

The polypoid granuloma of the pharynx occurring during pregnancy is probably the result of a localized stasis and inflammatory process. It becomes chronic and sometimes persists after pregnancy.

Granulomata along the sides or base of the tongue, if they recur promptly after removal particularly in patients beyond middle life, should always be regarded as possibly malignant, at least the possibility of a malignant change in the structure of the granuloma should not be disregarded. Granulomata developing along the side of the tongue are often caused by some constant irritation such as from the sharp edge of a tooth. They also occur on the mucous surface of the cheek from the same cause.

Chronic granulomata on the gums, except those associated with Vincent's angina, indicate a diseased tooth or teeth. This may be regarded as pathognomonic even without a roentgenograph. The constant irritation from excessive smoking or from a pipe-stem is responsible for some of the non-specific granulomata on the tip of the tongue. In this region they also at times take on a malignant change, and this is particularly true of the granulomata of this type which become papillary.

Another type of non-specific granuloma of the tongue is caused by blastomycetes. Two cases have been reported by New.<sup>1</sup>

Syphilitic granulomata of the mouth, fauces, or pharynx are practically always tertiary manifestations of the disease.

Tuberculous granulomata of the mouth and pharynx are only seen in cases of general tuberculosis as localized lesions of the disease.

The writer has been unable to find authentic records of primary tuberculous granulomata in the literature.

The non-specific granuloma of the lateral pharyngeal columns is either caused by a previous infection (abscess), or may be the result of a chronic non-pyogenic inflammatory process.

In a recent German publication, Paul Balog<sup>5</sup> has given a very complete description of the granuloma pediculatum which occurs on both the skin and mucous surfaces. This is similar to the pedunculated granuloma which is sometimes observed on the posterior or lateral pharyngeal wall, and etilogically may be classified with the non-specific granulomata. Balog believes that important etiological factors in this variety of granuloma are irritations of a chemical or parasitic nature. Workers in chemical factories whose throats are constantly subjected to irritation sometimes develop it.

Pusey, Sutton,<sup>7</sup> and Durante<sup>8</sup> believe that the staphylococcus is the main factor in the etiology of this variety of granuloma, and Falkenstein<sup>6</sup> has determined the presence of Gram-positive masses of cocci.

**Diagnosis and Differential Diagnosis.**—The varieties that are perhaps most difficult to differentiate clinically are the tuberculous and syphilitic.

In appearance they are very similar, the syphilitic granuloma breaking down more rapidly however, and resulting in much more destruction of the surrounding parts and cicatrices. These, if the granuloma has been located in the nasopharynx, may cause adhesions between the soft palate and posterior pharyngeal wall with a complete occlusion of the posterior nares and nasopharynx.

Microscopically, the differential diagnosis is readily made. Clinically a positive Wassermann, a negative one not positively excluding syphilis, and the fact that a syphilitic granuloma is always a tertiary lesion is sufficient in the majority of the cases to establish a diagnosis.

The ulceration of a tuberculous granuloma when it breaks down is of a different character, having a worm-eaten appearance and not the dirty yellowish base of the syphilitic ulceration. The fact also that this type of granuloma is not a primary lesion, but a secondary manifestation of a general tuberculosis, will help to prove the diagnosis. Granulomata of the tongue, particularly the papillary forms, in some cases can only be positively diagnosed in the laboratory. Clinically they may resemble malignant neoplasms very closely.

The finding of blastomycetes is an important diagnostic factor in some cases of granuloma of the tongue, just as the presence of the *Staphylococcus aureus* in granuloma pyogenicum is a diagnostic aid.

This last mentioned variety on the mucous surface of the lips is a very small, pedunculated, dark red growth. On the tonsil it grows to a much larger size, and has usually a rather broad base. In this location it is dark red or yellowish-red in color with an irregular granular surface. Its structure is fairly soft and spongy, and it bleeds readily when probed.

In connection with the diagnosis of tuberculous granuloma the following case seen by the writer some years ago may be of some interest:

The patient, a young woman aged twenty-three years, a stenographer in one of the State Departments in the Capitol, consulted the writer for an irritation of the throat causing more or less constant cough. There was a mass about the size of a cherry attached to the posterior pharyngeal wall by a broad base, just above the epiglottis and near the median line (Fig. 137). This was removed with forceps and curet, and microscopical examination proved it to be a typical tuberculous granuloma containing centrally located

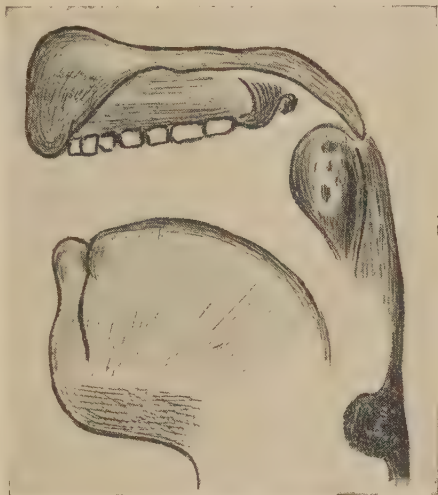


Fig. 137.—Author's case and drawing. Tuberculous granuloma on the posterior pharyngeal wall, low down. Diagnosis proved by microscopical examination. Centrally located giant-cells, Langhan's type, and some tubercle bacilli found. This drawing shows the approximate size of the growth. Granulomata of this type break down, resulting in ulceration of the pharyngeal mucosa. This growth was removed and the seat of attachment cauterized before ulceration started. The patient had pulmonary tuberculosis. This is a rare form of pharyngeal granuloma, the writer having found only a few references to it in the literature. Thickening of the mucosa at the base of the growth is shown in the drawing.

giant-cells. A few tubercle bacilli were found. Sputum examination was positive, as was the examination of the lungs. The patient got a position in Denver and made a complete recovery. The writer had occasion to examine her after she had been there a few years, and she was apparently perfectly well. Where the granuloma had been removed there was a little scar tissue, but no indication of a recurrence of the growth. Clinically it looked like a polypoid granuloma, as no ulceration had started when it was seen by the writer.

The diagnosis of granulomata of the lateral columns of the pharynx at times will present some difficulties as they may resemble malignant neoplasms very closely.

The history of a previous infection, particularly abscess, is of considerable assistance in making a diagnosis, as non-specific granulomata in this part of the pharynx are practically always associated with abscesses or a chronic non-pyogenic inflammation. They are rarely pedunculated, of a dark-red or yellowish-red color, and have a somewhat nodular surface. They are fairly soft and not freely movable, as they are attached to the pharynx by a rather broad base. They look very much like a raspberry. The form associated with a chronic non-pyogenic inflammation of the lateral column has more the appearance of a polypoid granuloma, is more apt to be somewhat pedunculated, and is lighter in color. It has a much smoother surface. They vary very much in size from very small masses of granulation tissue to a growth with a base of  $\frac{1}{2}$  inch or more.

The non-specific granulomata that develop in the tonsillar fossa after tonsillectomy may become chronic and take on the appearance of tonsil tissue. They are made up of granulation tissue and can be readily diagnosed microscopically.

The diagnosis of granulomata, developing on the tonsils, gums, or mucous surface of the cheeks after attacks of Vincent's angina, can usually be easily made, as they grow fairly soon after the ulcerative stage of Vincent's. They show a tendency to break down easily with deep ulceration. They are usually dark-red or yellowish-red in color, have an irregular ulcerated surface, and are soft and spongy, bleeding readily when probed. They vary very much in size, sometimes growing very large. When ulceration occurs there is a characteristic foul odor from the mouth. This is quite similar to the typical odor of Vincent's angina.

**Symptoms.**—Practically all forms of non-specific granulomata associated with infections or chronic inflammatory processes of the tonsils, pharyngeal mucosa, or mucous membrane of the mouth, cause surprisingly few symptoms unless complications develop after the primary causal infection has subsided. When they attain any considerable size, some slight difficulty in deglutition depending upon the location of the growth may result.

Cough from the constant irritation produced by pharyngeal granulomata is a fairly constant symptom. When located at the base of the tongue this is at times very annoying and difficult to check unless the growths are removed.

There is rarely much pain associated with non-specific granulomata of the mouth or pharynx if they do not ulcerate, but after they are removed particularly from the tongue it is a most distressing symptom.

The two important symptoms of tuberculous granulomata of the pharynx are the great pain in swallowing when they ulcerate and progressive emaciation. Cough and expectoration are always symptoms of the associated general tuberculosis, with the usual temperature elevation.

Syphilitic granulomata of the mouth or pharynx are rarely attended with

much pain in swallowing even after they break down, causing destructive ulceration of the surrounding mucosa. This ulceration is the distinguishing symptom of this type of granuloma, unless vigorous antisyphilitic treatment is started before the stage of ulceration is well established. Symptoms of constitutional syphilis are usually prominent.

The dental granuloma is usually associated with symptoms referable to the diseased tooth or teeth responsible for it. The surrounding mucosa is dusky red in color and often swollen. Pus is frequently present in the granuloma and in the adjacent mucosa.

Granulomata of any considerable size situated so low down in the pharynx that they encroach on the laryngeal entrance may cause some difficulty in breathing. The distress in breathing caused by adhesions between the soft palate and posterior pharyngeal wall the result of ulcerating granulomata in the nasopharynx, is of course extreme.

Malignant granulomata of the soft and hard palate break down occasionally causing extensive tissue destruction. In such cases there is a good deal of difficulty in deglutition, and a very offensive breath suggesting the odor of Vincent's angina. This characteristic odor is present also in ulcerating granulomata of the tonsil following Vincent's angina. There is also considerable pain in swallowing. During the ulcerative stage there is at times involvement of the cervical lymphatics, the regional glands, with some temperature elevation. The glands are enlarged, tender to the touch, and in rare instances suppurate. Suppurative processes occasionally develop in granulomata following localized infections of the lateral columns of the pharynx. When this happens there is some temperature elevation and swelling and tenderness of the cervical lymphatic glands on the same side. Suppurative processes in granulomata of this type may be associated with considerable edema of the surrounding mucosa. When this occurs and the edema becomes extensive, there may be some difficulty in breathing, and considerable pain in deglutition.

Arthritis, polyarthritis, and endocarditis may also be complications, a septic endocarditis being at times a fatal one. The same complications may occur with granulomata situated at the apex of a tooth root if the diagnosis is not promptly made and the diseased tooth removed. In rare instances, a lung abscess may follow the extraction of teeth with suppurating granulomata of the roots.

Syphilitic granulomata of the hard palate are at times associated with extensive bone necrosis which may extend into the nose. There is always a foul odor from the mouth in these cases. In cases of non-specific granuloma of the tongue taking on a malignant change, with superficial ulcerations at times, severe pain is a common symptom and one difficult to relieve. Patients with ulcerating granulomata of the pharynx or mouth following Vincent's angina, are sometimes very ill with high temperature and rapid loss of weight caused by the great difficulty in getting sufficient nourishment. This is the result of the extreme pain in deglutition, particularly when the ulceration is extensive. Fatal terminations are not extremely infrequent.

The writer has had a recent fatal case of this kind in which the ulceration from an ulcerating granuloma of the tonsil following an attack of Vincent's angina extended to the posterior wall of the pharynx, base of the tongue, and mucous surface of the cheeks. The ulcerative process

finally destroyed both tonsils, uvula, and part of the soft palate. Just before death the patient, a man aged twenty-eight years, had hemorrhages from the nose, mouth, and rectum. He practically died of starvation, as it was impossible to give him sufficient nourishment.

**Prognosis.**—The prognosis of practically all varieties of non-specific granuloma is favorable, as with proper medical and surgical treatment recurrences can be prevented. The exceptions are the ulcerating granulomata associated with Vincent's angina, causing deep destructive, ulcerative processes of extensive areas of the mouth and pharynx with serious constitutional disturbances. Occasionally a case of this kind has a fatal termination. Some granulomata of the tongue starting as simple non-specific neoplasms, and later taking on malignant changes, must also be considered as serious from a prognostic viewpoint unless seen early enough for radium therapy. Other ulcerating granulomata of the non-specific type, causing by adhesions occlusion of the nasopharynx and posterior nares, have an unfavorable prognosis so far as the permanent relief of the extremely difficult breathing is concerned.

Tuberculous granulomata have an absolutely unfavorable prognosis, and, being associated with pulmonary or other general tuberculosis, are only amenable to treatment in a proper climate.

The prognosis of syphilitic granulomata may be said to be favorable if the underlying constitutional syphilis can be cured, and if they have not caused too much destruction in the pharynx.

Lupus granulomata are not as unfavorable as the tuberculous, as there is some chance of at least prolonging the patient's life by appropriate methods of treatment.

The prognosis of granulomata of the tonsil if they become malignant is usually hopeless. Operative measures are followed by prompt recurrences, and while radium therapy does sometimes result in a disappearance of the growth for a time, sometimes for a year or more, there is usually a recurrence or a metastasis. The author has seen some serious secondary hemorrhages following the use of radium needles in tonsillar granulomata of this type.

**Treatment.**—The fundamental principle underlying the removal of non-specific granulomata of the mouth, fauces, or pharynx is the complete and proper cauterization of the place from which the growth is removed. If this base which is usually infected is not thoroughly cauterized, recurrences are the rule. The method of cauterizing is of the greatest importance, the writer preferring the electric cautery, as its action can be localized to a much greater extent than that of an acid such as acetic, which is much used for this purpose. The use of the cautery is of particular value after the removal of specific or non-specific granulomata from the tongue, or posterior or lateral pharyngeal walls. The growth is first removed with a punch forceps followed by a curetage of the place of attachment, and a day or two later cauterization. Definitely pedunculated granulomata may be removed with the snare, the loop being slowly tightened to lessen the amount of postoperative bleeding. The treatment for pyogenic granulomata of the tonsil following abscesses or Vincent's angina is, of course the removal of the tonsil after the usual surface ulceration of the granuloma has been checked. The growth is usually removed for diagnostic purposes before the tonsil is removed. The sometimes extensive ulceration that accompanies this type of granuloma is at times difficult to check. If the

granuloma developed after a Vincent's angina, the same treatment used for the ulcerative stage of Vincent's is usually successful. Locally, applications of a 50 per cent. alcoholic solution of methyl blue, salvarsan, or a 20 per cent. solution of silver nitrate have proved very useful. The subcutaneous or intravenous injection of salvarsan in bad cases has also been followed by good results.

Potassium chlorate is a specific in some cases, and the writer has found the following gargle, the strength varying according to the patient's age, more useful than anything he has used:

R. Pot. chlorat. }	.....	āā 3j.....	Gm. 3.870
Powd. alum }	.....		
Glycerin.....		3j.....	Litre 0.0038
Carbolic acid.....		gtt. iv.....	Gm. 0.2592
Aqua.....	q. s. ad.	3viij.....	Litre 0.2365

This is to be used full strength, every hour or two during the ulcerative stage. It also relieves the great pain in swallowing which is always a distressing symptom.

The treatment just given is used *only* when there is ulceration and inflammation. If granulation tissue develops again before the tonsil is removed the careful use of the cautery is very valuable. For the pain on swallowing applications of orthoform in olive oil,  $\frac{1}{2}$  ounce of orthoform in 2 ounces of olive oil, applied directly to the ulcerated surface will give much relief. The use of orthoform lozenges, and a spray of 1 per cent. anesthesin, have also been found to be very good. The cautery should be used with great care after the removal of granulomata from the tongue. The pain following its use on the tongue is at times extreme, and it is best to cauterize a very small area at each sitting. For the great pain following its use the measures just recommended have proved successful in the writer's practice. If they show a tendency to return fairly promptly after their removal from the tongue, a malignant change should always be considered. This can usually be detected on microscopical examination, and if the recurring growth is at all suspicious, radium therapy should be started at once. The removal of granulomata from the tongue is sometimes followed by free hemorrhage which can be controlled by the use of ice and an iced adrenaline spray, 1 : 5000. Small pedunculated granulomata occurring on the mucous surface of the lips should be cut off and the base cauterized. There is usually no recurrence if this method is followed. Careful radium therapy may also be used to prevent recurrences.

The treatment of tuberculous granulomata is hopeless if the patient is living in an unfavorable climate. The growth should be thoroughly removed and the base cauterized, but recurrences will occur unless the patient is sent to a proper climate. As this form of granuloma is a localized lesion of the constitutional disease, the usual remedies, nourishment and fresh air, employed in the treatment of tuberculosis should be used while the local measures are being carried out. The pain on swallowing is sometimes so severe after ulceration begins that weak cocaine solutions must be used. The writer has found the following solution of the greatest service for this purpose:

R. Cocainæ hydrochlorate.....	gr. x....	Gm. .6480
Carbolic acid.....	gtt. v....	Litre .000308 (00.3080 mils.)
Glycerin.....	q. s. ad. 3j ....	Litre .0295 (29.572 mils.)

The patient can be taught to apply this several times daily, particularly before meals. A spray of 1 per cent. cocaine solution or a 2 per cent. anesthesin solution may also be used.

Syphilitic granulomata should be most vigorously treated, and this is important if they are seen before the destructive ulcerative process begins. Locally curetage after the removal of the granuloma, followed by thorough cauterization, is always indicated, and at the same time intensive constitutional treatment to prevent if possible the development of cicatricial tissue. Potassium iodide should be given to the limit of tolerance, and in bad cases salvarsan intravenously. The occlusion of the nasopharynx by cicatricial adhesions between the soft palate and pharyngeal wall, causing great distress in breathing, presents one of the most difficult surgical and therapeutic problems known to the writer. In cases of this kind in which the soft palate and posterior pharyngeal wall are welded together by a mass of cicatricial tissue, the result of ulcerating syphilitic granulomata, the writer knows of no surgical procedure that is entirely and permanently satisfactory.

John E. Mackenty (1927) has described an operation for the permanent cure of conditions of this kind. The problem in all such surgical procedures has been to keep the mucous surfaces separated after the adhesions have been removed. Mackenty's operation, particularly in his skilful hands, promises a real advance.

For granulomata of the tonsils becoming malignant radium offers the best chance. There are always recurrences or metastases however.

The writer would emphasize again the importance of thorough cauterization of the seat of attachment in all forms of granulomata. Without this no method of treatment will be successful.

In conclusion the writer would say that this article is largely based on his personal observations. A search of the literature disclosed a rather surprising scarcity of original articles on this important subject. There are very few references in the literature to it. Some of the late text-books devote a very small space to a consideration of granulomata, while in some of the older books the subject is not mentioned at all.

The same thing was found to be true in looking up the subject of granulomata of the ear.

The drawings used for the illustrations were made by the author while patients were under observation.

CLEMENT F. THEISEN.

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#### TONSILLOSCOPY AND ITS RECENT REVELATIONS

The trend of the world's medical literature is strongly in opposition to the surgical practice which has been in vogue for a generation or more of enucleating the faucial tonsils without a positive knowledge of the exist-

ence of potential pathogenes within those lymphoid organs. This trend is based mainly upon the conviction of an ever-increasing number of judicial observers that the destruction of large segments of normal and, therefore, fully functioning, lymphoid tissues has subsequently led to nutritional dysfunction.

Critical commentators on the existing tonsil situation do not question the necessity for complete eradication of extensively infected tonsils nor, indeed, in certain cases, those with a limited involvement, but they very properly and wisely insist that the best interests of the race will be served by allowing the tonsils of health, whether enlarged or not, to remain untouched.

The demand of the time is, therefore, that we should acquaint ourselves with every possible means of determining when the tonsils are those of health and when they are the seats of infection, and if the seats of infection in what way and to what extent. To assist in meeting the demand for such discriminating knowledge, a simple and everyday-workable method of diagnosis, by what is known as *tonsilloscopy*,<sup>1</sup> is offered in this section.

Tonsilloscopy is the method of determining by means of the *tonsilloscope*<sup>2</sup> the nature of the contents of the faucial tonsils, their branches, and all the other lymphoid structures in the laryngopharynx, known as the *infratonsillar nodules*, while they are lying upon their beds in the throat. The method is based upon the art of transillumination. During transillumination the lymphoid tissues are uniformly luminous, and while in a luminous state the conditions existing within them are to a large extent disclosed. Tonsilloscopy enables the examiner to instantly determine whether the tonsils and infratonsillar nodules are in a state of health or are the seats of infection. If they are the seats of infection, then, through an interpretation of the signs in the tonsil color code as displayed in Plate II, the method will enable one to quickly decide whether or not pus is present in the stroma of any of those lymphoid structures. The presence of pus in lymphoid tissues gives rise to local chronic irritation. Local chronic irritation produces local chronic hyperemia. The shade and disposition of the hyperemic coloring during transillumination are reliable indicators of the nature, extent, and often the exact locations of the collections of necrotic material.

**The Infratonsillar Nodules.**—To make this subject clear in the text it will be necessary, before describing the tonsilloscope and tonsilloscopy, to explain the meaning of the term "infratonsillar nodules" for those structures play an all-important part in the subject to be considered in this section.

In so far as they are the harbors of necrotic material the nodules were disclosed in tonsiloscopic explorations of the lymphoid tissues of the laryngopharynx.<sup>3</sup> The tonsilloscope revealed the fact that when the nodules are developed enough to be seen they are nearly always pathogenic culprits. The diagrammatic drawing of the pharyngeal cavity shown in Fig. 138 will convey a clearer understanding of the anatomical locations of those structures and their relation to neighboring tissues than could possibly be obtained from a prolixity of description. As the white silhouetted spaces in that figure indicate, the nodules are set upon the submucosa in much the same way that plaster forms are set upon the ceiling of a room.

The four tonsils are constant anatomical structures, but the infratonsillar nodules cannot be seen or felt when in a state of health. Their macroscopical presence in continuous lines or solid columns and fields is



Fig. 138.—Diagrammatic drawing of the pharynx as it would appear if viewed from the back of the neck. It shows in white and dotted-white silhouette the faucial tonsils lying in the palatine folds, the tonsil branches lying upon the lateral walls of the pharynx and the base of the tongue, the lingual lymphoid apron and the lingual tonsil. The dots show, approximately, the locations of abscesses and abscessed crypts in the infratonsillar lymphoid tissues of the average subject. In the group of structures on one side, T represents the faucial tonsil; PB, the pharyngeal branch; LB, the lingual branch; TR, the trunk, made by the union of the two branches; LA, the lymphoid apron; LT, the lingual tonsil; CP, the circumvallate papillae; and NP, the necrotic tufts on the papillae of the dorsum of the tongue.

nearly always an expression of a pathological state; but at times it is an expression of a physiological need of such tissues. The physiological need



PLATE II  
TONSILLOSCOPY



Colored figures showing the anterior pillars of the fauces and the faucial tonsils with their branches as they appear *in situ* during transillumination with the tonsil lamp alone.

## THE COLOR CODE OF THE FAUCIAL LYMPHOID TISSUES

The figures in the plate represent the luminous appearances of the tonsils, their branches, and the lymphoid apron on the base of the tongue while the lighted lamp is held behind them. With the tonsil microscope it is possible to make graphic displays of the contents of all illuminated lymphoid structures, but a knowledge of such details, while at times highly desirable, is not essential in every-day practice. With the object of reducing this method of diagnosis to its simplest form, microscopic details have been omitted in the figures. It can, however, be confidently asserted that the colors and the shades and values of colors shown in these figures, as produced by the lamp alone, clearly indicate the presence of the various conditions which may be found in the faucial tonsils and their branches, and the lymphoid apron on the base of the tongue, from early childhood to old age.

1. Represents Class 1—The uniform warm amber color of the tonsil revealed by transillumination shows that it is the tonsil of health. The anterior pillar always lights up in a bright-red-rose color which by contrast shows the amber tonsil clearly. The tonsil of health has no branches.

2. Represents Class 2—Transillumination of the tonsil in this class shows by a pink or pink-amber uniformly diffused coloring that detritus only is present in the tissues, for there are no evidences of pus.

Transillumination of the branches in this class shows small, bright-red-rose-colored areas in fields of pink-amber which indicate the presence of scattered collections of pus in the form of abscesses or abscessed-crypts, which give rise to the mottled appearance of the branch in this figure.

3. Represents Class 3—In which the tonsils are honey-combed with detritus and collections of pus, and the tonsil branches, the lymphoid apron on the base of the tongue, and the lingual tonsil contain detritus and pus in practically all of their crypts as well as various sized abscesses in their stromas.

The tonsils and extratonsillar lymphoid tissues in this class transilluminate in an evenly distributed bright-red-rose color of about the 60 or 70 per cent. shade in the hemoglobin scale. This high coloring during transillumination of lymphoid tissues always denotes the presence of pus.

As shown especially well in illustration 1 in this plate, the normal anterior pillars, because of their contained muscular fibers, also light up in a bright-red-rose shade of color and can, therefore, be used as controls for the pus shade of red in the tonsillar tissues. When the shade of red of the anterior pillar, faucial tonsils, and extratonsillar lymphoid tissues is practically the same, it indicates that all of those structures are honey-combed with collections of pus.

4. Represents well-developed lingual branches and, at each end, parts of the trunks of the branches as they appear at the back of the depressed tongue during transillumination. They always light up in a uniform shade of red as the lamp is swept rapidly behind them. An evenly distributed bright-red-rose shade of color, invariably shown in every part of all developed infratonsillar lymphoid structures, indicates the presence in those structures of numerous small abscesses and, therefore, when developed all of the lymphoid tissues in the laryngopharynx can be accepted as potential sources of infection.

When used as a key to interpret the appearances of transilluminated lymphoid tissues *in situ* the figures in the plate should be viewed by artificial light only.



in childhood and youth is frequently expressed by their development, often to a great size, in compensation for the loss of the faucial tonsils and adenoid growths soon after those structures have been removed.

The group of pharyngeal lymphoid structures in Fig. 138 consists, on each side and as initialed in the figure, of: T, a *faucial tonsil*. The faucial tonsil needs no description in this connection, for a knowledge of its anatomy and pathology, but not its function, is age-old. PB, a *pharyngeal branch* of the tonsil lying upon the lateral wall of the pharynx. The pharyngeal branch tapers off to a free end on the lateral wall, and as shown in Fig. 139,

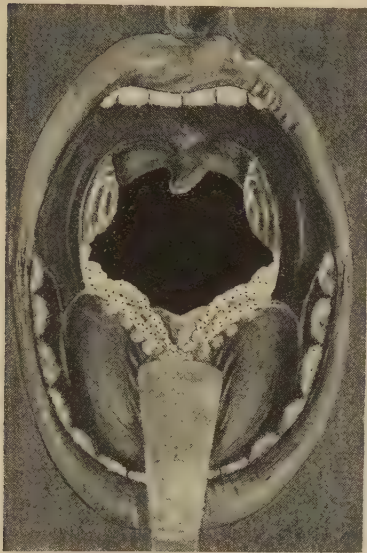


Fig. 139.—Direct disclosure of the laryngopharynx by deep depression of the base of the tongue showing: (1) In dotted-white immediately below the faucial tonsils, the trunks of the tonsil branches; (2) also in dotted-white, the lingual branches of the tonsils, directly in front of the epiglottis, extending from one tonsil to the other, and (3) in slightly shaded dotted-white, a perspective of the lymphoid apron on the base of the tongue, in front of the lingual branches and below the circumvallate papillæ. As in Fig. 138, the dots indicate the positions of chronic abscesses and abscessed crypts as disclosed by the tonsilloscope.

the upper extremity of that structure can usually be disclosed by deep depression of the tongue. If, however, it cannot be so disclosed, then its presence can readily be detected in the laryngeal mirror or by the sense of touch. LB, a *lingual branch* of the tonsil. The lingual branch is, in reality, a continuous structure extending from one

trunk to its fellow on the opposite side of the throat, with only a notch to mark the median raphé, but for the present it seems best to speak of that long, cord-like stretch of lymphoid tissue, measuring several inches, as having two parts and refer to them as branches. The lingual branches are not developed until late childhood. In early childhood the pharyngeal branches and undeveloped trunks only are found. Except in subjects with excessively irri-

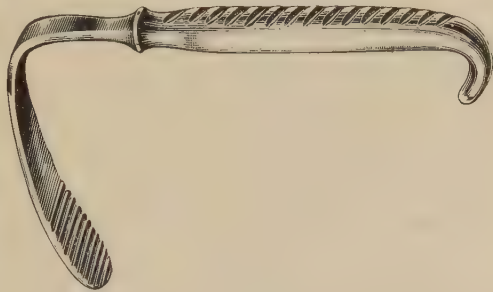


Fig. 140.—Tongue-base spatula. Its effective use is illustrated in Fig. 139.

table fauces the lingual branches can usually be directly disclosed by depressing the tongue with a strong tongue spatula. The view obtained in that way is represented in Fig. 139. The tongue-base spatula (Fig. 140) will flatten out the tongue for this purpose much better than the ordinary tongue depressor. TR, a *trunk* of the tonsil branches. The upper extremities of the pharyngeal and lingual branches are fused together immediately below the tonsil to form the trunk of the branches. The "trunk" lies in close contact with the inferior pole of the tonsil and is, in fact, attached to it by its capsule, for fibrous capsules underlie, in continuity, the

branches, trunks, and tonsils. The continuity of capsule of tonsil, trunk, and pharyngeal branch is illustrated in Figs. 156, 157. LA, the *lingual lymphoid apron*, which, perhaps, is the most important division of the infratonsillar nodules. It is a wide and usually thick blanket of lymphoid tissue framed in by the lingual branches of the tonsils. It overlies nearly the entire base of the tongue and supports near its center the LT, or the *lingual tonsil*.

In Fig. 138 the outer margins of the continuous capsules are indicated by heavy black lines on the outer sides of the silhouetted tonsils, trunks, and pharyngeal branches, and below the lingual branches. The display of this heavy line feature in the figure will, we believe, assist in making it clear that parts of the infratonsillar nodules are spoken of as branches of the tonsils because their underlying capsules being continuous with the underlying capsules of the tonsils make it easily feasible to enucleate them together with the tonsils. The photographs reproduced in Figs. 156-161 illustrate the one- and two-piece expanded tonsils which are removed from children, adolescents, and adults.

#### NON-INFECTED CRYPTS, ABSCESSSED CRYPTS, AND CHRONIC ABSCESES IN ALL DEVELOPED LYMPHOID TISSUES BELOW AND BETWEEN THE FAUCIAL TONSILS

Scattered over the free faces of the tonsil branches, the lingual tonsil and the lymphoid apron on the base of the tongue, there are many openings to crypts, the crypts themselves varying considerably in their capability of retaining foreign material. One variety of crypt which is capable of retention lies flat under the mucosa like a wall-pocket and has a long, slit-like mouth. Others, with small mouths, either penetrate part way through the lymphoid structures or extend to the underlying capsules. Deeply penetrating crypts frequently throw out branches along their courses (Fig. 142), or at their distal extremities. The mouths of the deeply penetrating crypts are commonly situated at or near the centers of the eminences of the lymphoid convolutions. Most of this type are filled and many are distended with necrotic tissue. Figure 145 is a photograph of a well-developed trunk of the branches which shows clearly upon its free face a variety of the shapes and sizes of the cryptic mouths which appear upon the surfaces of all infratonsillar lymphoid bodies.

After the enucleation of the branch structures the constricted mouths of some of their crypts are found to have been ruptured. A ruptured outlet is shown in Fig. 141. Many of the broken outlets are, doubtless, the result of the overstuffing of the crypts with pus and detritus. This is indicated by the occasional protrusion into the throat of white plugs which, apparently, are in the process of expulsion. Nevertheless, the constricting pressure exerted by the dull blade of the tonsillectome will probably account for many, if not most, of the ruptures appearing in enucleated specimens.

Convincing proof that the necrotic material in the crypts of the trunks, branches, and lymphoid blanket on the base of the tongue is the same in character and behavior as that in the crypts of the faucial tonsils is, in part, supplied during attacks of acute cryptic tonsillitis by the display upon the surfaces of the infratonsillar tissues of multiple discrete exudates which, in every way, appear to correspond to the display of exudates overlying

the faces of the tonsils. Indeed, in subjects from whom the tonsils have been enucleated discrete cryptic exudates appear upon the surfaces of all of the infratonsillar structures during attacks of acute sore throat.

In our earlier published studies of developed lymphoid tissues in the throat below the tonsils,<sup>3, 6</sup> we stated that pus was rarely present in the infratonsillar nodules. At the time those studies were made we were obliged to assume that the large number of definitely outlined bright-red-rose-colored zones which were disclosed in those tissues during transillumination, were produced by the highly irritating nature of the foreign

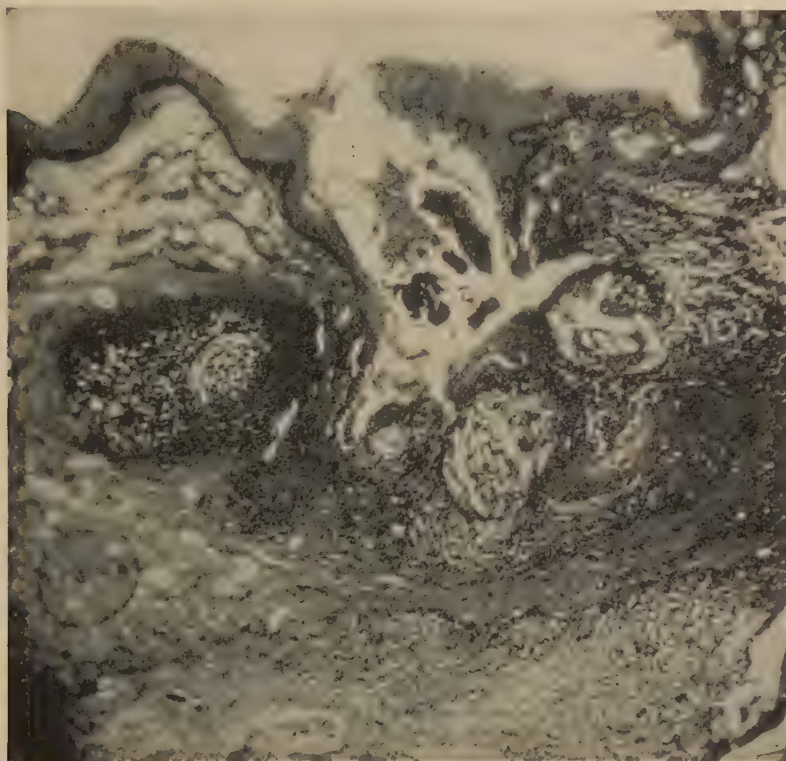


Fig. 141.—Section of pharyngeal branch of tonsil from a boy eight years of age showing a ruptured, dilated crypt from which the plug which blocked the opening of the crypt is escaping. The floor of the crypt is spread widely above inflammatory tissues, the result, no doubt, of the previous presence of pus behind the plug. At the left is a collection of necrotic material apparently on the point of breaking into the cryptic cavity. Part of the capsule of the branch structure can be seen in right lower corner. (Section by Dr. Greeley.)

material in the crypts, for all of the early reports from the laboratory refuted the insistently positive pronouncements of the tonsilloscope that such rosy spots in lymphoid tissues are always the outward signs of the presence of collections of pus. Despite the laboratory refutations, however, our faith in the tonsilloscope as an instrument of precision remained unshaken, and subsequent developments proved it to be well founded, for quite recently, under the guidance of the tonsilloscope and with the assistance of Dr. Horace Greeley in his private pathological laboratory, we began another investigation of the stroma of the lymphoid tissues below the

tonsils, which six years earlier had been fully exploited with the tonsillo-scope. That short study was sufficient to reveal the presence of many disseminated collections of pus.

Later, however, with the assistance of Dr. J. A. de Veer, assistant pathologist in the Hoagland laboratory of the Long Island College Hospital, and also with the co-operation of Dr. Harry Meyersburg, Chief of Clinic of the Department of Otolaryngology in the Long Island College Hospital, and Dr. G. R. Hageman, assistant in that department, a more intensive study revealed the practically invariable presence of great numbers of small and large abscesses and abscessed-crypts in all developed lymphoid structures in the laryngopharynx—and that means all of the fields of

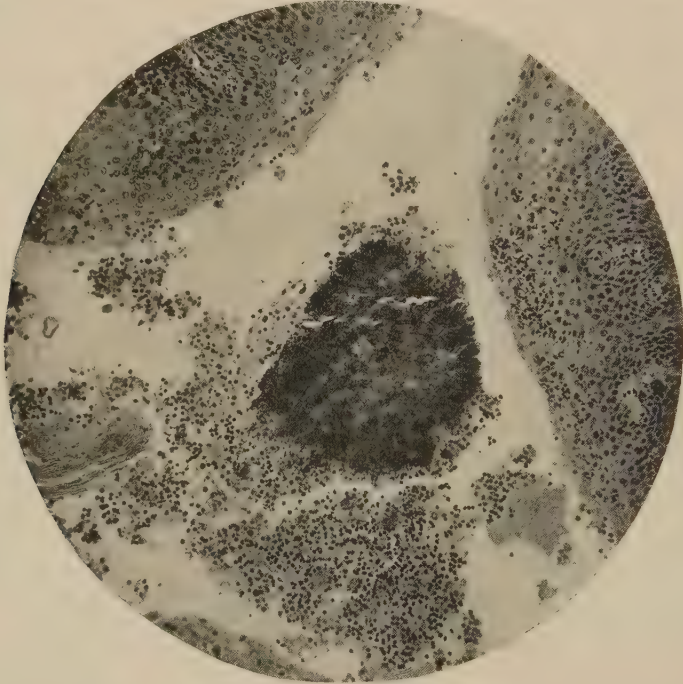


Fig. 142.—Deep, abscessed crypt in pharyngeal branch of a child nine years of age, showing, at bifurcation of cryptic branching, a plug composed mainly of bacteria. The plug evidently had been wedged into the tube above and, as a consequence, an abscess had formed behind it. The walls of the crypt show extensive polynuclear cell infiltration. The pus-cells can easily be seen through a magnifying glass. (Section by Dr. de Veer.)

lymphoid tissue outside of the faucial tonsils which are displayed in Fig. 138. The failure to demonstrate the presence of pus in those tissues in the earlier laboratory examinations (1918–19) is, we believe, adequately explained by the dearth of skilled workers in the research department as a transient consequence of the World War.

However, it is now known that the numerous small, bright-red-rose-colored zones which appear during transillumination of those tissues are produced by collections of pus within the walls of abscesses and the walls of crypts. With the exception of the large cystic abscesses about to be described, all abscesses and abscessed crypts appear by the diagnostic method of internal lighting up of the tissues as rosy zones and as those

solid, bright-red-rose-colored spots are set in fields of amber (the transillumination coloring of non-infected lymphoid tissues) they stand out very distinctly by contrast. The explanation of the occurrence of abscesses in the crypts is, apparently, that necrotic plugs hermetically seal up the cryptic mouths and, as a consequence, abscesses form in the blind ends of the crypts behind the plugs.

During its inquisitive wanderings through the fields of lymphoid tissues in the lower pharynx the tonsilloscope disclosed large, dusky areas which proved to be collections of pus. These large abscesses, some of which are cystic as shown in Fig. 144, occur in children as well as adults. At times they develop directly upon the free surfaces where their exposed walls resemble blisters. When embedded in the tissues they can often be seen by transillumination. We have located and afterward eliminated such

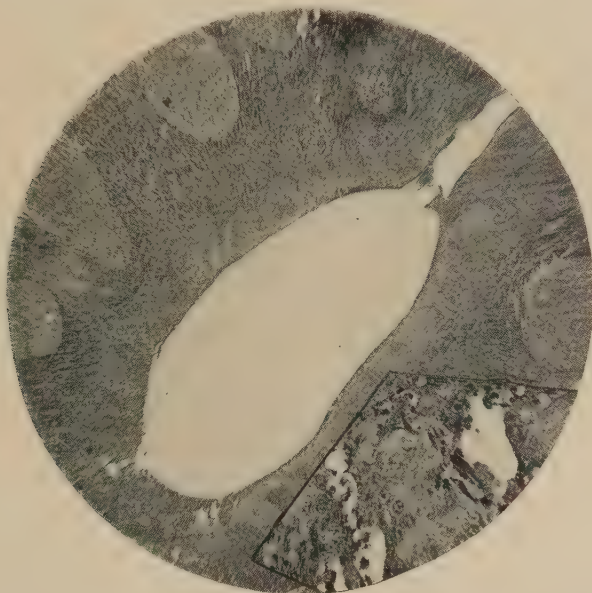


Fig. 143.—Ballooned abscess cavity at distal extremity of a tubular crypt in a pharyngeal branch of a child seven years of age. Part of necrotic plug which blocked the crypt and gave rise to the abscess still remains in outlet of expanded cavity. Insert is a high-power view of abscess wall showing extensive infiltration of polynuclears. A magnifying glass, if needed, will disclose them. (Section by Dr. de Veer.)

abscesses *in situ*. The limiting walls of the encysted variety, composed of fibrous connective tissue, are quite thin. The encysted abscess displayed in Fig. 144, which measures  $\frac{1}{2}$  inch through its longest diameter, is the largest we have seen in the infratonsillar structures. That this variety of abscess may remain in the tissues for many years is significantly suggested by an occasional history of outside tenderness for a long period of time over the site from which a pouch of pus has ultimately been removed.

As to the frequency of occurrence of abscesses and abscessed crypts we regard it as well within the limits of safety to say that they are present in varying numbers and sizes in all of the laryngopharyngeal lymphoid structures in the vast majority of human subjects beyond the stage of infancy—in other words, a practically universal condition.

The *size* of the abscesses and abscessed crypts varies from that of the head of a small pin to  $\frac{1}{2}$  inch in diameter. Even in children under ten years of age cold abscesses as large as kernels of corn are not infrequently found on or near the free faces of enucleated trunks and pharyngeal branches. In adolescents and adults large abscesses grouped in clusters of three or four or more on or near the surfaces of trunks, pharyngeal branches and lingual lymphoid aprons, can occasionally be seen *in situ* without transillumination.

The *number* of abscesses and abscessed crypts which may exist at any age or in association with any degree of infratonsillar lymphoid hypertrophy cannot, of course, be definitely estimated, for it varies with the degree of development of those tissues and, generally speaking, with the age of the subject and the number of inflammatory storms to which the throat has been subjected. Indeed, as in children they usually exist by the score and in adults often by the hundred, the tissues look as if they had been "peppered" with them.

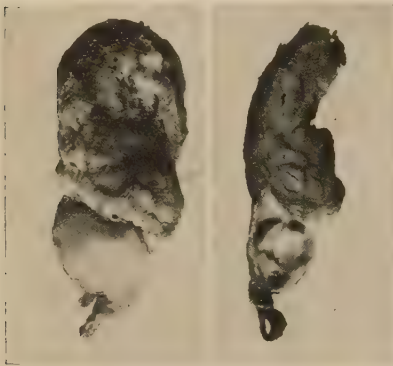


Fig. 144.

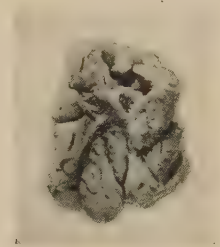


Fig. 145.

Fig. 144.—Front and side views of a tonsil with a cystic abscess attached. This type of abscess is quite frequently developed in the infratonsillar tissues. The specimen was enucleated with a blunt dissector from a man thirty-two years of age by Dr. Meyersburg. The ovoid pouch of pus was embedded in the trunk of the tonsil branches with only a thin shell of lymphoid tissue around it. When the dissector, descending behind the capsule, reached the trunk it forced the cyst through the thin lymphoid shell, thus leaving a part of the trunk and the entire pharyngeal branch to be removed immediately afterward. As shown in the second photograph the tonsil and cyst, attached by their continuous capsule, came out together.

Fig. 145.—A trunk of the tonsil branches from a man thirty-three years of age, showing many outlets of crypts.

After nearly three years of study of a very large number of sections of the infratonsillar nodules Dr. de Veer has reached the conclusion that in most subjects past the stage of childhood, and occasionally during childhood, those lymphoid bodies harbor as much pus and other broken-down tissue as do infected faucial tonsils. As the micro-organisms recovered from the necrotic material in the infratonsillar tissues are the same as those cultured from infected tonsils, it is clear enough that as factors of systemic infection the lymphoid structures in the lower pharynx are quite as potent as infected faucial tonsils. In the matter of bulk alone we find that in youth and early adult life the infratonsillar nodules are apt to represent a quantitative equivalent of the faucial tonsils, but in middle life and old age when the faucial tonsils are shrinking from atrophy and the infratonsillar nodules are increasing in size, a conservative estimate usually

gauges the relative measurements as at least three to one in favor of the nodules.

Of the bacteria which flourish in the necrotic material recovered from the infratonsillar nodules Dr. de Veer has isolated the *Streptococcus hemolyticus*, which predominates and is often found in pure culture, the *S. viridans*, the pneumococcus, the staphylococci, fusiform spirilla, diphtheroids and the *Micrococcus catarrhalis*. The presence of large numbers of such organisms in chronic states together with the occurrence of cryptic exudates on the surfaces of all of these lymphoid tissues during attacks of acute faucial inflammation will, doubtless, be regarded as sufficient evidence of the competency of the harbored necrotic material in the crypts and abscess cavities of the infratonsillar structures to alone act, in the absence of immunity, as a fertile source of systemic infection.

As the character of the necrotic material and the micro-organisms in the cryptic and abscess cavities of the infratonsillar structures is essentially the same as that in infected faucial tonsils, the infratonsillar lymphoid bodies as menaces to health through infection must be regarded as counterparts of the faucial tonsils and should, therefore, receive equal consideration with them, for in all respects they too are a pair of tonsils—a pair of tonsils which, unlike the faucial tonsils, are nearly always potential sources of systemic infection, for when they are developed enough to appear as entities they almost invariably contain necrotic material capable of producing infection, while the faucial tonsils may be, and not infrequently are, quite free from such necrotic material. Manifestly, this means that the infratonsillar tissues when developed are practically always potential sources of infection while the faucial tonsils very frequently are not.

We believe we have been able to prove that the virulent bacteria in the infratonsillar tissues are in a continual state of activation and, therefore, constitute a continually active obstacle to the attainment of full health. This proof was obtained in the past seven or eight years during which we employed the conservative surgical method of curetage to evacuate the contents of the cavities in the tonsil branches and lymphoid aprons on the bases of tongues in scores of adult patients, mainly those who had been tonsillectomized. The curetage procedure was carried out through the years for the purpose of making a clinical test to determine, if possible, in what ways the apparently dormant bacteria in the infratonsillar structures are detrimental to the health of the host. While for the most part this procedure was employed for the relief of symptoms which had not been dissipated by tonsillectomy, it also was employed very extensively for the sole purpose of elevating the plane of health of adults who were in a sub-normal condition, that is, they complained of symptoms which we believe have not been generally associated with focal infection—they were weary, “not feeling up to the mark” in body or mind and, as a consequence, were inclined to indolence. A review of the effects of curetage of the infratonsillar bodies in this large class of subjects brings into sharp relief the impressive fact that in every case, whatever the original objective, the removal of a considerable quantity of necrotic material from those tissues has been followed by pronounced physical and, usually, mental improvement, and that the elimination, in many seances, of large quantities of such material has almost invariably been followed by great refreshment of the body, mind, and spirits.

We are now satisfied that the clinical test by curetage has been made in a sufficient number of subjects to prove that the bacteria harbored in all the infratonsillar lymphoid structures, which, seemingly, are only occasionally active, are, in reality, in a state of continual activation, exciting and maintaining at all times a mild systemic poisoning, or toxemia, which finds expression in a lowered level of both physical and mental health and vigor. We believe this statement applies to a very large majority of the members of the human family in every decade of life past that of infancy, but especially to those in middle life and old age.

In middle life and old age the atrophic shrinkage of the various lymphoid structures in the throat is not uniform in degree. The infratonsillar masses in the laryngopharynx are often, perhaps usually, highly developed when the faucial tonsils are in a state of retrogression and are either very small or have almost completely disappeared. In other words, paradoxical as it may seem, in middle life and old age the lymphoid tissues in the lower pharynx increase in size as the faucial tonsils shrink. The relative sizes of a faucial tonsil, trunk of the branches and pharyngeal branch in middle life are fairly well illustrated in the fully expanded tonsil shown in Fig. 161.

Phrased in the form of a figure of speech, the alignment of the dusky-gray areas and rosy zones marking the sites of abscesses and abscessed crypts in the infratonsillar lymphoid bodies, quite naturally suggests an internal necklace of varying sized pearls and rubies, for their lines of arrangement are swung downward from one faucial tonsil across the throat under the lower edge of the lymphoid apron on the base of the tongue and then upward to the faucial tonsil on the opposite side, with a perpendicular pendant hanging from each tonsil, and a broad sunburst of the same brilliance in the lingual tonsil, and lingual lymphoid apron suspended in the center of the necklace.

The thick masses of lymphoid tissue below and between the faucial tonsils, which as possible generators of systemic disorders have always been devoid of significance, have, through the exploitation of their stromas, been disclosed as wide fields of thickly planted abscesses, like internal acnes, which, of necessity, must be regarded as potential sources, indeed, we believe, as potentially fruitful sources of systemic infection. The revealed nature of the necrotic contents of those tissues will now, doubtless, supply the reason for the belief entertained by internists that their patients suffering from a variety of disorders which, apparently, were due to infection, have shown greater improvement after expanded tonsillectomy than had others, with similar symptoms, after the enucleation of the faucial tonsils only. This brings into relief the automatically established fact that the large numbers of potentially infectious foci within the lymphoid addenda attached to and closely associated with the faucial tonsils have, of necessity, converted the operation now in vogue of enucleating the faucial tonsils only into an incomplete tonsillectomy or, as ultimately we believe it will be regarded as most correct to say, a tonsillotomY.

**Adenoid Growths.**—The finding of necrotic collections in the infratonsillar lymphoid tissues naturally led to the suggestion that the so-called "adenoid" growths in the vault of the pharynx must be similarly affected. A recent tonsilloscopic study of a large number of those growths after removal has proved this to be a fact. The abscessed crypts and small abscesses in those tissues are, however, neither as large nor as numerous

as those in the lymphoid structures in the laryngopharynx but, nevertheless, the small collections of pus are sprinkled quite liberally over the surfaces of the adenoid masses. The relative sparsity of abscessed crypts in those tissues is probably due to the gravity-drainage of the crypts because of the suspension of adenoid growths from the arched roof of the pharynx. An important contribution to this subject was recently made by I. D. Kelley, Jr.,<sup>4</sup> who graphically demonstrated the occasional occurrence of large abscesses deep in the stroma of those structures.

The two most pronounced reasons for the removal of adenoid growths have been obstruction to nasal respiration and chronic inflammatory disorders of neighboring tissues which remain as legacies of acute inflammatory storms to which these so-called "adenoid" but really *lymphoid* structures are peculiarly liable. The recent pathological findings have, however, made it clearly apparent that the necrotic contents of these growths must necessarily be more or less frequent sources of infection and, therefore, the finding of pus in the masses of lymphoid tissue in the nasopharynx adds another reason for operative interference.

#### THE TONSILLOSCOPE AND THE ART OF TONSILLOSCOPY

The **tonsilloscope** is the culminating product of a series of attempts to improve our means of making diagnoses inside of tonsillar tissues. It consists of a small lamp for transilluminating such tissues while lying on their beds in the throat, and a simple microscope for enlarging and clarifying the details in the substance of the lymphoid structures thus filled with light.

**Tonsilloscopy** is the method of examining the interiors of the tonsils and their branches as well as the lymphoid apron on the base of the tongue, while those tissues are in a luminous state from a hidden source of light. That is, the small lamp is embedded out of sight within the folds above, behind, or below the tonsils, and behind either of the trunks or branches, and also deeply into the lymphoid blanket on the base of the tongue, and while their stromas are in a luminous and somewhat translucent state they are examined with the tonsil microscope. The shade, or value, of the color revealed by the transilluminating lamp alone indicates the class to which the lymphoid tissues belong, but in the field of the microscope the details of the conditions within the tissues are often graphically portrayed.

The **tonsil lamp** (Fig. 146), of one-watt power, is enclosed in a small metal case with a glass window at or near its distal extremity. The lamp case is attached at an obtuse angle to an electric light shank which connects by cable with a dry battery balanced in power to that of the lamp. When the tonsils are large they can, of course, be most effectively transilluminated with a double lamp (Fig. 146), but the illumination produced by the single lamp is usually powerful enough for routine diagnostic work.

The **tonsil microscope** (Fig. 147) is a slender speculum about 6 inches long, inside of which, at the end of a telescopic tube, is a lens varying from 5 to 8 diopters according to the visual needs of the examiner. The distal end of the microscope is beveled for its close application to the free faces of the faucial tonsils, as well as to the free faces of the trunks, branches, and lingual lymphoid aprons. If irritability of the fauces makes it difficult to use even the lamp the microscope must, of course, be dispensed with and the diagnosis then be quickly made from the color findings of the lamp alone. Happily, with the lamp alone it can at once be determined whether

a tonsil and its associated lymphoid tissues are those of health or are the seats of infection, and if they are the seats of infection whether pus as well as detritus is present.

An evenly diffused transillumination of any or all of the lymphoid tissues can be instantly secured by placing the cool, but bright, lamp behind, below, or above the tonsil, or behind a trunk or branch, or by sinking it into the lingual lymphoid apron, and then pressing it into the tissues. The whole of a tonsil, or a part of the infratonsillar structures, will then light up as does a stained-glass window with sunlight shining through it.



Fig. 3.

Fig. 146.—Single and double tonsil lamps.

The outlet of the tonsil microscope is then applied to any and every surface of the luminous tissues not occupied by the lamp, including even parts of the capsules of the faucial tonsils. In this way many of the conditions of health or disease are clearly disclosed which are not revealed by the lamp alone.

When the lymphoid tissues are in a state of health, or nearly so, they are relatively translucent and permit a considerable insight into their contents. When, however, they contain collections of necrotic material, they are less translucent in proportion to the density of the hyperemia which is produced by irritation from those collections, so that in extensive involvement of the tissues by infectious material it is usually impossible,

even with the microscope, to definitely detect the outlines of anything of a foreign nature other than collections of pus lying upon or close to the surface. This fact is, however, of little consequence, for we know that the degree and character of the hyperemic displays produced by the necrotic collections indicate, in a general way, their number and it may be the approximate locations of some of the collections themselves.

**The External Tonsilloscope.**—The all-essential prerequisite of a successful tonsilloscopy is the ability to interpret the signs of health and disease which exist within lymphoid tissues when disclosed by transillumination. The color code of those tissues, while their interiors are lighted



Fig. 147.

Fig. 147.—Tonsil microscope.



Fig. 148.—The external tonsilloscope for the study of excised lymphoid tissues.

up, is a language of signs, and to one who learns to interpret those signs the interiors of the tonsils, their branches, the lingual tonsil and the lingual lymphoid apron become, practically, as an open book. The truth of the principles upon which this diagnostic method is based can be tested and at the same time a knowledge of the art can be acquired, by the study of excised pharyngeal lymphoid tissues in the external tonsilloscope, for that contrivance, as shown in Fig. 148, involves the principles of the tonsilloscope, that is, it consists of a lamp and a simple microscope.

With this device the lamp and microscope can easily be assembled by placing a magnifying glass in the metal frame of a tripod stand upon the flat glass top of a 6½-inch *Eveready flashlight*. The tripod and flashlight are commercial articles which are sold everywhere. A piece of blackened cardboard with an elliptical opening  $\frac{3}{4}$  inch long and  $\frac{3}{16}$  inch wide in the

center, fitted underneath the glass top of the flashlight, will exclude most of the light. Small perforated corks glued to the glass top to act as foot-holds for two of the legs of the lens-tripod add greatly to the efficiency of this instrument.

The fresh specimen is laid over the elliptical opening, capsule down, and changed in position from time to time, by sliding it around on the glass top, to display its various segments and pathological features. The light is then turned on and maintained. If too much light leaks out around the edges of the specimen it should be excluded with strips of black paper or cardboard. In this way an excellent opportunity is afforded to study the abscess formations in the tonsil branches, lingual tonsils, and lymphoid aprons on the bases of tongues as they appear by transillumination, as well as by surface light, in the living tissues.

The tonsils proper retain most of their transillumination color characteristics for a time after removal. This also is true of the infratonsillar lymphoid tissues when they are only partially developed and, therefore, small, but it is not true of them when they are well developed and large, for then they show in an almost uniformly bright-red-rose color *while in the throat*, but in spotted red and amber when laid over the flashlight immediately after enucleation. This difference is, of course, the result of the draining of the dilated blood channels by the undercutting of the tissues. During transillumination uninfected lymphoid tissues show in a light shade of amber. The rosy, or hyperemic, coloring is the result of irritation from pus in abscesses and abscessed crypts. The rosy coloring during transillumination *in situ* can, especially in the young, be largely dissipated temporarily by the application of a 1 : 5000 solution of adrenaline chloride to the pharyngeal branches and base of the tongue. The blanched tissues then show again in mottled red and amber, thus often disclosing the locations of the areas of necrosis. Studies with the external tonsilloscope will enable the student to prepare himself to instantly recognize and differentiate the various conditions during the necessarily rapid transillumination view of the faucial tonsils, their branches, the lingual tonsil, and lingual lymphoid apron as they lie upon their beds in the pharynx. As the tonsil of health ought never to be excised it is to be hoped that the student of this subject will have few, if any, opportunities to study that beautiful anatomical structure in the external tonsilloscope but, fortunately, only a very little practice with the tonsilloscope is needed to learn how to recognize the absolutely normal tonsil *in situ*.

The tonsilloscope has differentiated a variety of conditions in the tonsils which elsewhere<sup>2</sup> have been described in six classes, but in order to simplify this diagnostic scheme it is thought best to group all of the varieties under three general heads, namely: (1) The tonsil of health; (2) the tonsil and small infratonsillar nodules containing a few crypts filled with detritus only, a few scattered abscessed crypts and, possibly, a few small abscesses; and (3) the tonsil containing quantities of detritus and pus, with fully developed infratonsillar nodules in which most of the crypts are the seats of abscesses, a few of them only containing detritus without pus and, also, numerous extracryptic abscesses varying in size from the dimensions of the head of a small pin to encysted collections of pus  $\frac{1}{2}$  inch in diameter. This simplified classification and an epitomized description of the tonsil color code will be found in the frontispiece folder of this chapter.

In this connection it may be helpful to note the fact that the contents of the crypts of the infratonsillar structures which consist of detritus only, transmit light and, therefore, do not show during transillumination. When, however, as is usually the case, the crypts contain pus as well as detritus, they then appear as bright-rosy-zones the same as small independent abscesses, for small collections of pus are always surrounded by zones of hyperemia, and only the zones of color show. Pus, however, does not transmit light. This is illustrated when large cystic abscesses on or near the surface are revealed by transillumination, for they appear as dusky gray areas. By surface light, especially after adrenaline has been applied, the large abscesses can often be seen through the mucosa as yellowish, or grayish-white spots.

The tonsilloscope may be likened to a divining rod which instantly detects in any and every part of the elaborate fields of lymphoid tissue in the pharynx the hidden presence of the pathogene, pus.

It, doubtless, will be of interest to note, in passing, that the lymphoid masses commonly spoken of as *recurrent tonsils* are really nothing more than the superior extremities of the tonsil branches—that is, the trunks, for even after the faucial tonsils have been perfectly enucleated these large segments of the branches may, in the course of time, work their way upward into the empty fossæ which formerly were occupied by the tonsils. In such dislocated anatomical positions they not infrequently masquerade most effectively and successfully, more especially during attacks of acute cryptic inflammation, as parts or all of the original tonsils. Naturally enough, such out-of-position structures are, at times, mistaken for remnants of the faucial tonsils which escaped the attention of the surgeon who performed the tonsillectomy. To the uninitiated such apparent post-operative defects may appear to reflect discredit upon the operator's skill and care, and prompt unjustified criticism.

#### THE ELIMINATION OF CHRONIC ABSCESES AND ABSCESED CRYPTS FROM LINGUAL LYMPHOID APRONS, PHARYNGEAL BRANCHES, AND TRUNKS OF THE TONSIL BRANCHES

In subjects from whom the faucial tonsils only have been removed, the lingual lymphoid aprons, pharyngeal branches and trunks of the tonsil branches can be readily, and perhaps permanently, cleared of their infectious contents by curettage. In subjects who will not submit to or who



Fig. 149.—Single abscess-rupturing cone curet; in two sizes.

cannot be subjected to tonsillectomy, the extensive fields of abscesses in the aprons, pharyngeal branches, and trunks of the branches can be quite rapidly evacuated in office and bedside practice. The pharyngeal branches and trunks of the branches are the only infratonsillar structures which are removed with the tonsils in expanded tonsillectomy. The lingual lymphoid

apron rests upon a thin fibrous capsule, but its enucleation ought never to be attempted as that more or less thick pad of tissue is needed to shield the large vessels coursing close to its capsule, and also, probably, for the physiological functioning of its lymphoid tissue elements. For the dissipation of the necrotic contents of the lymphoid apron we must rely upon the art of curettage.

When the whole length of the tongue can be flattened out it is very easy to rupture the abscesses and abscessed crypts in the infratonsillar lymphoid structures with the abscess-rupturing cone curet illustrated in Fig. 149. A larger size than that is also made. If, however, the tongue cannot be flattened, then, the way may be lighted up and the movements of the cone be accurately guided by reflection in the laryngeal mirror. As shown in the figure the cone is truncated; it is a dull ring at the distal end and a cone and scoop above. When the ring is pressed into the lingual apron or into the pharyngeal branches or trunks it compresses and gently pinches the soft, yielding tissues, rupturing one, or several, of the abscesses at each introduction of the curet. The application of adrenaline chloride in rather strong solutions (1 : 2000–5000) together with a surface anesthetic facilitates the rupturing procedure.

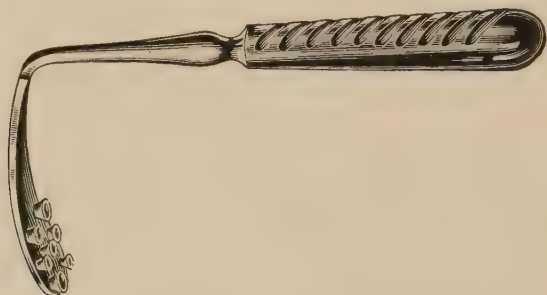


Fig. 150.—Multiple abscess-rupturing cone curet.

Firm pressure with the smallest cone will evacuate the abscesses and abscessed crypts and force the necrotic plugs and most of the contents of the cavities into the bell of the cone. The larger cone curet held at an angle of a little less than 45 degrees to the surface, to act as a scoop, is then swept, with a moderate amount of pressure, over the areas which were covered by the smaller cone to gather up the pus and necrotic debris which remain on the surface or in the crypts and abscess cavities after their rupture. In most cases, however, the contents of the infected crypts and abscess cavities, as well as the thin layer of necrotic material which accumulates upon and overspreads the surfaces of the branches and the apron on the base of the tongue, can be very effectively cleaned out and off with the larger cone curet when used as a scoop. The pus cavities in the pharyngeal branches and trunks can be emptied with the same instrument by sweeping it over the surfaces at an oblique angle from below upward.

Expeditious work in rupturing abscess cavities in the lymphoid apron on the base of the tongue can also be done with the multiple cone curet illustrated in Fig. 150. In each séance of treatment the six cones on the blade of this instrument should be pressed downward upon the surface of

the apron and rolled forward and backward a number of times over several sections of that area. Much of the liberated necrotic material will be recovered on the instrument, but the large single cone used as a scoop will gather up much more of the dislodged débris.

Close to the center of every convolution on the surface of all the infratonsillar lymphoid structures is a small opening leading into a crypt. The round mouths of such crypts are shown in Fig. 145. This variety of crypt usually penetrates to the capsule and nearly always becomes abscessed. Firm pressure with the smallest single cone curet accurately applied over the center of the eminence of a convolution will dilate or rupture the constricted cryptic mouth and force the contents of the distended cavity into the cone. As, however, the single cone is apt to slide off from the domes of convolutions it is usually best to employ the multiple cone curet, which, with one exception, has on its under surface the smallest sized cones, each of which, steadied by the others, is apt to remain wherever placed upon the surface of the apron until lifted off. Mainly to avoid injury to the epiglottis and large varicose veins it is advisable always to follow the movements of the distal ends of the cones either directly or with the aid of the mirror.

Cone curettage is quite effective in liberating the contents of infratonsillar abscesses but is somewhat limited in its usefulness. After an



Fig. 151.—The lingual plane. Insert shows size of end and shank. The reverse of this model is made for operating from patient's right to left.

added season's experience we find ourselves much more impressed with the expedition, thoroughness, and relative freedom from discomfort to the patient which can be obtained in this deterring work from the sweeping use of the lingual plane illustrated in Fig. 151. As shown in the figure, the plane is made in sets of two, the upper model for oblique sweeping of the base of the tongue from the patient's right to left and the lower model (reduced and actual size) for the same kind of use from left to right. These instruments are bent to fit over the saddle of the tongue and have beveled shanks as well as beveled hook-like ends with rounded backs. The wedge-like edges, below and above, are dull but not blunt. In a somewhat simpler form the plane was originally designed to shave the white coating from the dorsum of the tongue in preparation for expanded tonsillectomy. The principle in its construction is not unlike that of the snow-plow scraper. Drawing-pressure upon the surfaces of the infratonsillar lymphoid tissues with this instrument ruptures the abscesses and abscessed crypts directly

in front of the advancing beveled edge. As in cone curettage the movements of the operating end of the plane should always be seen directly or in the throat mirror.

While the patient is making traction upon the tongue with a gauze sponge the curved end of the plane is passed straight downward over the saddle of the tongue to the epiglottis in the median line, or to the lower limit of the lymphoid apron on either side. From those positions it is drawn obliquely upward over various areas of the base, with rather firm pressure upon the tissues, and when part way up it is drawn straight to, upon and over the dorsum of the tongue, sweeping before it, like a "foaming bone in the teeth," many cryptic plugs and much pus as well as a layer of necrotic white coating from the dorsum. The usually large collections heaped up upon the beveled shank and curved end of the instrument are then scraped off over the edge of a spoon held at the lips.

The abscess cavities in the pharyngeal branches and trunks can also be ruptured and emptied with the lingual planes. For the right branch and trunk the best results are obtained by placing the back of the curved end of the plane shown in the upper model in Fig. 151, against the surface of the branch, the upper edge being pointed obliquely upward and outward, and then sweeping it straight upward, with moderate pressure upon the tissues, from the free end of the branch below to the inferior pole of the tonsil above, or, if the subject has been tonsillectomized, to the fossa formerly occupied by the tonsil. For the left branch and trunk the upper edge of the curved end of the reverse plane is employed in the same way. Such sweeping movements should, if possible, be repeated several times at each séance. Expanded tonsillectomy calls for the enucleation of the trunks and pharyngeal branches together with the tonsils. If they are left in their beds after the removal of the tonsils and the patient is unwilling to submit to further enucleating procedures, then they should be emptied of their pus contents by plane curettage, for each of those structures may harbor large cystic abscesses (Fig. 144), and from fifty to a hundred smaller collections of pus.

The edges of the planes are made dull to minimize the chances of the occurrence of acute auto-intoxication through breaks in the mucosa. Sharp instruments ought never to be used upon the tongue except by skilful and painstaking operators, for the lingual blood-supply is so full and elaborate that the unskilful or careless use of sharp instruments upon that organ is more than likely to be followed by hemorrhage which may prove to be difficult to control.

After a number of septic cavities have been evacuated relationships which really exist between this source of infection and any given symptoms are occasionally, but not often, almost immediately announced either by a marked increase or a marked decrease of such symptoms. Unsuspected relationships with ailments, to the presence of which attention had not been called, have not infrequently been revealed as having their source in infected infratonsillar lymphoid tissues, by the noticeable improvement in or the disappearance of the symptoms of such ailments during courses of curettage treatments for the relief of more pronounced disorders. Finally, to obtain complete and lasting relief, even of conditions in which cause and effect have been definitely established, many séances of curettage may be required.

**Local and Systemic Reactions.**—The *pharyngeal reaction* which is apt to occur from the contact of instruments with the tissues of the laryngopharynx often makes it difficult, and sometimes impossible, even after the application of a surface anesthetic, to carry out any minor surgical procedure in that cavity. Indeed, for most cases, there are few manipulative technics in surgery which require greater delicacy of touch and more display of tact for its successful accomplishment than this. If, however, adrenaline can be employed, that is, if the patient is not oversensitive to it, its use will increase the ease and decrease the time in which most of the necrotic cavities can be emptied and, perhaps, obliterated.

The *systemic reactions* following lingual curettage are most pronounced after the removal of large quantities of necrotic material containing great numbers of the most virulent forms of micro-organisms, but occasionally they occur, with very unpleasant emphasis, in subjects whose cultures show only a few of the weaker forms of pathogenic bacteria. The character of reactions which occur most commonly in subjects suffering from myositis is an increase of pain for a few hours, or even days, which, usually, is confined to or is most pronounced in or about the seat of disturbance for which relief is sought. The pain of reaction may, however, extend into other parts or occur in other localities. In arthritic subjects the commonest forms of reaction are a sense of general discomfort and uneasiness for a day or two and usually an increase of pain in the affected part or parts. In the subjects of gastro-intestinal disorders the reactions may take the form of nausea or even vomiting, with, perhaps, a brief attack of diarrhea accompanied by cramps. But in the majority of subjects with a variety of disorders, reactions either have not occurred at all or have taken the form of slight malaise in the night and on the day following a séance of this method of treatment.

While it may not be possible to prevent the occurrence of reactions after curettage they can often be ameliorated or avoided by taking pains to prevent injury to the epithelial layer, through guidance of the movements of the plane by direct vision or in the mirror, or by removing a smaller quantity of the necrotic material, especially in the early séances, or by making the interval between treatments two weeks instead of a week, or by all of those measures combined. The testimony of a number of intelligent and observant patients in whom severe early reactions had occurred was to the effect that their severity progressively declined, and in some cases disappeared during the course of the treatment.

**Systemic Infections.**—The wide prevalence of the presence of large numbers of abscesses in the lymphoid structures under consideration must, of necessity, be responsible for many of the systemic infections to which the human body is liable. A concerted clinical investigation is, of course, necessary to determine their relationships. Meanwhile, however, we venture to record a few of our own observations upon the results of the employment of the abscess-evacuating method of treatment which, in brief, are that in addition to the improvements in physical and mental health which, as noted earlier in this writing, almost invariably follow the disposal of the lingual abscesses, our experiences have proved that those tissues hold an especially intimate causal relationship with disorders of the digestive tract, as well as with chronic infectious polyarthritis, and are, also, contributive factors in, or, in some cases, the sole reasons for, the

occurrence of myositis, spasmodic obstruction to micturition, vasomotor rhinitis, inflammatory new-growths, reflex cough, enlarged thyroid glands, double chins, some of the simpler forms of skin diseases, varying degrees of blood-pressure, and arrhythmia. The above list of disorders can serve only as suggestive pointers, for our time has been too much occupied in an effort to ascertain the facts regarding those tissues and also in finding methods for ridding them of their infectious contents, to apply the curettage test for the relief of other maladies than those which fell incidentally under our observation and care during the course of this investigation.

In the subjects of the infectious form of polyarthritis whose faucial tonsils had been enucleated without giving relief to the accompanying systemic symptoms, such strikingly beneficial results have followed the removal of the pharyngeal branches and curettage of the lymphoid aprons on the bases of tongues that there is now no doubt that the necrotic material in the infratonsillar lymphoid tissues is, at least in a large percentage of cases in the infectious class of the disease, either partly or entirely responsible for that painful and disabling affection. In a number of cases of acute exacerbations of infectious arthritis with intense pain the removal of even small quantities of the necrotic contents of those tissues has acted like magic in giving almost instant relief to the pain for long periods of time. Since observing the beneficial effects of lingual curettage in the first few cases of chronic infectious arthritis tried out two years ago, we have given particular attention to the treatment by curettage in many cases in this class. The pleasingly surprising results thus far obtained, the details of which are beyond the limits of our space to publish here, convey the brightest promise that if the abscess-rupturing or eliminating treatment is begun in the early stages of infectious arthritis, this form of the distressing disorder is more than likely to fall into the province of easily controllable systemic affections. A number of subjects in this class in whom the disease had existed from three to five years—in one of the group locking of the jaw having begun—have, after from 15 to 25 treatments, entirely recovered and returned to the enjoyment of full health.

Judson Daland,<sup>5</sup> after presenting a surprisingly long list of systemic infections produced by foci in the tonsils and accessory sinuses, says: "This condensed summary resembles the table of contents of a text-book on the practice of medicine." We know of no other statement by an internist of high standing as an observer as Professor Daland which so clearly portrays the far-reaching extent of the etiological relationship of chronic infection of the tonsils and sinuses to systemic diseases.

The continuous development and retention of varying quantities of potentially infectious material in the lymphoid structures below and between the faucial tonsils in practically every human being beyond infancy, must, for at least a very long period of time, have caused and maintained a lowered level of the health of the race. Observant clinicians with a knowledge of the almost invariable presence of such material in those structures will ultimately, no doubt, determine the nature and extent of the relationships which exist between systemic disorders and this hitherto hidden source of physical mischief.

In this connection we wish again to direct attention to a fact, before alluded to, which may bear upon the source of some of the degenerative diseases of middle life as well as the decay of old age. The suggestive fact

referred to, and to which thought should be addressed with the view of divining its significance, is that the infratonsillar nodules planted as they are with large numbers of floating mines of pus and necrotic material containing the virulent varieties of bacteria, usually increase in size and in their necrotic contents as the faucial tonsils and all other lymphoid tissues in the throat undergo atrophic shrinkage as age advances. The specimen reproduced in Fig. 161 is, in part, an interpretation of the fact we wish to emphasize. The infratonsillar masses which develop with the advancing years have, apparently, an ever-increasing pathological basis, for if they obeyed the laws which apply to all other lymphoid aggregations, retrogression would begin in them in early adult life; instead, they increase in size and in their necrotic contents while the other tissues of the same anatomical construction, but located elsewhere in the throat, gradually fade away.

#### EXPANDED TONSILLECTOMY

The method of enucleating the faucial tonsils together with the pharyngeal branches and trunks is known as expanded tonsillectomy.<sup>6</sup> An outline of the technics of the operative procedures is essential in this section.

Developed tonsil branches and trunks are always the seats of potential pathogenes, and as those structures are invariably more or less developed when the faucial tonsils proper are infected, it follows, logically, that tonsillectomies of the future must be made more expansive than they are at present by including the pharyngeal branches and the trunks of the branches in all tonsil enucleations. The pharyngeal branches and trunks lie upon the surfaces of the lateral walls of the laryngopharynx and can be readily removed by inserting a wedge under their capsules, sliding them off of their attachments to the underlying basement membrane and then detaching them from the overlying mucous membrane. From the basement membrane they are quite separate and distinct.

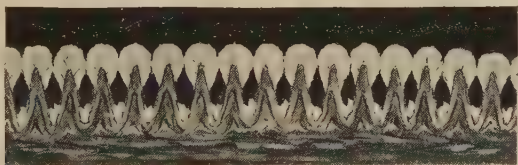


Fig. 152.—Diagrammatic drawing of a vertical section of a part of a row of filiform papillae on dorsum of tongue. It shows entangled tufts of necrotic tissue on the tips of papillae and entrapped masses of the same material around their bases.

**Preparation of the Tongue and Throat for Expanded Tonsillectomy.**—Bacteriological studies of the mouth and throat show them to be the most heavily germ-laden cavities of the body. The general surgeon would refuse to break through the skin anywhere if he knew it was covered with a marsh of necrotic material at all like that of the *white coating of the tongue*,<sup>7</sup> without first removing that material; and he would be even more reluctant to break through the skin if he knew that it was elaborately seeded with easily ruptured abscesses such as those which are embedded in the infratonsillar lymphoid tissues, until he had made an effort to rid it of its contents of pus.

Spread out upon the dorsum, and what might be called the saddle, of the tongue in, probably, every human being, there is a more or less thick layer of cheesy detritus made up of a thousand or more small tufts of ne-

crotic material caught upon rows of cactus-like, or *filiform*, papillæ, very much as cotton is wrapped around the ends of applicators. The detritus-crowned papillæ are diagrammatically illustrated in Fig. 152. Much of the lowermost stratum of the necrotic material which can be seen in that figure between the bases of the papillæ and below the tufts, as well as the tufts themselves, probably remain practically unchanged for a long period of time, and both the upper and lower layers of the coating are found to be rich in pathogenic micro-organisms—mainly the *Staphylococci*.

Convincing evidence of the deleterious effect of the white coating upon the surfaces bared by tonsillectomies has been abundantly presented by the improved character of the healing processes in subjects from whose tongues the "fur" had been repeatedly shaved with the lingual plane in preparation for radical operations. Healing in such cases was relatively rapid and even after the expanded operations we believed there was less soreness of the throat than heretofore had been complained of. The best results were obtained in subjects whose tongues had been thoroughly shaved six or eight times and whose mouths had been rinsed with a 50 per cent. solution of alcohol a number of times immediately before operation.

In the light of recent revelations, however, it is now clearly indicated that in addition to shaving the white coating from the dorsum of the tongue in preparation for expanded tonsillectomy the necrotic cavities in the pus-honeycombed lymphoid tissues on the base of the tongue in adolescents and adults, and even in some children within the age of reason, should, as far as possible, be evacuated prior to the date of the radical operation. The methods by which such evacuations can best be accomplished have been described in a previous section.

To aid in preventing infection of the cut surfaces after enucleation the shaving of the dorsum of the tongue and the evacuation of the necrotic cavities in the lymphoid tissues on the base of the tongue should be carried out before rather than at the time of, or after, the tonsillectomy. And we have learned that there is still another reason for clearing the tongue before the radical operation, which is, that if the then existing symptoms believed to be due to infection are not relieved by expanded tonsillectomy the patient, or parent, is usually unwilling to allow any further surgical interference by the same operator.

**Instruments for Expanded Tonsillectomy with the Tonsillectome.**—The instruments which are especially needed for expanded tonsillectomy by the tonsillectome method are: (1) A tonsillectome, so shaped that its distal bar can be readily swept under the capsules of the pharyngeal branches, trunks of the branches and faucial tonsils, and (2) block-molds, one for each side, for the outside support of the soft tissues of the neck below the jaw while the pharyngeal branches and trunks are being underswept.

The *tonsillectome* (Fig. 153) has an aperture which is practically square. Its very slightly bowed distal bar is wedge-shaped which insures its ready entrance under the capsules of the pharyngeal branches and trunks. The side bars are straight on the outside and slanting on the inside—that is, wedge-shaped also. With the exception of the features just noted, and also a supplementary push to aid the hand to thrust the blade so that during the operation the hold of the distal bar upon the dislocated trunk and branch will not be lost, this instrument is the same as the Sluder guillotine.

The *block-molds* (Fig. 154) are made of hard rubber, with soft-rubber

shelves to fit under the edges of the jaws of subjects of any age. The bodies of the blocks are so molded that the shelves will readily catch and cling to the inferior edges and the angles of the jaw.

**Technic of the One-piece Expanded Tonsillectomy with the Tonsillec-tome.**—The one-piece operation not only saves time, but it also enables the

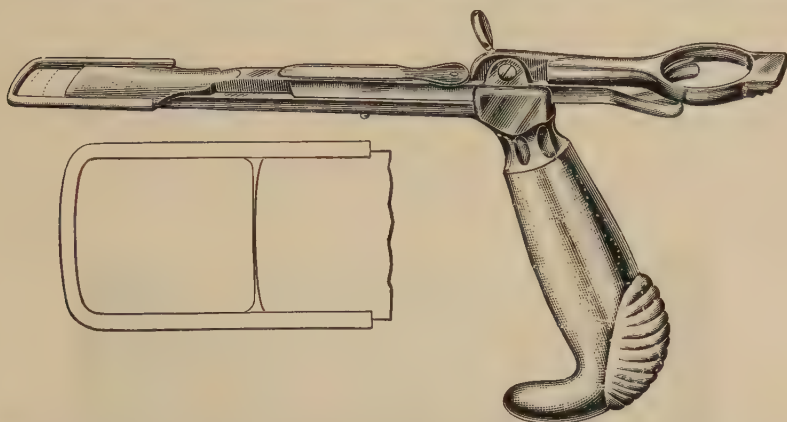


Fig. 153.—Tonsillec-tome for expanded tonsillectomy. Insert shows actual size and shape of aperture.

operator to determine in a glance at the specimens whether every vestige of the faucial tonsils and attached trunks and pharyngeal branches has been removed.

The one-piece procedure is carried out as follows: An assistant, usually the anesthetist or nurse, adjusts a block-mold around the angle and under the lower edge of the jaw on the side to be operated upon and, guided by



Fig. 154.—Block-molds for outside support of neck in expanded tonsillectomy.

the operator, holds it firmly and steadily in position. The tonsillec-tome is then passed into the mouth over the saddle of the tongue and downward into the laryngopharynx opposite the block-mold being held on the outside. The distal bar of the instrument is laid flat on the lateral wall of the pharynx and held so in its downward passage until it meets with the resistance of the lateral pharyngo-epiglottic fold. Figure 155 shows the

position of the tonsillectome upon its introduction into the throat as well as the grip of the hand upon the instrument; it also illustrates the manner in which a block-mold is adjusted and held in place.

After the distal bar reaches the lateral pharyngo-epiglottic fold the handle of the tonsillectome is tilted backward and upward until it is slanted to about 5 degrees above the horizontal axis of the head. The slant of the flat shaft, the flat part being at a right angle to the handle, is also shown in Fig. 155. With the instrument in that position the distal bar is pressed outward against the block-supported wall, and *while maintaining firm pressure* against the tissues supported by the block-mold, and later by the jaw-bone, the bar is swept directly upward in a straight line to and against the inferior pole of the tonsil. The fact that the aperture of the tonsillectome is nearly square should be kept in mind in making the sweep,

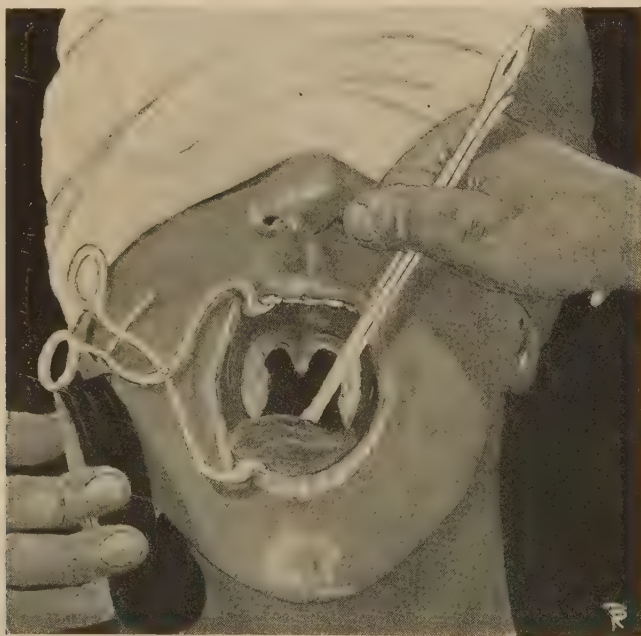


Fig. 155.—Shows position and direction of tonsillectome in the first step of expanded tonsillectomy. It also shows the manner in which the block-mold is held under the edge of the jaw and against the side of the neck.

for it is essential that the distal bar shall lie flat upon the side wall of the throat as the instrument ascends. All that is necessary to carry out this mechanical feature is to rotate the hand slightly forward during the upward sweep—that is, turn the handle backward and upward. When with this movement the distal bar has reached the inferior pole of the tonsil, the shaft should lie in the corner of the opposite side of the mouth, with its flat top at an angle of about 45 degrees to the horizontal axis of the head.

After repeating this movement once or twice it will be found that the trunk and pharyngeal branch have been dislocated and are freely movable under the touch of the forefinger. They are then in a perfectly prepared condition for their enucleation together with the tonsil. The dislocation of

the trunk and branch having been thus accomplished, the tonsillectome should again be passed downward into the throat in exactly the same manner as has just been described for the dislocation movement, and when the distal bar reaches the lateral pharyngo-epiglottic fold it should be pressed firmly outward against the block-supported wall, and then swept straight upward under the branch, trunk, and tonsil, high into the dome of the tonsillar fossa, high enough, indeed, to put the palatine tissues on the stretch. Meanwhile, however, the forefinger of the disengaged hand is introduced into the throat to press the tonsil downward and backward to assist in engaging the inferior pole in the aperture. The upper corner of the aperture having now reached the upper level of the tonsil, the distal bar being pressed firmly against the bone to maintain its hold upon the dislocated trunk and branch, the uppermost side bar is rotated forward so that the aperture will be carried over the top of the tonsil, while the forefinger



Fig. 156.—Capsule surface of a one-piece expanded tonsil removed from a girl eight years of age with the tonsillectome as described in this chapter. Photograph shows the continuity of the capsule underlying the faucial tonsil and the associated infratonsillar lymphoid development, which at this subject's age is about the size usually found.



Fig. 157.—Lateral view of a somewhat macerated one-piece expanded tonsil removed from a boy eleven years of age with the tonsillectome as described in this chapter. Photograph retouched by the author to accentuate the junction of the one-piece capsule with the lymphoid tissue of the faucial tonsil and the infratonsillar lymphoid structure.

manipulates the tonsillar mass, lying under the anterior pillar, wholly through the opening. When the distal bar, or side bars of the aperture, are felt through the anterior pillar, then, in order to insure the best results in faucial cosmetics, the pillar itself should be drawn forward, either with a broad retractor, or, if the lower incisors are not too sharp, by drawing the tongue well out of the mouth, while the edge of the blade is thrust between the edge of the pillar and the tonsil. The forefinger then follows, through the pillar, the edge of the blade until all of the tonsillar tissues are felt to have been pressed beneath it. The thumb lever on the shaft of the tonsillectome is then raised until moderate hemostatic pressure is applied, when, after a half minute or so, still greater pressure is made upon the thumb lever to sever the constricted tissues. During the whole of the procedure, from the time the distal bar is swept under the free end of

the pharyngeal branch until the blade is closed, firm pressure of the distal bar against the soft tissues supported outside of the neck by the block-mold, and also by the jaw-bone, must be evenly maintained.

The above description may make this method of operating seem elaborate and difficult. If so, the writing must be lacking in clarity, for in reality the evenly successful carrying-out of this technic is simplicity itself.

**The Two-piece Enucleation of the Faucial Tonsils, Trunks, and Pharyngeal Branches.**—Experienced surgeons who have acquired skill and easy usages by years of drill in tonsil work will, no doubt, prefer to remove the faucial tonsils in their own way and enucleate the trunks and pharyngeal branches separately. In that case, the tonsils should be removed first and



Fig. 158.—Photograph (somewhat enlarged) of tonsil, trunk, pharyngeal branch, and severed base of lingual branch (marked *L.B.*) enucleated in two pieces with a Demorest-Sluder guillotine and the block-molds, by Dr. G. R. Hageman. Patient was a boy fourteen years of age.

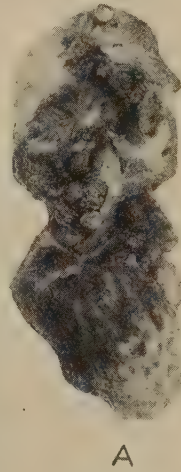


Fig. 159.—Faucial tonsil and large trunk of the branches removed from a woman in middle life, by the dissection-snare method, in a two-piece operation by Dr. Charles W. Stickle, Director of Otolaryngology in the Long Island College Hospital.

the trunks and pharyngeal branches afterward. Immediately after the tonsils have been enucleated the trunks and pharyngeal branches should be dislocated, as described under the head of the one-piece operation, and then swept upward with the distal bar of the tonsillec-tome into the then tonsillar fossæ and against the anterior pillars, through which the forefinger manipulates them into the grasp of the blade of the tonsillec-tome.

Even though the knowledge of the practically universal presence of potentially infectious material in these regions and also the knowledge that all of these lymphoid structures possess underlying capsules, prompts an almost irresistible desire to resort to radical measures, that prompting must be restrained, for it cannot yet be regarded as justifiable to strip the thick protective layer of lymphoid tissue from the base of the tongue. As the abscesses and many of the abscessed crypts are, however, on or near the surface of the blanket structure, it is feasible, if the varicose veins coursing on the surface are neither large nor numerous, to undercut and remove many of the independent collections of pus, mouths of crypts, and even some of the abscessed crypts in their entirety, by shaving or mowing off the tops of these tissues with the tonsillectome. Theoretically, such a method of eliminating the necrotic collections on the base of the tongue during a tonsillectomy may seem ideal, but as infection would be invited by liberating pus to flood freshly denuded surfaces, the topping operation should be done at least a week before the expanded



Fig. 160.

Fig. 160.—One-piece partially expanded tonsil of adult enucleated under local anesthesia by the dissection-snare method, by Dr. Harry Meyersburg.



Fig. 161.

Fig. 161.—Photograph from Dr. Meyersburg's collection of a fully expanded one-piece tonsil which was enucleated from a man thirty-five years of age with the dull dissector, but *without the snare*. This is the type of expanded tonsil now being removed from adults under either local or general anesthesia with the dull dissector or tonsillectome. The upper third of this long column of tonsillar tissue is a retrogressive faucial tonsil; the middle third is a well-developed trunk of the branches; the lower third is a well-developed pharyngeal branch. Attached end of lingual branch, severed by dull dissector from the long segment on tongue, can be seen curving downward to the right from the trunk.

tonsillectomy. For the present, however, we believe the wisest plan is to dispose of the necrotic collections by the "office method" at the time the "fur" is shaved away from the dorsum of the tongue in preparation for expanded tonsillectomy.

**Expanded Tonsillectomy Under Local Anesthesia.**—One-piece expanded tonsillectomy under local anesthesia can be easily and painlessly performed in adults with the tonsillectome or by dissection. That this method of operating is being extensively practised is sufficiently well shown by the work of a single operator, Dr. Harry Meyersburg, one of our associates in

this research study, who in the past four years has performed by the dissection-snare method under local anesthesia nearly 2000 partially expanded tonsillectomies. Figure 160 is a photograph of a specimen from his collection of partially expanded tonsils—meaning, the tonsils and trunks, but not the pharyngeal branches—which had been removed by dissection and the snare under local anesthesia.

The snare, however, has no place in the dissection method for expanded tonsillectomy. Its use always means an incomplete operation in that it leaves the pharyngeal branches on their beds in the throat. The whole of the pharyngeal branches should invariably be removed as at all ages they contain many abscesses and abscessed crypts and, occasionally even in children, large cystic abscesses similar to that in the specimen displayed in Fig. 144. After passing behind the capsule of the faucial tonsil with the blunt dissector, that instrument should be carried from above downward, outside of the continuous capsules of the tonsil, trunk, and pharyngeal branch, until, by following the line of aponeurotic cleavage, it automatically emerges from under the free end of the branch. The whole mass, consisting of the tonsil, trunk, and pharyngeal branch, is then lifted out as an entity exactly as a piece is lifted out of its fitted place in a picture puzzle. In accordance with our suggestion Dr. Meyersburg has discarded the snare in this operation and now enucleates the tonsil, trunk, and pharyngeal branch with the dull dissector in one uncurtailed piece. Figure 161 is a photograph from his collection showing an example of the fully expanded one-piece tonsil specimens as they appear after removal by dull dissection alone.

**The After-effects of Expanded Tonsillectomy.**—Eight years of experience with expanded tonsillectomy—the operation meanwhile having been performed by many operators, in many hospitals, thousands of times in the Long Island College Hospital alone—has shown it to be devoid of added dangers and complications. Hemorrhage has not been noticeably increased, and in the subjects from whose tongues the white coating on the dorsum and the abscesses and abscessed crypts in the lingual lymphoid apron had been well disposed of prior to the radical operation, the expansion of denuded surfaces has neither delayed healing nor added to local discomfort during the process of active repair. Indeed, as the healing process has been attended with less sepsis and is, therefore, more rapid, local discomfort has been decreased. During those eight years neither reports nor visual evidences have been presented to show the occurrence of remote discomforts nor the existence of pharyngeal deformities which could be attributed to the proper expansion of the field of operation.

THOMAS RUSHMORE FRENCH.

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## THE LINGUAL TONSIL

The lingual tonsil is that part of Waldeyer's ring of lymphoid tissue which is situated at the base of the tongue, lying directly behind the papillæ circumvallatæ and stretching in the adult across the whole width of the tongue. Usually this is the last part of the ring to take on full development and function and frequently at birth is quite a simple structure with no well developed crypts or follicles. Later as the individual grows into adult stature there appear well-developed masses on each side of the median line frequently so close together as to be practically one mass, but always capable of one-sided inflammation and enlargement. When fully developed it is like any other tonsil, a circumscribed mass of lymphoid tissue with crypts and follicles. Especially when hypertrophied there is a well-organized layer of connective tissue underneath the lymphoid mass which to all intents and purposes acts as a capsule and definitely separates the tonsil from the large venous trunks which underlie it. This congery of blood-vessels is many times of such proportions as to constitute a veritable plexus, and whether large or small always responds to the emotional states of the host. When fully engorged, and hence a thicker layer, it gives rise to a sensation of a lump in the throat and the so-called globus hystericus is anatomically explained:

The lingual tonsil and this portion of the base of the tongue can be well seen or treated only when the laryngeal mirror is used as for examination of the larynx by the so-called indirect method.

## ACUTE INFLAMMATORY CONDITIONS

**Pathology.**—The fact that this lingual portion is the last part of the ring to come into mature form accounts, when things progress normally, for the added fact that in the main it is beyond puberty when one notices the more frequent involvement of this tonsil in the acute seizures of the throat while the chronic enlargements may not appear until quite late in life. The operative zeal of the present generation of those interested in this part of the body has, however, somewhat altered this sequence of events and the child who has been early bereft of his faucial tonsils and adenoids in their entirety, eventually acquires compensatory enlargement of the lingual tonsil. This makes such children earlier subject to whatever troubles are in store for those so afflicted.

The lingual tonsil is in every way constituted like the rest of the lymphoid ring and like other tonsils has acute inflammations of every variety including abscess and quinsy, has chronic inflammation, may have hyperkeratosis, leptothrix, and permanent hypertrophies.

Inasmuch as the crypts are shorter even though often spread out at the lower end, still there is less constitutional disturbance on the whole in the acute seizures and less tendency for focal infection in the chronic troubles than is the case with inflamed faucial tonsils. On the other hand the lingual tonsil as a whole is much more sensitive and when inflamed or traumatized can be even more painful than other parts of the throat. Other than this, the course of acute or chronic inflammation and hypertrophies differs in no way from that of the faucial tonsils, and the symptoms differ merely because of the position in the throat. The duration and severity, as usual, depend on the nature of the germ and the existing susceptibility of the

patient. Acute inflammation may occur with or without involvement of the rest of the throat, often only on one side, and much more frequently than is generally supposed because the routine examination with the throat-mirror, which is the only way it can be easily and fully seen, is rarely done except by a specialist in his office.

**Symptoms.**—The symptoms of acute inflammation are pain in the region of the hyoid bone which may be referred down into and below the larynx as a sense of fulness (lump feeling), difficult or painful deglutition, exasperating cough, voice fatigue if not actual hoarseness, fever, constitutional disturbance. These may one or all be present. The soreness complained of is entirely different in location and character than when the faucial tonsil is involved. Examination with the throat-mirror shows the lingual tonsils swollen, red, often with follicular plugs exactly as in the case of the faucial tonsils. Rarely an abscess may form in the substance of the tonsil proper or in the peritonsillar tissue (quinsy).

When the latter occurs we should be on the lookout for edema of the glottis. In fact whenever edema of the epiglottis or the aryepiglottic fold exists it would be well to look over the base of the tongue to seek a probable cause.

**Treatment.**—The treatment is exactly the same as is usual for any acute inflammation elsewhere in the throat. Sprays, or gargles, the latter being of special value when hot and performed by swallowing with the mouth open to make the solution drop well down into the inflamed region before doing the actual gargling. Cold packs are very comforting and may be repeated. Adrenaline spray and application should be made by the attending physician, and much relief may be given by gently swabbing with boroglyceride or 20 per cent. argyrol, being careful to apply these to the whole of the inflamed area.

The slight shrinking which results from having these solutions applied between the epiglottis and the lingual tonsil, or between this and the faucial, or between the lingual and French's folds, makes for relief of the irritation and of the cough. This last is often most exasperating as already stated. Abscess or ordinary edema we treat with incision or multipuncture, and when either or both are present a steam atomizer which sprays some alkaline solution made from Seilers' saline and alkaline tablets, also containing an abundant amount of adrenaline, may be almost life-saving.

Constitutional treatment must be given as indicated.

#### CHRONIC INFLAMMATORY CONDITIONS

Subacute conditions cause a continuance of the various symptoms and easily in the adult female lead to a chronic inflammation with or without attending hypertrophies. The complaint is often made of a "feeling of lump in my throat which I keep trying to swallow." The wording varies according to the individuality of the patient, but invariably indicates some kind of distress in the hyoid region. Trouble frequently appears in the use of the voice, mostly a voice fatigue, but in singers a real difficulty in rendering certain tones, especially those of the middle register. In speakers the feeling exists of speaking over something which acts as an impediment and this effort is most fatiguing. An especially annoying symptom is a chronic, dry, often spasmodic, and distressing cough. This is so frequently present that when it occurs in any patient we should always look to the lingual

tonsil for the explanation. Patients are further annoyed by an inordinate desire to swallow something thinking it is due to mucus, but really it is a variation of the "foreign body" feeling so frequently complained of. Curiously it will exist only when nothing is swallowed and ceases to be annoying at mealtime. The ingestion of food seems in these cases to entirely relieve for the time being the sense of something wrong in that locality.

It should be borne in mind that the acute especially, but the chronic inflammations also, may disturb the thyroid gland leading to actual perversion of function. Rarely an accessory thyroid gland may be found as a median round swelling at the base of the tongue.

**Symptoms.**—The symptoms caused by chronic swelling or inflammation are principally due to the actual enlargement or thickening being sufficient to touch the epiglottis or to actually impede it in its excursions in phonation, or deglutition, or the outer edges of the mass may impinge upon the lower end of the faucial tonsils or upon the enlarged tonsillar lingual folds of French. Slight contact gives tickling and cough, broad contact the "lump feeling" and reflex disturbance in the use of the voice and deglutition. So slight is the contact which will make itself felt by the patient that often the simple filling of the blood-vessels of the region will produce a sudden onset of the symptoms when for hours or days there has been complete remission. For this same reason lying down in bed will almost always produce disturbance in one suffering from cough, and this can only be combated by gradually reclining or sleeping with the head high.

**Treatment.**—To treat the varied chronic conditions it is always safe to assume that simple measures are likely to succeed, for ever so slight a shrinking may give present, and by frequent repetition of the treatment, permanent, relief. The patient should gargle weak saline or alkaline solutions, and the physician should treat any obvious trouble existing above in the nose or pharynx. He should at least twice weekly paint quite thoroughly the enlarged or inflamed areas with some simple solution as the following: iodine, 0.8 gm.; potassi iodide, 2.5 gm.; glycerine, 25 gm. This, assisted by the patient gargling two or three times daily, will in an astonishing number of cases afford the immediate relief desired. Later, when necessary, the hypertrophies can be shrunk by galvanocautery or removed by snare or guillotine, but one always must bear in mind that the region is so sensitive that only a few spots can be cauterized at one time, and that when using radical surgical means serious hemorrhage may occur.

In cauterizing, the usual procedure is to apply, three or four times, a 10 per cent. solution of cocaine on the side of the lingual tonsil selected for that day. Then under the guidance of the laryngoscopic mirror, the patient holding his tongue with his hand, the operator selects certain prominent areas into which he buries the tip of the heated electrode deeply enough to cause a definite sear and subsequent contraction. In well-developed enlargements as much as  $\frac{1}{8}$  inch is perfectly easily penetrated. One should always endeavor to penetrate between the crypts rather than into them. In the latter instance one might readily seal up a crypt, with all it contained—a most undesirable result. Four or five thrusts are surely enough at a time, and a week or ten days later when the inevitable and sometimes not inconsiderable soreness has subsided the other side will be cauterized the same way. This necessitates a number of

repetitions, and it often hastens the final result to remove very prominent or isolated enlargements by the snare or guillotine. After cocaineizing as thoroughly as possible the selected mass may be encircled by the ordinary wire snare or better still the galvanocautery snare and the mass slowly snared away. A Myles lingual tonsil guillotine often serves to remove the mass even more readily than the snare, is less painful, and is more quickly accomplished, but does make more bleeding. Dr. Thomas R. French and Dr. Sydney Yankhauer have both recommended and accomplished the removal of large masses stripped from the base of the tongue together with the tonsillar-lingual folds in operating on the latter. Such a procedure requires skill and boldness, but has as a reward the same satisfactory results as we are familiar with in the complete tonsillectomy as contrasted with the older tonsillotomy.

In striking contrast to this radical procedure some observers report excellent results from the Roentgen ray and radium used as is done when the faucial tonsils are treated by this conservative method.

Nothing in the foregoing pages should be interpreted as in any way minimizing the importance of the lingual tonsil and the necessity of treating diseased conditions in it, for in the author's experience pathology in this region demands attention quite as frequently as in any other portion of the throat and rewards the attention given as no other does.

HENRY L. SWAIN.

### THE PALATINE TONSILS

**Anatomy.**—*Position.*—The palatine or faucial tonsils are two masses of lymphoid tissue partly embedded, one in each lateral wall of the oral part of the pharynx. In shape and size they may be likened to almonds, but they vary considerably in different individuals and at different ages.

Each tonsil lies in the lower part of a recess, the "sinus tonsillaris," which is bounded in front and behind by two folds, the anterior and posterior pillars of the fauces; these are projections of mucous membrane covering the glossopalatine and pharyngopalatine muscles respectively, hence the term "glossopalatine" and "pharyngopalatine arches" also applied to them. The sinus is limited below by the margin of the tongue, and above by the soft palate. Its upper part presents a "supratonsillar recess," but since the tonsillar tissue extends upward, beneath, and around this recess into the soft palate it is better termed the "tonsillar recess" (Sir St. Clair Thomson) or "intratonsillar fossa" (Tilley); it is inconstant and variable in shape and size. On drawing the anterior pillar of the fauces forward, outward, and slightly downward, a triangular fold of mucous membrane is seen passing backward to the tonsil. This is known as the "plica triangularis" (His), and consists of mucous membrane covering a layer of fibrous tissue which is continuous with the capsule of the tonsil; its upper part may be distinguished as the "plica semilunaris" or "supratonsillar," and forms the medial wall of the supratonsillar recess.

*Relations.*—The projecting or free surface of the tonsil is covered by mucous membrane and is studded with small crypts, from which numerous follicles branch out into the tonsillar substance. The deep or attached

surface is adherent to a fibrous capsule, continuous with the plica triangularis; the capsule is separated by loose connective tissue from the superior constrictor muscle of the pharynx, external to which lies the facial (external maxillary) artery with its tonsillar and ascending palatine branches; rarely the lingual or facial vessels may extend up higher than usual and lie in close relation with the lower part of the tonsil. The internal carotid artery lies about an inch posterior and lateral to it.

**Vascular Supply.**—The arterial supply is derived from several sources, and the arrangement of the vessels is irregular (Fig. 162). All are ultimately derived from the external carotid artery, and for the most part pierce the capsule to enter the lower half of the organ; an important branch lies just behind the anterior pillar of the fauces. The ascending palatine and tonsillar branches of the facial artery enter the base of the tonsil; anteriorly it receives branches from the lingual; posteriorly from the ascending pharyngeal branch of the external carotid; above from the descending palatine branch of the internal maxillary together with a twig from the small meningeal.

The veins form a tonsillar plexus on the lateral aspect, which drains into the lingual vein and the pharyngeal plexus.

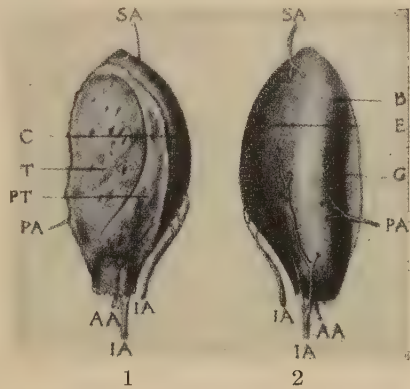


Fig. 162.—Actual shape of left tonsil, with arterial supply: 1, Mesial aspect; 2, posterolateral aspect; *T*, tonsil tissue; *PT*, plica triangularis; *C*, capsule; *AA*, anterior tonsillar artery; *PA*, posterior tonsillar artery; *SA*, superior tonsillar artery; *IA*, inferior tonsillar arteries; *E*, lateral surface; *B*, posterior surface; *G*, groove for palatopharyngeus (Fetterolf).



Fig. 163.—Enlarged tonsils becoming pedunculated.

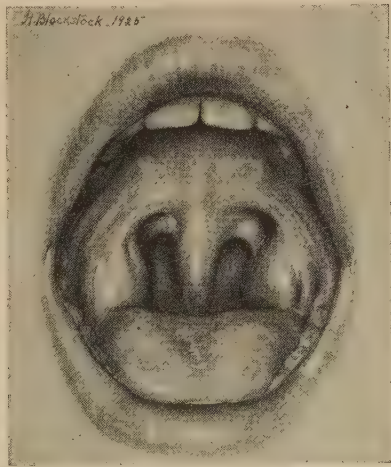


Fig. 164.—Buried tonsils.

The lymphatics, which are connected with adjacent areas of the mucosa in the pharynx, mouth, and lower part of the nasal cavity, pass chiefly to the upper deep cervical lymph-nodes. One of these, situated just behind

the angle of the jaw beneath the anterior edge of the sternomastoid muscle, is so constantly enlarged in tonsillar infection that it is called the "tonsillar lymph-gland" (Wood). There are also connections with the submaxillary and superficial cervical nodes.

The innervation is from the sphenopalatine ganglion and the glossopharyngeal nerves, which form a plexus—the "circus tonsillaris."

*Types of Tonsils.*—The palatine, like the lingual and pharyngeal tonsils, is very variable in size. Many variations are developmental, but like all lymphoid structures they exhibit age changes. Usually they are best developed in childhood, often abnormally so; they show retrogressive changes after puberty and atrophy in old age.

Two distinct types are recognizable:

1. The projecting type, which varies in size and shape; such tonsils may be so large as to meet in the midline, or assume almost a pedunculated form (Fig. 163).

2. The buried type (Fig. 164). Usually one-third only of the tonsil is embedded in the wall of the pharynx, but in this type the greater part of the organ is hidden; it may be made manifest by pressing on the anterior pillar, and is often accentuated if the patient retches during examination. As already noted, the tonsil extends upward, beneath or external to the supratonsillar recess, into the soft palate; it also extends downward to the tongue in the form of infratonsillar nodules, and passes forward for a variable distance beneath the plica triangularis and anterior pillar.

The *physiological rôle* of the tonsil is still imperfectly understood. By some its function, as of lymphoid tissue elsewhere, is considered to be merely that of the production of lymphocytes (Oppel). I believe, however, that in conjunction with the rest of the scattered circular band of adenoid tissue which guards the pharynx at the openings of the digestive and respiratory passages (the "ring of Waldeyer," comprised by the palatine and tubal tonsils at the sides, the pharyngeal tonsil above, and the lingual below, with smaller masses in between) it acts as a first line of defence against bacterial invasion, the second being the lymphatic glands. The experiments of v. Lenart, who found that substances injected into the nose are partly intercepted in the tonsils as well as in the cervical nodes, appear to support this view.

HERBERT S. BIRKETT.

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### ACUTE CATARRHAL TONSILLITIS

In this affection the mucous membrane covering the tissue is the part chiefly affected, and the condition is nearly always a part of an acute pharyngitis.

*Etiology.*—This disease is most pronounced in children following sudden changes in temperature and exposure to cold, gastro-intestinal affections, and many of the exanthemata.

*Symptoms.*—The first symptom may be a sense of chilliness, or in the case of young children possibly a convulsion. Usually the chill is followed by

headache, general depression, temperature ranging from 100° to 102° F.; the pulse is full and bounding; deglutition is painful; and the muscles of neck and the cervical glands may be tender. Upon examination in the early stage of the disease the surface of the tonsil and of surrounding structures presents a marked degree of hyperemia, and later on a very thin, whitish exudate may be seen at the opening of the crypts. The disease usually runs its course in about four days if unattended to, and may by its extension involve the middle ear in an acute inflammatory process, either with or without suppuration.

**Treatment.**—Begin with a purgative, such as calomel and soda in small doses, gr. 1/10 each, frequently repeated. If the temperature be high, and the pulse full and bounding, small doses of tincture of aconite, U. S. P.,  $\pi j$  would be of service. Cold alkaline sprays, such as the following, will be found to give relief in the early stages of the disease:

R. Sodii bicarbonatis }	.....	āā gr. xx.....	1.3 gm.
Sodii biboratis }	.....		
Ol. cinnamomi.....	$\pi ij$	.....	0.12 c.c.
Aquæ.....	$\mathfrak{z} iv$	.....	120 c.c.

Applications of a solution of nitrate of silver (gr. xxx to the ounce) once a day, or guaiacol ( $\mathfrak{z} j$ – $\mathfrak{z} j$  glycerine) once every four hours, to the surface of the tonsil and into the crypts, are strongly recommended. Should the disease progress to such an extent that the inflammatory process shows by its intensity that the underlying structures have been invaded, then local depletion (scarification) is advisable, and the spray or gargle should be used hot.

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## ACUTE LACUNAR TONSILLITIS

This affection is an inflammation of the crypts or lacunæ of the tonsils, and is characterized by the filling up of these crypts with inflammatory products which appear on the surface as a white or yellowish-white exudate, accompanied by an involvement of the adjacent and deeper lying structures in the inflammatory process.

**Etiology.**—Among the *predisposing* causes a lowered state of the general system stands pre-eminently first. Sudden changes in the temperature: imperfect sanitation; close, overheated rooms and a foul atmosphere; contamination of the milk or water supply of the house; an unhealthy condition of the tonsils, teeth, and gums; suppurative conditions of the nasal accessory sinuses; mouth breathing due to obstructed nasal respiration, all act as predisposing causes. This disease is most prevalent in the spring and autumn, and most frequently met with between the ages of ten and fifty years. One attack is apt to predispose the patient to future attacks. Operations upon the nose are at times followed by acute tonsillitis, the infection being carried by way of the lymphatics. The *exciting* cause is the presence of certain bacteria, notably the pyogenic cocci (Streptococci, Pneumococci, and Staphylococci).

**Symptoms.**—*Subjective.*—The disease is usually ushered in by headache, pain in the back or extremities, chilliness, or even rigor. The throat

is painful, especially when swallowing, and even speaking may be painful. The pain extends upward when swallowing to both ears if both tonsils are involved, or, if limited to one tonsil, to the ear on the affected side. The temperature varies from 100° to 104° F.; the pulse is rapid (100–120), full and bounding. The ears have a sense of fullness due to acute catarrh of the eustachian tubes, which is not infrequently brought about by the extension of the inflammatory process to the lymphoid tissue in the vault of the pharynx, which causes mouth-breathing, a distressing symptom. The urine is highly concentrated, increased in specific gravity, and sometimes shows the presence of albumin during convalescence. Usually the disease begins on one side, and within twenty-four hours the other side becomes involved. The glands behind the angle of the jaw are frequently swollen and tender. The inflammatory condition may extend to the parenchyma (lymphoid follicles) of the tonsil when the symptoms become intensified (*parenchymatous tonsillitis*).

*Objective.*—The tonsils are seen to be swollen and hyperemic, and if the parenchyma is involved the hyperemia may be of a livid hue. On the surface of the tonsil are to be noticed small, isolated spots of exudation, their size and shape corresponding very often to the openings of the crypts. This exudative material varies in color; it may be white, yellow, or gray. The exudations may be limited to the openings of the crypts, or may extend and coalesce with that from the neighboring crypts, giving it, in some cases, a distinctly membranous appearance. The exudation may be easily wiped off by means of a cotton-wool swab, or it may be so dense as to be adherent to the mucous membrane and require the use of a pair of forceps to loosen it, and when removed exposes a hyperemic but not bleeding area. When the disease occurs in children, and a rhinoscopic examination is possible, the lymphoid tissue in the vault of the pharynx will be found studded with a similar exudate; but in the case of the adult the lingual tonsil also is often similarly affected. The uvula is usually hyperemic and sometimes swollen. In persons who have suffered from recurring attacks of acute lacunar tonsillitis the secretion is apt to have a very fetid odor.

**Diagnosis.**—As a rule there is very little difficulty in recognizing this condition, but sometimes it may not be easy to distinguish it from diphtheria. A differential diagnosis may be assisted by a comparison of the symptoms of the two diseases:

*Acute lacunar tonsillitis*

1. The onset is sudden.
2. The temperature is elevated (101°–104° F.).
3. The pulse is full and bounding.
4. The face is flushed and the expression is often one of alertness.
5. The odor of the breath, while it may be heavy, is not foul.
6. Vomiting is uncommon.
7. A chill frequently ushers in the attack.
8. The exudate is thin, and of a pale yellowish or whitish color.
9. The exudate may be readily wiped off, or if removed by forceps leaves no bleeding area, and it does not re-form.

*Diphtheria*

1. The onset is slow and insidious.
2. The temperature is lower (99°–100° F.).
3. The pulse is slow and compressible.
4. The face is apt to be pale, and the patient depressed.
5. The breath has a distinctly foul odor.
6. Vomiting is frequent with the onset, especially in adults.
7. A chill at the onset is uncommon.
8. The membrane is thick, and of a dirty gray or deep yellowish color.
9. The membrane cannot be wiped off, and when removed by forceps leaves a bleeding surface, and the membrane readily re-forms.

*Acute lacunar tonsillitis*

10. Examination (when possible) at the onset shows the lymphoid tissue in the vault of the pharynx and at the base of the tongue to be involved with a discrete exudate similar to that seen on the tonsils.
11. The submaxillary glands are generally enlarged on both sides.
12. Knee-jerk is present.
13. Cultural examination usually shows streptococci.

*Diphtheria*

10. Examination under similar conditions of the corresponding areas will not show any membrane present such as is seen on the tonsil.
11. The submaxillary glands are involved only on the affected side.
12. Knee-jerk is absent in the early stages.<sup>1</sup>
13. Klebs-Löffler bacillus is always present.

**Treatment.**—As these acute inflammatory processes of the tonsil are of an infectious character, it is advisable to isolate such cases. A purgative of calomel and soda should be given at the outset, about 4 grains of calomel, 3 grains of soda. Small doses of the tincture of aconite (℥1–2) may be given hourly until the pulse and temperature are lower, provided of course that the pulse is rapid, full, and bounding. The salicylates in various forms have in some cases proved advantageous: 5 grains of salol and 3 grains of phenacetin given every two hours in combination, I have found most beneficial. Guaiacum, however, a very much vaunted remedy, has not in my experience proved to be especially efficacious. Benzoate of soda in 10-grain doses is warmly advocated by many authors. Locally, the tonsils should be sprayed with a warm alkaline and antiseptic solution:

1. Acidi carbolici .....	gr. j.....	0.065 gm.
(Calvert's No. 1.)		
Sodii bicarbonatis .....	gr. v.....	0.325 gm.
Aquæ .....	℥j.....	30 c.c.
2. Sodii bicarbonatis .....	gr. v.....	0.325 gm.
Listerine .....	℥j.....	3.56 c.c.
Aquæ .....	℥j.....	30 c.c.

Whenever possible it is advisable to clear the crypts of any retained secretion, and this can be readily done by means of a small, dull curet or cotton-wool swab. Another method which I have found beneficial for dislodging these inflammatory plugs is to play against the tonsil, by means of a syringe, a stream of rather warm boric acid solution (20 grains to the ounce). A still equally ready and efficacious manner of carrying this method into practice is by means of an ordinary syphon of soda-water to which a couple of feet of rubber tubing is attached, with a glass pipette fitted at the other end. In carrying out either of these methods it is necessary to have the patient leaning a little forward, and thus facilitate the escape of the fluid by the mouth. The application of either guaiacol, the muriated tincture of iron, or tincture of iodine (℥j to the ounce of glycerine), by means of a cotton-wool swab, into each crypt will tend to cut short the course of the disease in many cases. If it is impossible to make these applications, then these remedies may be used in the form of a spray, which is more efficacious than gargling; in this case, the guaiacol should be used as a 1 per cent. solution, tincture of iodine in the same strength, and muriated tincture of iron in doses of ℥v combined with gr. iv of chlorate of potassium to the ounce; acriflavin (1 : 2000) as an application to the tonsils will be found useful. There is little to choose between any of the above remedies, as each drug has its own advocates. Hot fomentations to the neck will afford relief in many cases, especially when the glands are

tender. The diet in cases of acute tonsillitis should consist of liquids and semisolids, such as milk, soup, jellies or custards. The course of the disease is usually favorable, terminating in recovery in five to seven days.

**Complications.**—It must be borne in mind that acute lacunar tonsillitis may be the forerunner of specific infectious disorders, especially scarlet fever; further that acute rheumatic fever, endocarditis (especially Libman's type), albuminuria (glomerulonephritis), and erythema nodosum, may be a sequence of this affection. Therefore the patient's general condition should be carefully watched not only during the course of the disease, but also during convalescence. As a sequence of acute lacunar tonsillitis, peritonsillar abscess occasionally develops, and the infection may extend to the eustachian tube or the middle ear.

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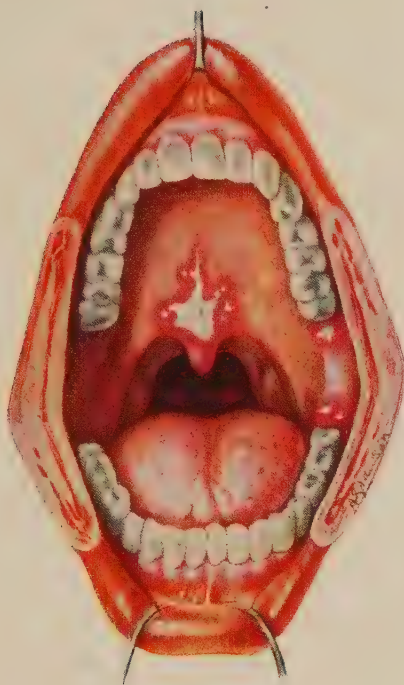
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### VINCENT'S ANGINA

This is an infection of the tonsils due to the presence of a fusiform bacillus and a spirochete, first discovered by Vincent. It is characterized by deep crater-like ulcerations involving the tonsil. Until 1914 this affection was regarded as a rather unusual one, but during the Great War, and especially in the earlier years, it was frequently met with amongst the troops; but in those units which had a dental surgeon attached, the condition was seldom seen, due to the attention given to mouth conditions. From my experience with this affection overseas, I believe that the chief predisposing cause is invariably an unhealthy condition of the teeth and gums (pyorrhea).

**Symptoms.**—The usual complaint is that of a sore throat on one or both sides. The discomfort is not greatly increased by mastication or swallowing, and the moderate degree of pain present does not radiate. There is usually very little, if any, general disturbance; the temperature seldom rises above 99° to 100° F.; the submaxillary lymph-glands are enlarged but not tender if only the tonsil is slightly involved; an odor to the breath is noticeable when the affection is extensive. *Objectively*, the ulcers may appear either single or multiple on any portion of the tonsil. The affected area in the early stage is covered with a grayish-white, pulpy looking exudate which is easily removed without bleeding, exposing a superficial ulcerated surface, the edges of which are clearly defined but not indurated. The surrounding area of the ulcer is quite hyperemic. If, however, the disease has existed for some time, then the ulcers have a deep crater-like excavation (Plate III), and may be so large as to involve the whole area of the tonsil. Both tonsils may be involved to a greater or less degree at the same time, and may continue to destroy the greater portion of the tonsillar tissue. Occasionally it involves the lymphoid tissue on the lateral wall of the pharynx, and more rarely may extend to the larynx or may invade the bronchi secondarily.<sup>1</sup> Frequently the gums on the buccal

PLATE III



Vincent's angina involving the soft palate.



Vincent's angina, showing deep crater-like excavation.



surface about the teeth show at the same time superficial ulceration from which the specific organisms may be obtained. In one case I found an ulceration at the base of the uvula, as shown in Plate III. The termination of the disease when extensive, manifests itself in definite cicatrices, which, on account of their extent and position, resemble those seen in healed lues.

**Diagnosis.**—The affection may possibly be mistaken for diphtheria, syphilis, or malignant disease. It is readily differentiated from diphtheria by the fact that the condition in Vincent's angina is more localized; there is general absence of hyperemia in the surrounding tissues; the exudate is readily removed, and there is not the general depression manifested in Vincent's angina as in diphtheria. A smear submitted to the microscope will at once show the presence of the fusiform bacillus and spirochete in the one, and the Klebs-Löffler in the other. Regarding the syphilitic lesion, the area involved in syphilis does not show such extensive deposit of exudate. The granulations are much more florid, and the edges of the ulceration are very definitely indurated in a syphilitic lesion. The presence of positive Wassermann will support the luetic condition. If it is malignant, the edges of the ulceration are very firm and hard, and the surface bleeds readily; the submaxillary glands are found to be hard and firm; a section will reveal its nature.

**Treatment.**—The exudate should be removed by means of any form of alkaline spray; this would be further facilitated by applications of peroxide of hydrogen by means of a cotton-wool swab, and when the ulcerated area is thoroughly cleansed, an application of liq. arsenicalis has been found to be very effectual. This should be applied four times daily. It is very essential in the beginning of treatment that all defective conditions of the teeth and gums should receive prompt attention. In obstinate chronic cases the application of salvarsan and glycerine, or neosalvarsan, 1/12 gr., with 2 drams water, is found to be of considerable benefit. Either of these remedies used intravenously has been found to cure certain very obstinate cases.

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#### KERATOSIS

This is a condition manifested by a deposit of a distinct white, horny-like substance, involving any portion of the lymphoid "ring of Waldeyer." The predisposing cause is usually a debilitated condition of the system. The majority of cases occur in adult life, and more often in women than in men.

**Symptoms.**—Very frequently there are no symptoms until the patient has accidentally discovered the presence of white spots in the throat, and has become uneasy about them; or he may complain of a feeling of a foreign body in the throat, and of a desire to swallow or to clear the throat repeatedly. Upon examination "*white spots*" varying in number, are to be seen distributed on the tonsils (palatine and lingual), on the posterior and

lateral walls of the pharynx, and in the nasopharynx. These "spots" look like bristly points standing out from the mucous membrane of the involved part, to which they are intimately adherent (Fig. 165). In this affection there is an entire absence of any general disturbance.

**Diagnosis.**—This condition is very frequently mistaken for chronic lacunar tonsillitis, but the readiness with which the plugs of this latter affection can be removed should serve to differentiate it at once from keratosis.



Fig. 165.—Keratosis tonsillaris.

**Treatment.**—As this condition is apt to occur in neurotic and rundown individuals, attention to the general health is most important. The use of the curet and galvanocautery does not lend itself to recommendation, requiring, as it does, extended treatment with a great deal of subsequent discomfort to the patient and uncertainty of the result. I have found, however, that the application of a 10 per cent. solution of salicylic acid in sulpho-

ricinate of soda thoroughly rubbed in, has in some cases been the means of entirely removing this condition.<sup>1</sup> The following local treatment is advised and found beneficial by Portmann:

R.	Resorcin	}	.....	āā	℥iss.....	6.0 gm.
	Sod. benzoatis					
	Glycerine.....				℥ij.....	60 c.c.
	Decoct. althææ rad.....ad.				℥xvj.....	480 c.c.

To be used as a gargle.

or

R.	Iodi.....	gr. iv.....	0.26 gm.
	Pot. iodid.....	gr. v.....	0.325 gm.
	Glycerine.....	℥iij.....	90 c.c.

To be applied to the tonsils.

Enucleation of the tonsils is advocated as a desirable procedure in this affection; but I have not found such radical measures necessary, as the condition has usually given way under the treatment as above mentioned.

HERBERT S. BIRKETT.

#### REFERENCE

1. Grant, Dundas: Brit. Med. Jour., 1907, May 11, p. 1155.

### TONSILLOLITHS

Frequently the retained "cheesy" secretion in the crypts undergoes calcareous degeneration, constituting what is known as a calculus or tonsillolith,<sup>1</sup> the nucleus of which is of an organic nature. They vary in consistency, chemical composition, and size. Usually there is only one, but occasionally several small ones may be present.

**Symptoms.**—The presence of the calculus may produce no symptoms at all, and may be accidentally discovered. Usually, however, there is a sense of fulness or soreness about the tonsil. Recurring attacks of subacute

tonsillitis, clearing of the throat, and an irritable cough may be present. It occurs in adults approaching middle life. Upon examination the tonsil involved may seem unduly prominent in a limited area. The existence of such a condition is usually discovered by probing the tonsil, although the calculus may sometimes be visible within the crypt.

**Treatment.**—A single small calculus may often be dislodged by means of a curet or probe, or it may be so embedded in the substance of the tonsil that it is necessary to enlarge the opening in order to extract it. If there is a marked chronic lacunar tonsillitis associated with the tonsillolith, enucleation then is preferable.

HERBERT S. BIRKETT.

#### REFERENCE

1. Weller, C. V.: The Incidence and Pathogenesis of Tonsillar Concretions, *Ann. Otol. Rhinol., and Laryngol.*, 1924, vol. 33, No. 1, March, p. 79.

### FOREIGN BODIES IN THE TONSIL

These may be of any kind or nature, the most common being fish-bones, spiculæ of bone, bristles of a tooth-brush, and husks of grain. The most common site is the center of the tonsil, but frequently they are hidden behind the anterior pillar of the fauces. By repeated swallowing the foreign body, especially in the case of fish-bones, is apt to be driven deep into the substance of the tonsil, leaving a very minute portion exposed. Sometimes this portion is covered with secretion, and at first sight may be overlooked. It is therefore necessary, where there is difficulty in locating it, to mop off any secretion which may be covering the tonsil. Sometimes palpation with the index-finger will locate a spicule of bone, or a fish-bone, or a bristle buried out of sight in a crypt. If no foreign body can be found the patient should not be dismissed without complete Roentgen-ray examination. Removal of the foreign body in this region is easily accomplished by means of a pair of forceps, giving immediate relief to the symptoms produced by its presence.

HERBERT S. BIRKETT.

### OSSEOUS AND CARTILAGINOUS FORMATIONS IN THE TONSILS

In a most recent and elaborate investigation, of which the following is an abstract, Irwin Moore<sup>1</sup> has divided the subject into two groups, namely, one in which the bone is extrinsic in origin, and extends from the skeleton into the tonsil; the other in which the bone is intrinsic, originating in the tonsil itself.

**Group I.**—The styloid process, which is derived from the second branchial arch, normally reaches  $2\frac{1}{2}$  cm. in length, and extends from the under surface of the petrous portion of the temporal bone, forward, downward, and inward. The tip is cartilaginous, and the abnormal length of the styloid process may reach as far as the hyoid bone, or it may be *abnormally directed inward and forward*, and encroach upon the tonsil. This condition may be

unilateral, but is frequently bilateral. It is found usually in adults, and is more common in men than in women.

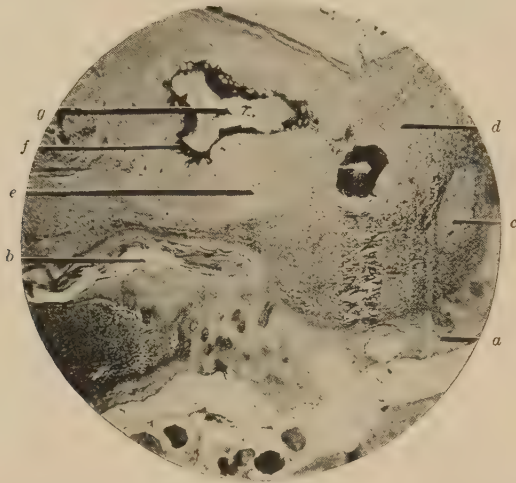


Fig. 166.—Section of tonsil (low power) showing: *a*, Surface covered by stratified epithelium; *b*, crypts lined by stratified epithelium; *c*, lymphoid tissue showing germ-center; *d*, hyaline connective tissue; *e*, cartilage; *f*, bone; *g*, bone-marrow.

**Symptoms.**—The condition may cause abnormal sensations in the throat, such as the feeling of impaction of a bone or other foreign body,

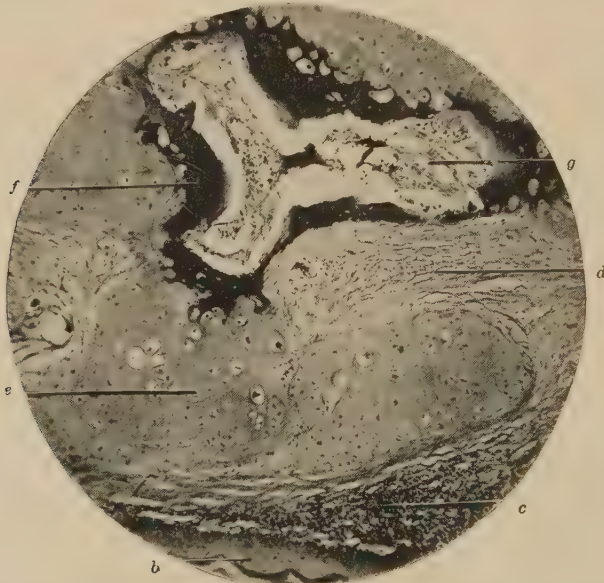


Fig. 167.—Section of tonsil (same as Fig. 166—high power): *b*, Crypts lined by stratified epithelium; *c*, lymphoid tissue; *d*, hyaline connective tissue; *e*, cartilage; *f*, bone; *g*, bone-marrow.

irritation and frequent sore throat: in some cases a continued impediment in swallowing.

**Diagnosis.**—Bimanual palpation shows a rounded piece of bone coming to a point in the tonsillar tissue, which in some cases may reach close to the surface. The Roentgen ray will assist in the diagnosis. One should bear in mind the possibility of an elongated styloid process being mistaken for a bone or other foreign body, especially in cases in which a history is given of a foreign body having been swallowed. The importance of this abnormality is shown by the fact that difficulties may arise during enucleation of the tonsils, especially when the guillotine is being used.

**Group II.**—Bone or cartilaginous formation scattered through the tonsils is a fairly common condition, and one generally discovered accidentally through the microscopical examination of the tonsils (Figs. 166, 167). This condition has been found in an embryo of thirteen weeks, and in a patient of seventy-five years. Cartilage is invariably situated in the fibrous capsule of the tonsil in close proximity to the pharyngeal muscle.

HERBERT S. BIRKETT.

#### REFERENCE

1. Moore, Irwin: Osseous and Cartilaginous Formations in the Tonsils, Jour. Laryngol., Rhinol., and Otol., 1924, vol. 39, Nos. 3 and 4.

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### HYPERTROPHY OF THE TONSILS INCLUDING CHRONIC LACUNAR TONSILLITIS

Hypertrophy of the tonsils is a condition which may be found in any age of life, although there is a tendency to atrophy at the age of puberty. An inherited tendency to hypertrophy of the lymphoid tissues is evidenced in many families. The *most common types* of tonsil are already depicted and described elsewhere. In character they may be either soft or lymphoid, firm or fibroid. The former is more common in children, the latter in adults.

**Etiology.**—All inflammatory infections of the throat, whether due to local or systemic causes (infectious fevers), will produce chronic enlargement of the tonsils. Stoker<sup>1</sup> states that tonsillar hypertrophy is more common in children where there is malnutrition. Clark found that a high fat and carbohydrate diet increased the size of the lymphoid areas both in the throat and in the intestine. Stucky found that tonsillar hypertrophy was very common in children of Kentucky mountaineers who were fed on a devitaminized diet. Even after tonsillectomy these children did not improve until they were given a diet of milk, eggs, butter, and wholemeal. Stoker believes that wholemeal, green vegetables, raw fruit, fat soluble A foods and an allowance of meat as a diet would lead to an enormous shrinkage in the number of hypertrophied tonsils and adenoids. Several local conditions are apt to lead to a chronic hypertrophy of the tonsil, especially in those cases where there are recurring attacks of tonsillitis due to a diseased condition of the crypts (retention of secretion, chronic lacunar tonsillitis, tonsillar and peritonsillar abscesses), and mouth breathing dependent upon nasal obstruction. In young individuals the condition is usually found co-existing with the enlargement of the pharyngeal tonsil

(adenoids); but in older individuals this is not always the case, as the lymphoid growth in the vault of the pharynx is often very atrophied, while in the fauces it is very marked. Lymphoid leukemia produces a marked enlargement of the tonsils which is apparently increased by the enlarged lymphatic glands of the neck forcing the tonsils inward, in some cases to such an extent as to make them meet.

**Symptoms.**—*Subjective.*—When the tonsils are considerably enlarged they produce mouth breathing, thick speech, and frequently a cough when the patient is lying down, due to the lower part of the tonsil being so enlarged as to irritate the upper part of the epiglottis. In lesser degrees of hypertrophy there may be no symptoms or inconvenience. It is to be borne in mind that the above mentioned symptoms, especially in children, are apt to be associated with the enlargement of the pharyngeal tonsil. In adults the presence of enlarged tonsils is apt to produce fatigue of the voice in either speaking or vocalizing. Frequently, patients complain of noticing white spots on the tonsil, and of a foul breath, and upon manipulation of the tonsil itself these spots are extruded in the size and shape of a very small pea, and when crushed produce a very disagreeable odor. The effect on the hearing is indirect, and largely due to the interference with the action of the palatal muscles, and not to direct pressure upon the openings of the eustachian tubes.

*Objective.*—1. The tonsils may be *enlarged* to various degrees, from just beyond the edges of the pillars of the fauces to meeting each other in the midline. In children the tonsil is rather soft and of a pale rose color, and the surface is smooth. In the adult the tonsil is hard and fibroid in appearance, and paler in color; the surface irregularly divided by bands of fibrous tissue, and the crypts often widely open, and sometimes filled with caseous plugs.

2. The tonsils may be *submerged*. In this type the tonsil seems relatively small, being hidden by the expansion of the plica triangularis; but when the anterior pillar is drawn to one side and pressure made backward, the tonsil is thrown out into relief, and reveals its enlarged condition. At the same time a thin, milky-looking material may be squeezed out, especially from the upper pole, in addition to caseous plugs. Sometimes the posterior pillar may also be found firmly adherent to the tonsil. It is this type of tonsil which most frequently is the source of focal infection. The submaxillary lymphatic glands are often enlarged.

Occasionally it happens that the opening of a crypt becomes closed, producing a condition known as a *retention cyst*, which shows itself as a white, or yellowish-white, spot covered by a thin mucous membrane which may vary in size from that of a pin head to that of a large-sized pea.

**Diagnosis.**—This usually presents no difficulties; but hypertrophied tonsils may be found in lymphoid leukemia, when the condition will readily be recognized by a general but discrete enlargement of the lymphatic glands, and through an examination of the blood. Sarcoma of the tonsil may be mistaken for a simple hypertrophy, but the hardness of the tonsil to the touch, and the distribution of numerous fine vessels in the mucous membrane covering the tonsil will serve to distinguish it from simple hypertrophy. Chronic lacunar tonsillitis may be mistaken for keratosis tonsillarum; but reference has already been made to this condition. (See page 289.)

**The Tonsils as a Focus of Infection.**—The lacunæ, containing accumulations of lymphocytes, leukocytes, and epithelial débris, are aptly called "culture tubes," in which are to be found by cultivation various forms of bacteria, notably pyogenic cocci (Streptococci, Pneumococci, and Staphylococci) which may enter the system either directly by the blood-stream, or through the lymphatics. The tonsils therefore may most frequently be regarded as responsible for many systemic infections both of the acute and chronic variety. Among the systemic conditions following an acute or subacute lacunar inflammation of the tonsil, may be cited acute rheumatic fever, endocarditis (especially the Libman type), hematuria, orthostatic albuminuria, acute nephritis, erythema nodosum, chronic rheumatism, infectious arthritis, myalgia, chorea, and a few skin diseases: urticaria, purpura, erythema multiforme recurrens. In approximately 5 per cent. of cases with the tonsils as a portal of infection, may be mentioned tuberculosis in the tonsil itself and in the lymphatic glands.

The question of the tonsil acting as a portal of infection, or as a primary focus to systemic infection, is one which of recent date has attracted considerable attention, and it is a matter of extreme importance that any decision regarding the part played by the tonsils in the infection should be arrived at by a process of elimination, and not by a mere hasty diagnosis. Therefore, to arrive at an intelligent conclusion there should be co-operation between the specialist and the internist. If this be done thoroughly and conscientiously it will lead to a clearer understanding as to which class of patients will be benefited by operative interference, and which will not, thus placing the operation in a better light, and doing away with the discredit upon such procedure which evidently follows indiscriminate decisions.

"The factors<sup>2</sup> which must be considered in deciding whether a particular tonsil is acting as a focus or not" are extremely well stated by Pavey-Smith as follows:

"The *history* cannot be depended upon; but careful enquiry must be made about sore throat and quinsy. A word of caution with regard to this. Many patients admit to sore throats, which, on closer questioning, turn out to be laryngeal, and in no way connected with the tonsils. Again, a distant attack of tonsillitis may have been forgotten, or, more commonly, the patient is inaccurate. Further, a history of tonsillitis is by no means an essential feature of chronic tonsillar infection. This lack of symptoms and history is a familiar feature of other focal infections.

"A positive history, therefore, especially if bearing a definite time-relation to the systemic disease, is strongly suggestive; but a negative history is no guide.

"The *size* of the tonsils, both apparent and real, conveys little information. Enlargement is certainly no criterion of the extent of the infection. A highly infected tonsil may be almost invisible, and a conspicuous one innocent.

"A more reliable sign is *redness*. If this is limited to the tonsil and its immediate surroundings, it may be taken as definite evidence of tonsillar infection. In some cases there is only a vertical red streak on the anterior pillar, the rest of the mucous membrane of the mouth and throat being normal in appearance. This red streak is often little more than  $\frac{1}{8}$  inch wide, but runs the whole length of the pillar. Sometimes it takes the form of minute dilated vessels."

In an interesting observation H. H. Lott has drawn attention on the subject of tonsillar focal infections to "a new diagnostic point,"<sup>73</sup> in which he recognizes two distinct types of redness of the anterior pillar of the fauces; in one there is a narrow, sharply limited and very dark red zone, which he states is typical of an infective tonsillar focus predominatingly streptococcic in character; in the second type the zone of redness is broader and a paler red, which fades off gradually into the velar mucosa with no perceptibly definite border. This latter, the author states, is "diagnostic of a tonsillar infective focus in which Streptococci do not predominate, and in which a symptom of the remote pathology, such as arthritis, neuritis and the like, is not to be expected, even though tonsillectomy may be indicated for purely local reasons; whereas, in the first type in which the infection is predominatingly a streptococcal one, good results may be expected from operative interference. In this non-streptococcic type of tonsillar focus the predominating organisms are often Staphylococci." Reference to the accompanying colored illustration (Plate IV) serves to demonstrate the ideas the author wishes to convey.

Attention has already been drawn to the condition of the crypts, and how readily the presence or absence of any contents therein may be demonstrated.

I do not place much confidence in the ordinary bacteriological investigation of the tonsil as a factor in determining whether it is a focus of infection, as the various forms of infective bacteria may be found in the tonsils of every living individual. In nearly all tonsillar infections the lymphatic gland just behind the angle of the jaw is usually enlarged.

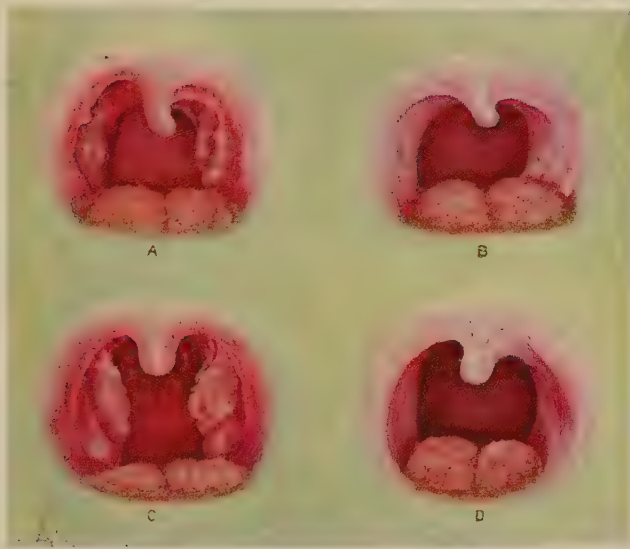
**Prognosis.**—There is a tendency for enlarged tonsils to become smaller as age advances, although I have known of some cases where the tonsils have remained enlarged without ill results.

**Treatment.**—Enlargement of the tonsils is sometimes counteracted by the removal of any obstruction to nasal respiration, and of any infection in the nasal accessory sinuses, teeth, or gums. Local medication, on the other hand, has no effect in reducing the size of the tonsils. If, therefore, the aforementioned corrections have been undertaken, but without satisfactory results, and *the patient does not desire* radical measures, then tonsillotomy may be undertaken. On the continent of North America, however, this operation and other partial interferences have practically been discarded in favor of the more radical measure—tonsillectomy.

In children from the age of a few months to four or five years the removal of adenoids only (provided nasal respiration is thereby established) often does reduce the size of the tonsils, and is, therefore, in my experience, a preferable procedure to enucleation of both tonsils and adenoids.

The question of interfering with the tonsils of singers is one which requires careful consideration. In the case of the young singer whose training has hardly begun, I believe that the tonsils may be removed without damage to the future training of the voice; but when a singer has reached an advanced stage in voice production, then I doubt the advisability of interfering by operative measures, for the reason that the singer is by nature very temperamental, and anything which in the least disturbs his usual method of vocalization will be attributed to the operation.

## PLATE IV



At *A* is seen a characteristic streptococcic tonsillar infection. The inflammatory zone on the anterior pillar is narrow and sharply limited at its outer margin. In such cases a cure of arthritic neuritis and other remote manifestations of focal infection may be expected. At *C* is shown an inflammatory zone on the anterior pillars that is not streptococcic. In such cases no improvement of remote manifestations of a focal infection need be expected from tonsillectomy because the culpable focus is somewhere else. At *B* and *D* are shown the respective cases (*A* and *C*) after tonsillectomy. (Color drawing by Chevalier Jackson.)



## TONSILLECTOMY

**Indications.**—1. Recurring attacks of tonsillitis and peritonsillitis.

2. Interference with respiration.

3. Chronic lacunar tonsillitis, which, after careful consideration, is regarded as the focus of infection in certain systemic conditions.

4. Recurring joint symptoms following attacks of acute or subacute tonsillitis.

5. Proof that the tonsils are carriers of diphtheria.

6. Chronic enlargement of the cervical glands situated near the angles of the jaw.

**Contraindications.**—1. Hemophilia.

2. The period just preceding or during menstruation.

3. Cases of chronic arthritis which show changes in the joints.

4. An abnormal course of arteries, especially the internal carotid coursing in close proximity to, or even in the substance of the tonsil itself (Figs. 168, 169).

5. Acute inflammatory conditions of the tonsil or of the peritonsillar tissues call for postponement of the operation until subsidence of all acute symptoms.



Fig. 168.—Showing an anomalous course of the right internal carotid artery, and its close proximity to the tonsil on the same side.



Fig. 169.—Transverse section showing relations of loop of sigmoid internal carotid artery to tonsil and pharynx.

6. If the patient shows any degree of anemia, and an examination of the blood gives evidence of a marked diminution of the number of red cells, a lowered percentage of hemoglobin, and a delayed blood-clotting period—longer than six minutes—I believe that operative interference should be postponed until these conditions are decidedly improved.

**Preparation of the Patient.**—A thorough physical examination must first be made by an internist, and all other possible sources of infection must be considered, including the nasal accessory sinuses and the oral cavity. Should defects exist in the teeth, or should the gums be unhealthy, these must first be put right. If any apical lesions be present, then these should first be rectified, as I have repeatedly found that when this is done, although the tonsils may show a coexisting chronic lacunar tonsillitis, the patient has thus got rid of his systemic symptoms. In children an enlarged thymus must be excluded, and one must beware of acidosis.

I firmly believe that the modern surgical interference with the tonsils (tonsillectomy) should always be done in a hospital, where the patient is placed under the best surroundings not only for the carrying out of the operation, but also for dealing promptly with conditions which may arise, either during the operation or subsequently.

My usual procedure is to order a purgative in the case of children the night before, and in the case of adults an enema the morning of the operation. A routine practice of mine, in the case of children, is to prescribe orange juice with glucose in the proportion of 50 grams of the latter to 200 c.c. of the former. Half of this quantity is given to the child the night before, and the balance the morning of the operation. I have found that this moderates to a very great extent the postoperative vomiting and the lessening of acidosis. I believe that an early hour in the morning is the time best suited not only to the patient, but also to the surgeon.

In the case of nervous and apprehensive adult patients a hypodermic injection of morphin ( $\frac{1}{6}$ – $\frac{1}{4}$  gr.) with atropin (1/150 gr.) is given half an



Fig. 170.—Nasal tubes in position.

hour before administering the anesthetic. This serves to quiet the patient and to lessen the amount of mucus secreted during the anesthesia.

**Anesthesia.**—The anesthesia may be: (a) *General* or (b) *local*.

(a) *General Anesthesia.*—The following method of using general anesthesia for the removal of tonsils is described by Dr. W. B. Howell, Chief Anesthetist to the Royal Victoria Hospital, Montreal:

“It is the practice at the Royal Victoria Hospital to use ether in practically all operations for the removal of the tonsils where a general anesthetic is necessary.

“In children under five or six years of age, anesthesia is induced with ethyl chloride given on an open mask, ether being substituted as soon as the characteristic change in the child’s cry, or its breathing, indicates that consciousness has been lost. Older children are given gas at first, instead of ethyl chloride. As soon as fairly deep anesthesia is obtained the mask is removed and the nasal tubes, connected with the ether insufflation apparatus, are inserted (Fig. 170). Unless the patient is well under the influence of ether before the change is made he may partially recover and delay the

operation by coughing or gagging. A mouth hook, inserted into the corner of the mouth, may be used instead of nasal tubes. Sometimes in spite of his receiving the full strength of ether vapor the patient shows, by coughing, swallowing, or retching, that anesthesia is too light. A change from the mouth hook to the nasal tubes, or *vice versa*, may solve the difficulty at once. We use the ether insufflation apparatus devised by Kelly of Liverpool. By means of an electric heater placed in the water-container the vapor can be warmed so that it reaches the patient at about the temperature of the body. This prevents loss of heat during the operation and lessens undesirable after-effects.

"For adults we use the intratracheal method (Fig. 171). Anesthesia is commenced with nitrous oxide, oxygen, and some carbon dioxide (5 to 10 per cent.). As soon as consciousness is lost ether is gradually added and a little later the nitrous oxide is replaced by oxygen. The deep inspirations due to the inhalation of carbon dioxide lead to rapid absorption of ether so that the stage of full muscular relaxation is quickly reached. The mask is now removed from the face, and anesthesia is maintained by means of the



Fig. 171.—Intratracheal tube in position.

mouth hook, connected to the tube of the insufflation apparatus. The use of the mouth hook at this stage may seem unnecessary, but we have learned by experience that it is useful because it eliminates undue haste in introducing the catheter before the patient begins to show signs of recovery. When the patient is completely relaxed the introduction is generally quite easy, but with incomplete relaxation it may be very difficult, and injury to the teeth, pharynx, or vocal cords may be inflicted.

"We have had no cases of pneumonia after tonsillectomy done under intratracheal anesthesia which could in any way be attributed to the method. We decided on one occasion not to use this method for an adult patient because she was the daughter of a member of the staff of this hospital who had a prejudice against it. The operation was therefore carried out under pharyngeal insufflation, and was followed by an attack of apical pneumonia. We felt that this would not have occurred if we had adhered to our usual method. Intratracheal anesthesia greatly facilitates the work of the surgeon by allowing him to operate upon the throat with the least

possible amount of interference with the patient's breathing. It also enables the anesthetist to vary quickly the depth of anesthesia in spite of gagging, coughing, or apnea."

(b) *Local Anesthesia*.—When using local anesthesia I allow the patient to have a light breakfast one hour before the operation. This form of anesthesia is reserved for cases in which a general anesthetic is contraindicated, for such reasons as diabetes in the adult, incipient pulmonary tuberculosis, or if specially selected by the patient.

I prefer novocaine, as it possesses fewer toxic properties than cocaine. The solution used is as follows:

R.	Novocainæ.....	gr. iiss..	0.163 gm.
	Suprarenin boratis..	gr. j....	0.065 gm.
	Aquæ distillatæ.....	℥j.....	30 c.c.

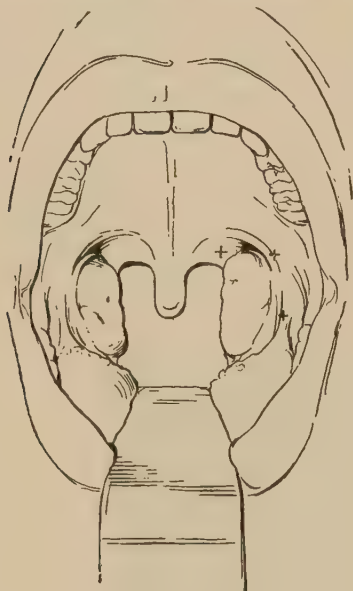


Fig. 172.—Points (+) for injection of local anesthetic.

Of this solution (which is  $\frac{1}{2}$  per cent.) ℥j (for each tonsil) should be injected in equally divided quantities into the points indicated (+) in Fig. 172, by means of the syringe shown in Fig. 173. In making these injections the needle should not penetrate the substance proper of the tonsil, but only the peritonsillar tissue. About fifteen minutes should elapse between the time of the injection and the beginning of operative interference.

I have not found that the introduction of the needle of the syringe is rendered any less painful to the patient by first swabbing with a solution of cocaine the mucous membrane overlying the areas to be injected.

*Position of the Patient*.—Be the anesthetic general or local, my preference is always to have the patient in a recumbent position with a round sand-pillow under the neck, as indicated in Figs. 170, 171.



Fig. 173.—Syringe for local anesthesia.

The following are the operative methods now generally used for the complete removal of the tonsil with its capsule intact:

1. *Guillotine*.
2. *Dissection*.

1. The **guillotine** or tonsillotome is an instrument long in use, and even before the introduction of the operation of tonsillectomy one occasionally

did a complete operation with this instrument when only a tonsillotomy was intended. The tonsillotome of today in its many forms has for its object the complete removal of the tonsil with its capsule intact. Whillis and Pybus<sup>4</sup> and Sluder<sup>5</sup> introduced the guillotine with this object in view. The former use the forefinger of the disengaged hand to force the tonsil through the ring of the instrument, and the latter makes use of the "alveolar eminence" of the lower jaw to accomplish the same object; more recently, however, Sluder has also made use of the finger to push the tonsil through the fenestra.<sup>6</sup>

*Instruments.*—The instruments which I use in performing the operation by this method are depicted in Fig. 174.

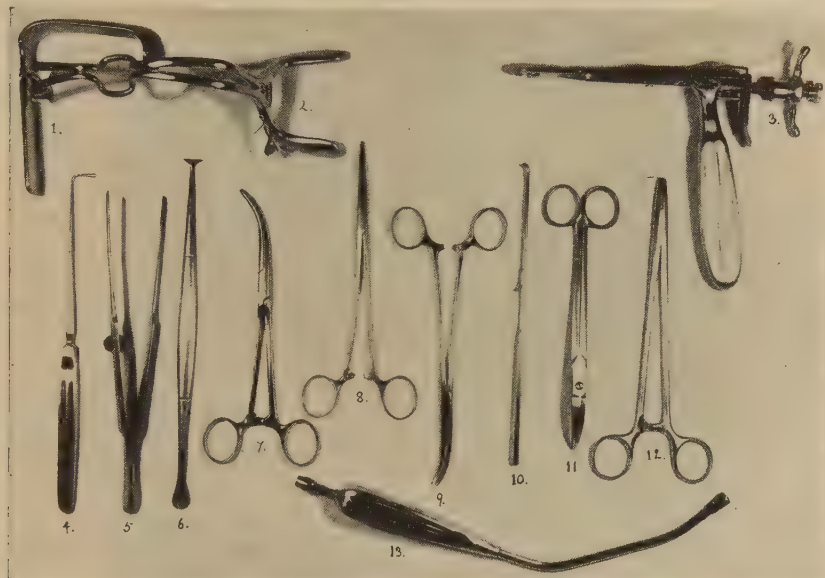


Fig. 174.—Instruments used when performing tonsillectomy by means of the guillotine. The numbers correspond with those in the text.

1. Royal Victoria Hospital tongue depressor.
2. Jennings' mouth-gag.
3. Sauer-Sluder guillotine.
4. Schmidt's tonsil pressor.
5. 8-inch dressing forceps.
6. Hurd's dissector and pillar retractor.
7. Birkett's sponge-holding forceps (six).
8. Birkett's straight artery forceps (four).
9. Birkett's curved artery forceps (two).
10. Knot tier.
11. Prince's curved scissors.
12. Hegar-Mayo needle-holder.
13. Pharyngeal suction-tube.

*Illumination.*—The means of illuminating the field of operation are: (a) Reflected light from the standard laryngological lamp; (b) forehead lamp; (c) the "Le Scialytique" lamp (Fig. 175); this lamp eliminates heat and glare and all shadows on the operating field.

*Precautions.*—Before introducing the mouth-gag, especially in children, one must ascertain that there are no loose teeth which might thus be dislodged and possibly aspirated.

Having gently but firmly depressed the tongue to the floor of the mouth, one should inspect and palpate the field of operation with the object of reassuring one's self that no abnormal distribution of a large artery exists, and that there is no extension of the stylohyoid process into the substance of the tonsil. It is next advisable by means of the tonsil expressor to force out of the lacunæ any cheesy material which may be present, and to wipe it away by means of a gauze sponge on a holder, thus minimizing the possibility of infection of the lungs by aspiration of this material. Any excessive mucus in the pharynx may be removed by means of the suction tube or by sponges.



Fig. 175.—“Le Scialytique” lamp.

*The Operation.*—Greenfield Sluder introduced in the United States of America the operation which bears his name for removing the tonsil by means of the guillotine, and his technic is the one now most generally adopted on the American Continent. The details of the method are those given by Sluder in his recent work on tonsillectomy.<sup>6</sup>

The operator stands at the right, and the assistant at the left of the patient. The mouth-gag is sufficiently opened to obtain a good view of the field, but not so wide as to interfere with respiration. The assistant depresses the tongue by means of the tongue depressor held in his left hand.

The guillotine,<sup>11</sup> with the transverse axis of the aperture downward and outward at an angle of 45 degrees, is passed from the opposite side of the mouth back to engage the lower part of its arc under the lowermost limit of the tonsil. It is then held firmly to the ramus of the jaw. If this

*first movement* (Fig. 176) be done carefully, it will nearly always include a complete separation of the faucial tonsil from its junction with the tongue or lingual tonsil.

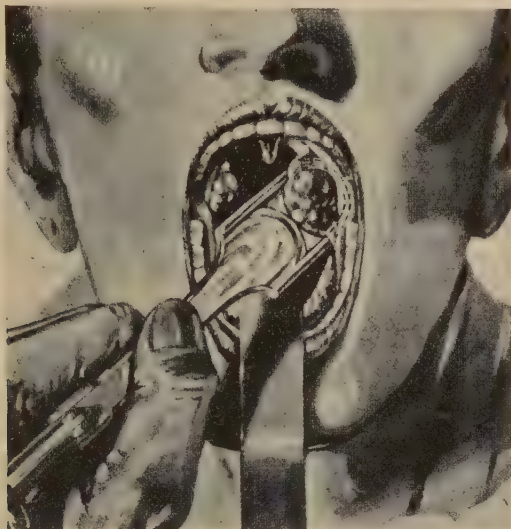


Fig. 176.—First movement. (From Sluder's Tonsillectomy, published by C. V. Mosby Co., 1923.)

“The second movement is performed by raising vertically the aperture of the guillotine while held firmly against the ramus of the jaw. By this movement the entire tonsil is raised in an upward direction.

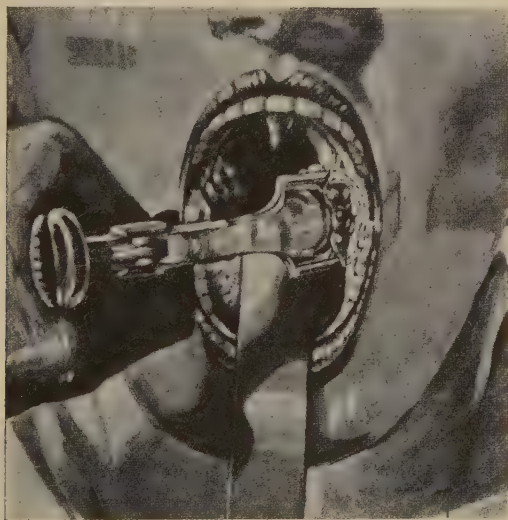


Fig. 177.—Second and third movements completed. (From Sluder's Tonsillectomy, published by C. V. Mosby Co., 1923.)

“The third movement consists in now rotating the upper part of the arc of the aperture outward to definitely include all of the upper part of

the tonsil. By this movement the tonsil has been completely included in the aperture. . . . Figure 177 shows the result of the second and third movements.

"The fourth movement consists in setting accurately the direction of the shaft for the perfect performance of this technic. This is probably the most important of all the movements. All others may be more or less faulty in their execution and still permit the surgeon to do a perfect tonsillectomy in a high percentage of his cases. If, however, his results are to go to the highest percentage (99.6 per cent.) this movement must be uniformly accurate and right. It consists, *roughly stated*, in pointing the shaft to the *shoulder tip* of that side. This movement puts the transverse axis of the aperture parallel to the mylohyoid line and at the same time directs the shaft 45 degrees downward, backward, and about 80 degrees outward.



Fig. 178.—Fourth and fifth movements completed. (From Sluder's Tonsillectomy, published by C. V. Mosby Co., 1923.)

"The fifth movement consists in moving the guillotine slightly forward and upward when the tonsil will be found to be resting in the aperture ready to be pushed through by the finger of the surgeon's unoccupied hand. . . . Figure 178 shows the result of the fourth and fifth movements.

"The sixth movement consists in closing the blade; and should be *carefully* done. After the tonsil has been put through the aperture the finger-tip recognizes the rigid arc of the aperture covered by two layers of mucous membrane one from in front and the other from behind the tonsil. The blade is now pushed gently down and soon is recognized by the finger-tip that is holding the tonsil through the aperture. As soon as its edge is felt the blade with the finger-tip on its edge holding the tonsil through the aperture is kept *stationary* and the closing accomplished by bringing the arc of the aperture *up to it*. . . . It is of very great importance that the guillotine should have been accurately pointed to the shoulder

tip, and that the blade should have been held still while the arc of the aperture was brought up to it and *not* that the *arc* have remained *still* while the blade was pushed *over to it*. . . . When the aperture has been closed satisfactorily under the finger-tip, the finger-tip is in position to be passed over the full extent of the arc and determine whether the tonsil in its entirety be through it. . . . Should he discover that everything has not gone through in the finally closed aperture, the blade is withdrawn 2, 3, or 4 mm. according to the necessities, by placing the tip of the thumb of the hand holding the guillotine under the flange on the distal end of the blade. The finger which has put the tonsil through, remaining in its position, at once recognizes the opening of the aperture and then pushes the tag through. As soon as the surgeon is satisfied that everything is in readiness for cutting the tonsil off or out," he requests the assistant with his disengaged hand to turn the screw rapidly downward.

A large gauze swab is immediately introduced into the fossa by means of a sponge holder, and firmly held there by the assistant for a few moments to arrest any hemorrhage. After removal of the swab, if any hemorrhage is still present, its origin is to be carefully sought. The most frequent sites are in the region of either the upper or lower pole, and the floor of the fossa. By means of the pillar retractor (Fig. 174<sup>1</sup>) the upper pole is easily exposed by drawing the arch of the pillars upward, and similarly the lower pole is exposed by retracting the anterior pillar. Should, however, the bleeding be found to come from the floor of the fossa, this is best exposed by retracting the anterior pillar, while the assistant grasps the posterior pillar by means of the long pair of dressing forceps, and draws it inward toward the midline of the pharynx, thus making the floor of the fossa a flat surface (Fig. 179). The point of the hemorrhage is thus more easily visible than when the floor lies more deeply embedded and partially folded.

The bleeding points are at once secured by either straight or curved forceps (Fig. 174<sup>3, 4</sup>) as the situation requires. Arterial or venous hemorrhage of any moment should, in my opinion, be arrested by ligation rather than by temporary compression. The method of ligation is shown in Fig. 179.

*Before proceeding to the removal of the other tonsil it is essential that all bleeding should have been completely arrested, after which the fossa should be packed with a strip of gauze 1½ inches wide, the proximal end of which should be drawn out to the angle of the mouth on the same side and slipped under the side of the mouth-gag. In the adult, in whom I use intratracheal anesthesia, that portion of the tube in the oropharynx can be moved to the side which has been operated upon, and it will thus keep the gauze in position.*

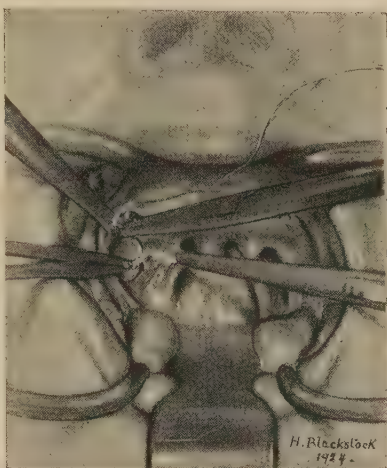


Fig. 179.—Method whereby the floor of the fossa is exposed, searching for any bleeding point.

The other tonsil is dealt with in the same manner except that the guillotine is held in the opposite hand.

*It is absolutely essential in order to minimize the possibilities of immediate or secondary hemorrhage that the patient be removed from the operating table only with a dry field.*

2. **Dissection.**—This method of operation, which also has for its object the removal of the tonsil with its capsule intact, was placed on a satisfactory basis by Waugh<sup>7</sup> in 1909, and has since been modified in very small details only.

The remarks made under the headings such as Preparation of the Patient, Anesthesia, Position of the Patient, and Illumination, apply equally to this method of tonsillectomy.

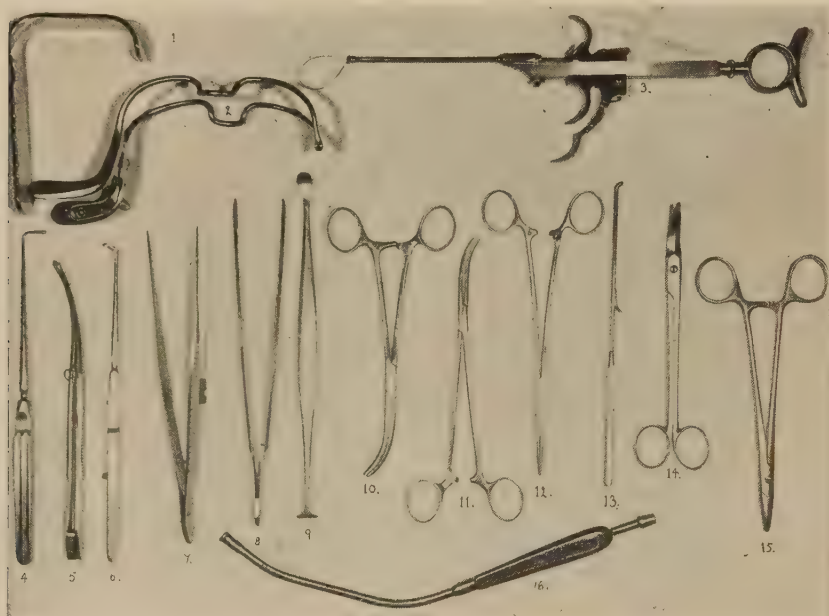


Fig. 180.—Instruments used in the dissection operation for removal of tonsils. The numbers correspond with those in the text.

*Instruments.*—(Fig. 180).

1. Royal Victoria Hospital tongue depressor.
2. Jennings' mouth-gag.
3. Beck-Muller snare.
4. Schmidt's tonsil pressor.
5. Tilley's grasping forceps.
6. Douglas' knife.
7. Waugh's dissecting forceps.
8. 8-inch dressing forceps.
9. Hurd's dissector and pillar retractor.
10. Birkett's sponge-holding forceps.
11. Birkett's curved artery forceps.
12. Birkett's straight artery forceps.
13. Knot tier.

14. Prince's curved scissors.
15. Hegar-Mayo needle-holder.
16. Pharyngeal suction-tube.

*The Operation.*—The operator stands at the right with the assistant at the left of the patient, and, the mouth-gag being in position, he thoroughly exposes the field of operation by means of the tongue depressor (Fig. 180, 1), observing the same *precautions* as noted under the method of operating with the guillotine (p. 302).

By means of the tongue depressor now held in the assistant's left hand the tongue is pressed firmly to the floor of the mouth, and the anterior pillars are thus brought into relief; at the same time the assistant holds up the chin with the little finger of the same hand to prevent the jaw from dropping, and to make the airway freer. The tonsil is seized by the grasping forceps held in the left hand of the operator, and is gently drawn toward the midline of the oropharynx. The knife (Fig. 180, 6), held in the left hand of the operator, is made to enter the plica well behind the left anterior pillar, at the lower pole, and is carried forward to the posterior pillar just beneath the mucous membrane covering the tonsil (Fig. 181). The assis-



Fig. 181.—Shows the tonsil grasped by the forceps and the knife entered behind the anterior pillar, releasing it from its attachment to the tonsil. The dotted line shows the continuance of the incision.



Fig. 182.—Shows the capsule exposed.

tant with pillar retractor in his right hand draws back the anterior pillar, thus exposing the vascular areolar tissue which, by means of the long fine dissecting forceps (Fig. 180, 7), or a very small gauze sponge held in a pair of forceps, is gently broken through in a line parallel to the anterior pillar, thus bringing into view the glistening, bluish-white capsule (Fig. 182). Close contact with the capsule is now necessary. The operator, working upward, continues to separate this loose areolar tissue by pushing it outward; and in this manner the upper pole is readily exposed and then displaced downward by the grasping forceps. In the place of the pillar retractor the assistant now introduces a small gauze sponge held in a sponge holder, into the upper pole of the fossa which is made vacant by

the displacement of the tonsil (Fig. 183). This sponge serves a double purpose; it acts as a retractor of the tissues of the fossa, and secondly by keeping the tissues, still attached to the capsule, on the stretch, it enables the operator to discover any vessel, which he should then carefully push to one side, or if necessary seize with the artery forceps and ligate. Should the oozing be more than the sponge will hold, the overflow is readily removed by means of the suction tube which the anesthetist is always in charge of. In this way the field is kept free of blood. The tonsil is now separated from its remaining attachments to the posterior pillar, leaving it attached only by its inferior portion to the lower part of the fossa. At this stage the wire loop of the snare (Fig. 184), is passed over the grasping forceps down to the pedicle and then tightened as soon as the operator is assured that the lower pole of the tonsil is well within its grasp. The operator allows the forceps which is still holding the tonsil to lie across the patient's mouth, while with his disengaged hand he *slowly* tightens the

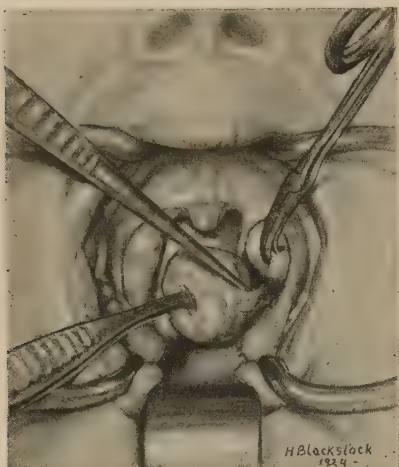


Fig. 183.—A sponge on holder introduced into the upper pole of the tonsillar fossa, acting as a retractor and controlling any bleeding.



Fig. 184.—Shows the wire loop of the snare being applied to the pedicle of the tonsil.

wire by means of the screw, thus removing the tonsil completely. The bed of the fossa now being thoroughly exposed, is treated as already described when the tonsil is removed by the guillotine. The other tonsil is now removed in the same way, except that the operator uses the pillar knife with his right hand, and the pillar retractor with his left, while the assistant grasps the tonsil with the forceps. One must not proceed to the removal of the opposite tonsil until quite satisfied that all sources of bleeding have been arrested, and it must here again be emphasized *that the patient should not be removed from the operating table until the field is quite dry.*

**Hemorrhage.**—After removal of tonsils by either of the above methods there may be hemorrhage of a *primary* or *secondary* nature.

*Primary* hemorrhage is that which occurs at the time of the operation, or within a few hours after. The evidences of hemorrhage after the patient has been returned to bed are manifested by the patient frequently swallow-

ing, rapidity of the pulse, pallor, and the vomiting of quantities of bright colored blood. Such a condition demands immediate inspection of the throat under good illumination. With the *very* exceptional child this is possible, and on examination a blood-clot of varying size will frequently be found in the area in which the bleeding is taking place, and around the base of the clot will be noted the freshly escaping blood. The clot should be removed, and the bleeding-point grasped with a pair of long artery forceps, and a ligature slipped on. Most children, however, in my experience are not submissive to such treatment without anesthesia, and when such is the case, then I believe the most satisfactory way of dealing with such hemorrhage is to return the child to the operating table, and under general anesthesia thoroughly attend to the situation. Adults, on the other hand, are more reasonable to deal with, and one can *occasionally* govern the situation without proceeding to general anesthesia. If the hemorrhage is found only to be an *oozing*, the administration hypodermically of 1/12 gr. morphine for a child, and  $\frac{1}{4}$  gr. for an adult, will often quiet the pulse and the restlessness of the patient, and with these two conditions modified the oozing will frequently stop.

*Secondary* hemorrhage usually arises any time from two to ten days after the operation, due to a detached slough, or an erosion opening a vessel. Such a condition calls for immediate ligation of the bleeding-point. The clot in such cases is often large, and great care must be taken in the moving of the patient, as any exertion on his part may cause the sudden dislodgment of the clot and its possible aspiration.

The bringing together of the pillars of the fauces, either by sutures or clamps, over a roll of gauze placed in the fossa for the purpose of arresting hemorrhage has never appealed to me as a desirable method.

Cases of severe hemorrhage or oozing which have not responded to the usual methods of arresting it, will require intravenous injection of horse-serum or calcium chloride.

In the Royal Victoria Hospital during the years 1926 and 1927 in 2984 tonsils and adenoid operations, primary postoperative hemorrhage occurred in 0.4 per cent. of the total number of operations. Secondary postoperative hemorrhage occurred in 1.1 per cent. of the total number of operations.

**After-treatment.**—The patient should be put to bed, lying flat and on one side, so that the secretions and blood accumulating in the throat instead of being swallowed may find their way out by the mouth. An ice-collar should immediately be applied and worn continuously for twenty-four hours. Most patients in my experience find this comforting. My usual instructions are that the patient shall have nothing for twelve hours except cold, sterilized water, and after that time liquid or semiliquid nourishment is ordered according to the patient's taste, such as orange juice, jellies, ice-cream, milk, beef-tea, and chicken broth. Talking and clearing of the throat should be avoided. On the second day a purgative is advisable. At this time pain in the throat or ears (reflex) is often complained of, and relief is given by irrigating the throat with Dobell's solution made hot. This of course can only be used by the adult; children as a rule complain little of sore throat.

Twenty-four hours after operation usually there appears a thin, white membrane covering the fossæ, which has been mistaken for diphtheria. It

is well, therefore, that patients should be informed of the possible occurrence of this false membrane, and thus saved considerable anxiety later on.

The patient should not return to his usual duties until ten days subsequent to the operation, especially in the case of children going to school.

Singers require a long rest to allow of the complete healing of the wound, and their lessons or exercises when resumed should be of short duration at first, and gradually lengthened, because the conditions under which they now use the voice are very different, and any attempt to begin singing where they left off is very apt to be a partial failure at least, and necessarily a disappointment, and owing to their rather sensitive and impressionable nature, they are apt to become discouraged at once.

*Pulmonary complications following tonsillectomy* are treated elsewhere (See p. 316).

*Morcellement* and the *galvanocautery snare* as a means of removing tonsils have nothing to commend them, and I have long discarded their use in favor of the methods already described.

#### THE USE OF RADIUM AND ROENTGEN RAYS IN THE TREATMENT OF HYPERTROPHIED TONSILS AND CHRONIC LACUNAR TONSILLITIS

The following are the methods of the use of radium and Roentgen rays in the treatment of hypertrophied tonsils and chronic lacunar tonsillitis as recommended and described by Dr. A. Howard Pirie, physician in charge of the Roentgen and Radium Department of the Royal Victoria Hospital, Montreal:

1. **Action of Radium on Tonsils.**—"Operation is the only means of removing completely a tonsil. Roentgen rays and radium do not remove tonsils, they cause atrophy of them. Normal adult healthy tonsils are atrophied tonsils and it is imitation of nature's own method which is aimed at when radium and Roentgen rays are used. Adult tonsils when diseased are frequently hypertrophied. Radium acting on such tonsils causes atrophy of them with disappearance of the disease. A similar action takes place on the hypertrophied healthy tonsils of children. Different degrees of atrophy can be produced up to a certain point, but a maximum degree of atrophy is eventually reached, after which radium has no further action.

"The microscopical changes seen in a tonsil after proper use of radium are: (1) Increase of fibrous tissue; (2) decrease of lymphatic tissue; (3) disappearance of the follicles; (4) decrease of lymphocytes.

"The amount of radium used and its duration of exposure govern in general the degree of atrophy produced. If the exposure has been such that only slight atrophy has been produced, further atrophy (short of the maximum atrophy possible) can be produced by further radium treatment.

"The success of the treatment depends largely on the skill of the radiologist. His object is to get as equal a dose as possible delivered to the whole tonsil. A 30-mg. unit of radium contained in a brass capsule 1 mm. thick causes an active effect as shown in Fig. 185. The capsule is 2 cm. long and  $\frac{1}{2}$  cm. broad. It is laid on photographic paper for different lengths of time viz.: A, three minutes; B, twelve minutes; C, twenty-four minutes. It shows how radium laid on a tonsil for too short a time as in A causes only slight effect whereas in C it causes a general effect. D shows the effect of four radon seeds of 1.5 mc. strength, and E the effects of six seeds of strength  $\frac{1}{2}$  mc. The effect in E is better than that shown in D, as a more equal effect

is obtained, and smaller areas of local necrosis would follow. F shows the effect of a single radon seed.

"In actual use it has been found that a decided atrophic action can be obtained on the tonsil whether surface application or burial of radium is used."

*Surface Application.*—"For enlarged tonsils of children about 200 mc. minutes with a filter of aluminium 0.58 mm. thick applied at intervals of two weeks for three applications has been found sufficient to cause marked atrophy.

"For tonsillitis with arthritis due to absorption from tonsils, from three to eleven similar treatments have been found necessary. Another successful method is to use a plaque, oval in shape, and 2 square cm. in area over which not more than 30 mg. of radium is spread having a covering of rubber composition. The plaque is kept on the surface of the tonsil by a compass-shaped hemostat for thirty minutes. In this case the action of

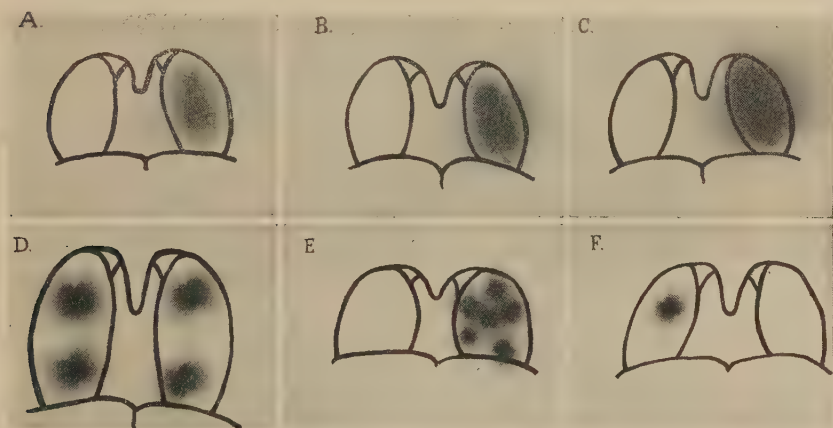


Fig. 185.—Experimental demonstration of the effect of radium of different quantities and strength on tonsils.

the beta rays is made use of. Three treatments are made at intervals of three weeks. Saliva is removed by a dental suction apparatus."

*Burying Needles of Radium.*—"Two needles of platinum containing a total of 30 mg. of radium can be buried for two hours in each tonsil. This will cause slight reaction. The treatment may have to be carried out three times at intervals of two weeks.

"Another method is to bury two radon seeds in each tonsil. Each seed should be  $\frac{1}{4}$  mc. in strength. More seeds can be buried after six weeks if sufficient effect is not obtained from the first seeds. The tonsil is painted with 2 per cent. cocaine before implantation."

*Clinical Results.*—"Where tonsillectomy is contraindicated the use of radium can be recommended, as in hemophilia, certain chronic heart, kidney or lung diseases, or where operation is refused. Total disappearance of the tonsil is not to be looked for, but simply marked reduction in size, as the fibrous tissue of the tonsil does not atrophy to any extent."

**2. Action of Roentgen Rays on the Tonsils.**—"The action of Roentgen rays on tonsils is the same as that of the gamma rays of radium. The main

difference is that radium can be applied inside the mouth, whereas Roentgen rays have to pass through the tissues of the neck before reaching the tonsil."

*Treatment of Enlarged Tonsils in Children.*—"Tonsils which nearly meet in the middle line in children shrink under the action of Roentgen rays till they become nearly but not quite flat on each side. They can never completely disappear as their fibrous tissue never disappears although their lymphatic tissue does so. The writer has seen such a result in a hemophiliac patient after thirteen treatments at intervals of a week to two weeks. If there is no skin reaction, treatment is given every week for the first seven treatments and then every two weeks for five more treatments. The method of treatment is shown in Fig. 186, and the electrical constants are as follows: Skin focus distance 15 inches; milliamperes 8, voltage 70,000; time seven minutes; filter 3 mm. of aluminium. Treatment of enlarged tonsils



Fig. 186.—Method of treating tonsils by Roentgen rays.

in children is indicated when operation is refused, in hemophilia, and certain chronic heart, lung, or kidney diseases."

*Treatment of Septic Tonsils in Adults.*—"Cases of arthritis and recurrent attacks of tonsillitis are successfully treated by Roentgen rays. The writer has treated 70 cases during the last three years. No bad results have been reported. The size of the tonsils after the treatment has diminished and the effects of treatment have been disappearance or lessening of arthritic pain, fewer or no attacks of tonsillitis.

"Dryness of the throat is the only unpleasant result of the treatment, but it has not been constant or excessive. The treatment is given every

week till the slightest degree of reaction is noted on the skin, and then the treatment is given every two weeks till twelve treatments have been given.

"The treatment by radium is inconvenient and troublesome to carry out, while that of Roentgen rays is easy, causing little or no inconvenience."

HERBERT S. BIRKETT.

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#### COMPLICATIONS OF TONSILLECTOMY

The operation of tonsillectomy is in the great majority of instances followed by uneventful convalescence. For this reason such complications as do arise are all the more striking and worthy of notice. Their recognition and treatment are matters with which every laryngologist should be familiar since their relative rarity is no guarantee against their unexpected occurrence. Prompt measures for dealing with them may prove of inestimable value.

**Shock.**—A condition of surgical shock may follow tonsillectomy either as a result of undue length or difficulty in the operation, of undue loss of blood, or even in the absence of these causes. A rapid pulse-rate at once suggests hemorrhage as a factor, in which case measures for the control of the bleeding are of first importance.

In the absence of bleeding postoperative shock is manifested by a cold clammy skin, a weak pulse, and a lowered blood-pressure.

For this condition heat is the best remedy, applied all about the body, together with elevation of the foot of the bed. Hot coffee by mouth or rectum will promote stimulation following which hypodermic injection of morphine is helpful. In shock following hemorrhage intravenous or subcutaneous injection of saline is of great value provided all bleeding has been controlled.

Ether lowers body temperature, and patients who must be conveyed some distance after operation should be protected by warm blankets from all possible loss of body heat. Hospital garments are frequently none too warm, a fact which, especially in cold weather, should be borne in mind by both nurses and doctors.

**Hemorrhage.**—A hemorrhage may be said to exist when active bleeding begins some minutes or hours after a patient has left the operating room with complete hemostasis. In children bleeding may continue for some time without detection since they are prone to swallow the blood which an

adult readily expectorates. Since ether and even local anesthetics tend to lower blood-pressure it is perfectly possible for subsequent bleeding to occur even when the patient left the operating table free from hemorrhage.

The mode of procedure will vary somewhat in accordance with the severity of the bleeding, which may be considerable in amount or only a slow but persistent ooze. An immediate hypodermic injection of morphine ( $\frac{1}{8}$  to  $\frac{1}{4}$  gr.—.008 to .015) will tend to quiet an otherwise frightened and intractable patient, will slow the heart action, and help to favor coagulation. A careful examination of the throat should next be made for the source of bleeding, whether from a broad oozing surface or from a single spurting vessel. Should the tonsillar fossa be filled with a clot it should be wiped away with a pledget of gauze or grasped with sponge-forceps and removed. Such a clot has no hemostatic value and only favors the bleeding which is going on beneath it. Removal will permit the underlying source of bleeding to be dealt with either by grasping the oozing point with a broad hemostat and applying a ligature or by simply leaving the clamp *in situ* for a few hours. There is no pain after the first application of the hemostat, the mouth can be closed over it, and a pillow so placed as to take off all pressure from the instrument.

In fossæ with broad prominent pillars mild oozing can sometimes be controlled by a gauze sponge with a string attached and so placed as to be grasped and held in position by the pillars themselves. Attach a hemostat to the string. It is easily removed and free from danger.

The above described measures are applicable to patients in bed without resorting to removal to the operating room. Should further measures for the control of active bleeding be necessary, no time should be lost in anesthetizing and placing the patient again on the operating table. A second operation which successfully checks bleeding is far preferable to temporizing measures which do not.

With the patient adequately anesthetized a careful and tranquil search may be made for the source of bleeding which is then best controlled by grasping with a hemostat about which is placed a figure-of-8 catgut suture. On removal of the clamp the suture can be tied and will completely control any bleeding within its grasp. A stout full-curved needle of fair size and a good needle holder are essential. I use a curved Pratt rectal forceps and a Von Eiselberg needle holder for this work. The procedure of sewing a sponge between the pillars is not to be recommended, since it predisposes to sepsis, requires subsequent removal, and tends to produce deformity and adhesions. Except when direct suture of the vessel itself is fraught with insurmountable difficulty, as, for instance, deep down at the base, any form of pillar suture is to be avoided. Portions of tissue around the bleeding-point may be caught with a needle and sewed about it in a purse-string fashion with excellent results.

Occult bleeding with swallowed blood in children must be suspected when there is a steady rise in pulse-rate, undue restlessness, or the vomiting of any quantity of bright blood. Any such signs should lead to immediate inspection of the throat.

Tonsillar hemorrhage may prove a serious matter and patients must not be left without skilled supervision until it is certain that all bleeding has ceased. All operations should be performed in a hospital and the

patient should remain at least overnight in the case of children and for two to five days in the case of adults.

A delayed or secondary hemorrhage may occur on the fifth or sixth day after operation. Examination almost universally shows a clot, the removal of which usually checks the bleeding which is apt to be less severe and more easily controlled than primary bleeding. Rarely will any form of suture be required.

**Sepsis.**—True postoperative sepsis following tonsillectomy rarely occurs in spite of the freshly traumatized surface and the constant presence of bacteria in the mouth cavity. Actual infection is manifested by a considerable rise in temperature, the usual constitutional symptoms, and locally by a boggy swollen area around the site of the removed tonsil. As there is no obstacle to drainage this sepsis usually runs a short course, but the condition of the patient may appear rather alarming for several days. Treatment is usually ineffective, but quinine or aspirin at the onset are helpful. The routine measures for combating any systemic infection should be tried.

As the throat will probably be unduly sore and swallowing very difficult, external applications are indicated. These should consist of hot compresses soaked in normal saline solution, and covered with oiled silk and a gauze bandage, the whole being changed as often as the saline dries or becomes cold. Any tendency to cervical adenitis is particularly amenable to this form of compress. Urine examinations and white-blood counts should be made from time to time. Intra-orally the usual gargles or applications may give some relief, but the pain may render these of little aid. Dysphagia will always be marked. Dyspnea to the point of requiring any measures for relief is not to be expected.

**Pain** following tonsillectomy is a relative sensation and varies greatly with the age and temperament of the patient. As a rule children complain but little while adults are apt to suffer more or less for several days.

Pain is due to the presence of an open wound which has no dressing other than mouth secretions, and over which air and whatever enters the mouth must pass. Furthermore, the action of the palatoglossus and palatopharyngeus muscles produce pain during the act of swallowing, greater or less in amount according as these muscles were traumatized during the operation.

The pain described is of two types, a general ache of all throat structures intensified by deglutition, and an ache accompanied by pain radiating up to the ear. A true middle ear infection is rare in adults, but children, from whom adenoids have been removed, may have a severe otitis media, at times requiring myringotomy and occasionally leading to mastoiditis.

Relief of severe postoperative pain is best accomplished by small doses of morphine during the first few days of convalescence. A helpful gargle can be prepared by dissolving 20 grains of aspirin in a glass of water, and patients should be urged to use this at least every hour, swallowing a portion after the gargling. This often controls the pain so that nothing further is needed. External applications of hot saline compresses frequently changed will afford some relief.

**Edema** of the uvula and adjacent portions of the soft palate is quite common, especially in those cases where the tonsil lay high up in the supratonsillar fossa. This edema adds somewhat to the discomfort of swallowing,

but is self-limited and requires no special treatment. Astringent applications or lozenges will give some relief.

**Cervical Adenitis.**—Though tonsillectomy is often performed for the relief of adjacent glandular swelling, the latter sometimes appears postoperatively when not previously present. This is due to a mild degree of septic absorption from the wound area which is drained by the tonsillar lymphatics. Such an acute adenitis will soon subside and no treatment is indicated. Where the reduction of previously existing glandular swelling is the objective an ample period of time must be allowed before complete resolution can be expected.

**Alterations in the Voice.**—The question of any postoperative injury to the voice is one of utmost importance to those who gain their livelihood by speaking or singing. There is no doubt but that, for a time at least, the quality of the voice is altered following tonsillectomy, since the surrounding muscles which have much to do with vocal quality are temporarily disturbed. This is usually transitory and as time goes on most persons, even professional singers, find the voice improved, though any promises as to this result should be made with caution. Those patients to whom the quality of the voice is of moment should be encouraged to use it very early in convalescence and singers should begin practice while the throat still feels stiff and before contraction of the palatal muscles begins.

**Stiffness of the Pillars.**—After tonsillectomy there is left an open space bounded by the palatoglossus and palatopharyngeus muscles respectively, anteriorly and posteriorly and externally by the superior constrictor of the pharynx. A certain amount of contracture of these muscles, particularly of the first, gradually takes place, a condition which, especially in thick-set, short-necked individuals, may be quite disagreeable and a source of complaint. The patient accustoms himself to this in time and no treatment is required.

**Slow Convalescence.**—In adults a tonsillectomy is always a major procedure and is often followed by great general relaxation and a slow return to normal health and strength. This may occur in cases where the operation has presented no particular difficulty and where normal healing of the wound has followed in the usual time and without complications. Under these circumstances patients should be kept in bed until convalescence is thoroughly established, and their condition watched for some time thereafter. They can be assured that in the end only good will come from the operation.

GEORGE L. RICHARDS.

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## SUPPURATIVE DISEASE OF THE LUNG AS A COMPLICATION OF TONSILLECTOMY AND OTHER OPERATIONS ON THE UPPER AIR-PASSAGES

**Definition.**—The term “pulmonary abscess” signifies single or multiple collections of pus within the lung tissue. This condition usually is secondary either to the entrance of foreign bodies into the bronchi or to operations in any part of the body. Lung suppurations due to tuberculosis, bronchiectasis, parasites, etc. are usually not included in this group. To

otolaryngologists lung abscess became a live entity in 1912, when Charles W. Richardson<sup>1</sup> called attention to it as a postoperative complication following tonsillectomy.

**Frequency.**—It is not unfair to state that the published figures as to the frequency of this disease are necessarily but approximations of the true situation. Many cases are quite unrecognized, some being regarded as "heavy colds," some undoubtedly being incorrectly diagnosed, and many not coming to the knowledge of the operator. An otolaryngologist or a clinic may report, for example, 15,000 cases of tonsillectomy with one, two, or even no postoperative lung complications. A report of this kind is unenlightening and of little value. All that this report means is that few or no cases have come to the attention of the operator or clinic. It will not be until a very large series of operative cases is subjected to a painstaking postoperative "follow-up" that the true morbidity will be known. Cutler and Hunt<sup>2</sup> report from a large general surgical clinic that 3.93 per cent. (approximately 1 in 25) of the patients operated on in 1920 "developed a pulmonary complication that might be attributable to the operative intervention or to the anesthetic." In the light of such carefully collected figures as these, especially as but a small part of the surgery reported upon was done in the upper respiratory tract, it is irrational to believe that any otolaryngologist can perform many thousands of tonsillectomies with one, two, or no subsequent pulmonary lesions.

In the past four years approximately 950 cases<sup>3</sup> of lung abscess have been reported by observers in this and other countries. Almost 50 per cent. of these have occurred subsequent to tonsil removal. Among the reports at hand, in three the author states the frequency of post-tonsillectomic lung-abscess. Moore<sup>4</sup> makes the statement that it occurs once in from 2500 to 3000 tonsillectomies. Keiper<sup>5</sup> says that it occurs in 1 out of every 781 cases. Hedblom<sup>6</sup> states that in 692 cases of pulmonary abscess observed at the Mayo Clinic 21 per cent. followed operative procedures. Of these postoperative abscesses 31 per cent. followed tonsillectomy, 26 per cent. laparotomy, and 9.5 per cent. tooth extraction.

However, interesting as these figures are, it is the belief of the present writer that it is useless to quote them except for the purpose of pointing out the alarming frequency of the condition.

**Morbid Anatomy.**—The lung's periphery is the most frequent site of abscess formation, although centrally situated lesions are quite common. The size varies from a diameter of a centimeter to involvement of an entire lobe. A reactionary area, varying in appearance with the duration, size, and virulence of the abscess, surrounds the latter. Over a peripheral abscess adherent or some other form of pleurisy always is present.

In patients with a high degree of resistance a small abscess may terminate by absorption with formation of scar-tissue. The larger collections usually drain spontaneously into a larger bronchus and discharge their contents by that route. Sometimes the pleura is eroded with a resultant pyothorax or pyopneumothorax. When resistance to infection is low and virulence high, death from sepsis is the usual outcome.

**Location.**—These abscesses may occur in any part of the pulmonary structure. No lobe is immune. One observer<sup>7</sup> found the condition to be present in the upper lobes oftener than elsewhere. The majority,<sup>8</sup> however, report that the lower lobes are the ones most frequently affected. Prac-

tically all agree that the right side is more commonly abscessed than the left. If the available data from all reported cases is summarized we find that the right lower lobe has most often been so diseased. Bearing this in mind, it is interesting to note that this same area is most frequently involved in lobar pneumonia<sup>9</sup> and in postoperative pulmonary embolism.<sup>10</sup>

Cutler<sup>11</sup> found postoperative lung lesions to be located as enumerated in the subjoined tabulation. He had accurate data on 40 of his cases. Of these 21 were in the right lower lobe only and 9 were in the left lower lobe (21 to 9); 30 of the cases involving more than one lobe were located in this right lung and 13 in the left lung (30 to 13).

	1920.	1921.
Right upper lobe.....	0	2
Right middle lobe.....	2	1
Right lower lobe.....	14	7
Left upper lobe.....	0	0
Left lower lobe.....	5	4
Both lower lobes.....	2	1
Right upper and middle lobes.....	1	0
Left upper and lower lobes.....	1	0
Totals.....	25	15

In recent years closer study of postoperative pulmonary complications following any sort of surgical procedure under general anesthesia has shown us that many conditions which were formerly considered ether pneumonias are, in reality, septic or non-septic pulmonary infarctions. If they are septic, abscess usually follows. When we remember that the right lower lobe is the thoracic quadrant most frequently involved in lobar pneumonia or pulmonary infarction it is reasonable to expect it to be most often involved when abscess is the result of septic embolism.

**Mode of Infection.**—This has been a matter of much debate. The factors concerned are many—too many to be enumerated in full. The condition of the patient, both local and general; the anesthetic; the details of operative technic; the postoperative treatment; all have been given consideration. There, too, can be observed a slight tendency on the part of some of the fortunate ones to blame the other fellow's technic.

What is the pathway of infection? This is a question which has been the subject of much discussion. Infection by way of the trachea and bronchi—for a time the most obvious possibility—was the dominant and almost the only theory. More recently attention has been directed to infection by way of the venous channels. Lymphatic infection—the third possibility—has never had ascribed to it a rôle of any importance, largely because such a route, including as it does a series of lymph-nodes, would be a slow one and would be accompanied by a noticeable cervical adenitis.

The common route must be one of two—tracheobronchial or venous. Recently the writer, in collaboration with Dr. Herbert Fox,<sup>12</sup> reviewed the literature of postoperative lung complications in general, and in connection therewith conducted some experimental studies. This was done in the hope of securing data which would shed light on the problem. In the study of the operation area, following experimental tonsillectomy, it was found that "there are present hemorrhages, thrombi, necroses, and bacteria. Hemorrhage causes rifts in the tissues, opens pathways for infection and destroys tissue. Trauma and disturbed blood-supply result in necrosis. Injured blood-vessels are closed by coagula which may become

infected and loosened. Bacteria are carried into the tissues by physical force or are permitted to penetrate by the opening up of tissue clefts, especially when there has been some devitalization. These are present in an area which never can be put at rest, which constantly is open to reinfection, and which is subjected to a great deal of constrictor muscle action.

"What might be the consequences? Should a sterile thrombus be dislodged into the blood the result would be a small sterile pulmonary infarct which, however, later might become infected and give rise to pneumonia or an abscess. Should a septic thrombus be dislodged a lung abscess would be the probable result. Should a thrombus, either sterile or septic, reach a previously diseased area of the lung, such as a tuberculous focus, the lesion might be activated."

In the light of our present knowledge it can be stated that the evidence favoring the tracheobronchial route is as follows:

1. The obvious exposure, at operation, of these tissues to the entrance of blood, and septic mouth and tonsil contents.

2. The established fact of the frequent entrance of these substances into the trachea during tonsillectomy.

3. The fact that these abscesses locate in the various lobes of the lung about as do inspired foreign bodies.

Opposed to the acceptance of the tracheobronchial as the main or only pathway, and inferentially pointing toward the venous channels as the probable route are:

1. The well-known resistance of the lung to inhaled micro-organisms, as is evidenced, for example, by the difficulty of producing pulmonary infection by the introduction of micro-organisms into the trachea.

2. The fact that pulmonary emboli, especially massive ones, are found in the right lung in even a larger proportion than are foreign bodies.

3. The proved presence of numerous thrombi in the walls of the tonsil fossa.

4. The demonstrated presence of bacteria in these same tissues.

5. The fact that the three main factors in the formation of thrombi and their dislodgment into the circulation, trauma, sepsis, and muscular action are present to a notable degree in the operation field.

6. The fact that many cases have followed tonsillectomy under local anesthesia.

It would be difficult in any one case to determine how the lung became infected. A sane attitude to adopt would be that the prevalent pathways are the venous and the tracheobronchial. Which of these is the preponderating one it is impossible to state; the logic of the situation seems to the writer to point toward the former.

**Symptoms and Diagnosis.**—In all postoperative cases, especially when tonsillectomy has been performed, in which cough with or without expectoration is present, lung embolism or lung abscess should be suspected. In the average case the symptoms appear seven to ten days following operation, although they have been noted as early as the first day and as late as the fourth week. Chills, fever, rapidity of pulse and respiration, leukocytosis, expectoration, foul breath, and a bad taste may be present, depending upon the extent and virulence of the process. If the periphery of the lung is involved, pain in the chest is usually present. The physical signs are not characteristic and they vary with the location, the size, and

the condition of the abscess. In all suspected cases a Roentgen-ray examination is essential. Too often is the diagnosis of tuberculosis made when lung abscess is present. When expectoration is abundant, purulent, and without tubercle bacilli, the diagnosis is almost certainly not tuberculosis.

**Prophylaxis.**—Regardless of the route by which lung infection is established there are certain well-defined principles which should be observed in connection with tonsillectomy and other operations on the upper respiratory tract:

1. No operation, unless emergency in nature, should be performed in the presence of an acute respiratory tract infection or within two weeks after the subsidence of such infection.

2. A cleansing toilet should be made of the nose and mouth before operation.

3. All aseptic precautions should be observed during operation.

4. The teeth should be carefully examined before the mouth-gag is inserted, so as to guard against the dislodgment of any that are loose with subsequent entrance into the bronchi.

5. All possible care should be exercised to prevent the entrance of blood and throat secretions into the larynx and trachea.

6. All unnecessary trauma should be avoided, thus helping to prevent the formation of superficial necroses and septic thrombi which could give origin to septic emboli.

7. For several days subsequent to operation the patient and the throat should be given the greatest possible rest in the effort to prevent the conversion into emboli of thrombi necessarily produced in the peritonsillar area at the operation.

**Treatment.**—Close co-operation between the internist, roentgenologist, bacteriologist, bronchoscopist, and surgeon is necessary in order to adequately and thoroughly treat these cases.

1. *Medical.*—This form of treatment is usually first given in these cases. The aim of this form of therapy is to aid nature to overcome the infection, and bring about a spontaneous cure. The essential features are rest in bed, adequate diet, and abundant fresh air and sunshine. Mechanical aid to drainage by means of postural methods are worth the trial. Autogenous vaccine therapy should always be used when uncontaminated pus from the abscess cavity can be secured.

2. *Endobronchial.*—Aspiration, irrigation, and medication of the abscess cavity has given good results in the hands of a few. Quite frequently this maneuver is practised as an aid to the medical treatment outlined above.\* This procedure should be attempted only by a group thoroughly trained in endoscopy and then only in selected cases. Moore and Lukens,<sup>13</sup> who report 25 per cent. cure and 41.6 per cent. improvement in their series state that the bronchoscope should not be used (a) if recent profuse hemorrhage has occurred; (b) if the pulmonary involvement is quite extensive; (c) in a moribund patient; (d) when serious organic cardiac disease exists; (e) in existing laryngeal tuberculosis.

3. *Surgical.*—The surgeon usually is called on to apply his art to those cases which do not show improvement to either of the above mentioned procedures. Many varieties of operative procedures have been devised and carried out for pulmonary abscess:

\* See section on Bronchoscopic Aspiration under Bronchoscopy for Disease.

(a) Artificial pneumothorax has been used to advantage in some cases of central abscess. It is not advised where the abscess is peripherally situated. Tewksbury<sup>14</sup> reports 20 cases cured and 1 improved by this procedure in a series of 25.

(b) Thoracotomy<sup>15</sup> is usually the drainage method of choice when the abscess can be reached without the contamination of healthy lung tissue.

(c) Thoracoplasty<sup>16</sup> with collapse of the chest wall is advocated by some as an adequate means of treating central abscesses.

(d) Lobectomy<sup>17</sup> by means of the knife or cautery is a radical procedure having a high mortality rate and is recommended only in those cases that cannot otherwise be successfully treated.

(e) Phrenectomy and ligation of the pulmonary artery have given dubious results.

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## TUMORS OF THE MOUTH, PHARYNX, AND FAUCES

### BENIGN TUMORS OF THE LIPS AND TONGUE

**"Macrocheilia"** and **"macroglossia"** are terms applied to a more or less uniform enlargement of the lips and tongue respectively, in contradistinction to definite tumor formation. This terminology is used in a descriptive sense and has no definite pathological basis. The superabundance of tissue is frequently congenital, but may be the result, particularly in the lips, of prolonged infections inducing a chronic lymphangitis. In the congenital form the enlargement is usually composed of lymph channels or angiomatous tissue, but may show an excess of fibrous elements or glandular structures.

*Treatment.*—Boiling water may be injected into the hypertrophied tissue (see Angioma). A wedge-shaped piece may be taken out to correct the deformity and the edges brought together with waxed silk sutures. Bleeding is free.

**Hemangioma and Lymphangioma.**—Neoplasms of this character are not infrequent and may occur anywhere in the oropharynx. They are usually congenital and may remain small or, after a few years, begin to increase in size. These tumors are purplish or whitish in color, depending on the presence of blood or lymph in the vessels. Pressure on the mass will cause the tumor to diminish temporarily in size as the vessels empty. Bleeding may be a troublesome factor following slight irritation. On the lip the tumor may be a small elevated purple spot or grow to involve one or both lips and even extend over the cheeks and face.

Angiomas on the tongue are usually on the dorsum and the anterior portion. Butlin<sup>1</sup> says they are seldom large but reports 1 case of congenital angioma of the cavernous type which had grown to such size that it required excision of the whole anterior two-thirds of the tongue.

In the pharynx they may spring from the venous plexus around the base of the tongue, the lingual or the faucial tonsils. They may give symptoms of obstruction and irritation if they attain any considerable size.

Phillips<sup>2</sup> gives a picture of a typical case of angioma of the uvula.

*Treatment.*—Angiomas, regardless of size, should be treated as soon as they are noticed. On the lip, if not too large, they may be dissected out or preferably treated by radium. A large angioma may be obliterated by periodic injections of boiling water after the method of Wyeth as described by Blair.<sup>3</sup>

On the tongue they may be either dissected with a knife or removed by the actual cautery; radium applicators or seeds are effective. Hemorrhage is troublesome. Angioma of the uvula may be removed with the galvanocautery snare and this method is practicable whenever such a

growth is pedunculated. When a wide area of the pharynx is involved, a few vessels at a time either may be tied with ligatures or obliterated by the actual cautery or electrolysis.

**Papilloma.**—Warts on the lips and tongue are rather common. They are usually the result of irritation, as in smokers. They may be implanted by contact in removing a papilloma from the larynx. All warts should be removed because of their potential relationship to cancer. This may be done by a wide V-shaped incision through the thickness of the lip or they may be treated by radium or diathermy. Partial excision or the use of caustics may promote rapid growth.

**Mucous cysts** of the lip are frequently encountered. They usually follow some slight injury which obstructs the duct of a gland, with retention of a translucent fluid. The most satisfactory results are achieved by complete dissection, the defect being closed by horse-hair or dermal sutures.

**Congenital Cysts of the Tongue and Floor of Mouth.**—Of the congenital cysts the most important are: (1) Thyroglossal cyst; (2) ranula; (3) dermoid. They originate in remnants of the thyroglossal duct, in the branchial clefts, or in epiblastic buds left during the fusion of the ventral folds.

**Thyroglossal cysts** represent remnants of the thyroglossal duct and occur usually in the midline at the base of the tongue or they may divide the muscles and project beneath the mucous membrane on the pharyngeal surface. They may not appear or produce symptoms until adult life. The tumors are soft, usually sessile, and without surrounding induration. They vary in size, occasionally becoming as large as a hen's egg. Growth is slow and painless. Large veins usually seen on the surface of these vessels, together with small hemorrhages within the capsule, give the growth a dark color. Bleeding is not infrequent and, if the growth is small, may be the only annoying symptom. Larger tumors tend to project backward toward the pharynx and may produce a disturbance in speech and difficulty in swallowing. The growth is encapsulated and microscopically shows thyroid tissue.

**Treatment.**—Small tumors without symptoms require no intervention. If hemorrhage occurs or if the tumor increases in size, excision is the proper treatment. This is best accomplished with the actual cautery because of the free hemorrhage. Before removal a thorough examination for a thyroid gland should be made, as cases have been described in which the tumor contained the only functioning thyroid tissue. Should this be the case, some of the tumor must be left to avert hypothyroidism.

**Ranula.**—The term "ranula" is commonly used to designate mucous cysts under the tongue. Such cystic tumors are usually congenital. Obstruction of the ducts of one of the salivary glands may give rise to an acute and painful distention—a condition known as acute ranula. This obstruction may be caused either by an inflammatory process within the duct itself or by external pressure.

The theory of the branchiogenetic origin of deep cervical cysts is sponsored by Thompson<sup>4</sup> and others. Thompson's conclusions are that ranulæ, submaxillary cysts, and deep cervical cysts are derived from vestigial remains of the branchial clefts; the primary cyst is derived from the cervical sinus. The cyst is carried from its original position by the shifting of the muscles during the formation of the neck.

Sublingual cysts are the most common and are found at the side of the

floor of the mouth as tense, thin-walled, fluctuating masses pushing the tongue upward. They may cross the midline or may be bilateral.

Submaxillary cysts, while not common, are the deepest in their origin and are the most difficult to treat. They may bulge in the floor of the mouth, but frequently burrow deeply and appear as a swelling beneath the jaw.

Small cysts appearing in the glands under the frenum or in the glands of Nuhn and Blandin, under the tip of the tongue, are not to be confused with those of deep cervical origin.

*Treatment.*—Excision of the smaller cysts is an easy matter. In the sublingual and submaxillary types, simple incision will give relief for a short time but the cyst will soon refill. Complete excision or permanent drainage must be established. To effect this may be an annoying problem to the operator. Part of the cyst wall may be trimmed away with scissors, and the remaining portion of the wall may be sutured to the mucous membrane. Deep submaxillary cysts may require complete dissection if they are giving symptoms and are not amenable to permanent drainage. It is not always possible to do this through the floor of the mouth and an external incision may be necessary.

**Dermoid Cysts.**—These are usually met with in infants at birth or in early life. They may be found at the base of the tongue or in the floor of the mouth and may produce obstructive symptoms. As a rule it is not difficult to differentiate them.

#### BENIGN TUMORS OF THE OROPHARYNX

**Papilloma.**—Warty growths are fairly common and may grow from any part of the mucous membrane. The base of the uvula and the anterior pillars are the most usual location. They may be either sessile or pedunculated.

*Treatment.*—Cocainization and removal with a sharp knife or scissors, or destruction by diathermy or radium.

**Adenoma.**—Adenomas are usually found in the soft palate, but they have been found on the tonsil and uvula. They may grow to considerable size, are usually firm, embedded in the tissue, and covered by normal mucous membrane. If they are producing symptoms, the mucous membrane over them should be incised and the tumor shelled out.

**Fibroma.**—Fibromas are rarely found on the palate and are much more often seen in the nasopharynx. They may be removed in the same manner as papillomas, but when situated beneath the mucous membrane they are difficult to dissect.

**Miscellaneous Tumors.**—Lipoma, chondroma, teratoma, myxoma or almost any other type of tumor may be found in the oral cavity. Bone and cartilage are occasionally encountered in the tonsils, but are of no significance. Osteomas frequently occur along the maxillæ or the hard palate. They usually remain benign and require no treatment. If they attain great size or if the mucous membrane becomes ulcerated, excision is indicated.

Tumors of a mixed type may appear on any part of the oropharynx. The point of origin is often in the salivary glands, but there are exceptions to this rule. New<sup>5</sup> describes this tumor as one that is hard and feels malignant, but one of slow growth and with little tendency to break down. In the pharynx mixed-celled tumors must be differentiated from aneurysm of

the internal carotid artery, gumma, and growths of the carotid body bulging into the pharynx. The treatment is surgical.

### TUMORS OF THE JAW

Papilloma, fibroma, myeloma, carcinoma, and sarcoma are encountered in this region. These tumors may be sessile or pedunculated, and they have their origin in the periosteum or the root membrane. Tumors of the gums may occur at all ages. Of tumors of the malignant type, sarcoma is the most frequently encountered. Careful study should be given to neoplasms in this locality before treatment is instituted. Biopsy should not be done. The hypertrophy of the gums seen in women during gestation should not be mistaken for true tumor formation. Every effort to rule out syphilis must be made. If a tumor is found, whether sessile or pedunculated, it should be removed with a wide margin of healthy mucous membrane and a plate of underlying bone. Then, and not before, a careful microscopical examination should be made. McArthur<sup>6</sup> believes that "even though the tumor may reveal the cellular arrangements that characterize the most malignant sarcoma, if a capsule or limiting membrane, be it ever so thin, is found, radical procedures may sometimes be avoided."

### MALIGNANT TUMORS OF THE OROPHARYNX

**Leukoplakia** will be considered in this section because of its association with carcinoma.

This lesion is characterized by one or more bluish-white patches on the mucous membrane of the tongue, the buccal surface of the cheeks and, in rare instances, on the gums. Patches are sometimes seen on the lips and authentic cases have been reported<sup>7</sup> in which the process has involved the mucous membrane of the larynx. Typical lesions give the appearance of a painted or enameled surface and may involve a small area or the entire buccal mucosa.

*Etiology.*—Prolonged irritation of the mucous membrane by mechanical, chemical, or thermal agencies or by some combination of these factors, is regarded as the essential cause of leukoplakia. The most important irritants, in the order of frequency, are (1) rough teeth, (2) tobacco, (3) syphilis.

Rough, carious, or dirty teeth, or an improperly fitting denture will be found in the majority of cases.

A history of the prolonged use of tobacco is usually obtained. Bloodgood<sup>8</sup> states that excessive use of tobacco rather than the form in which it is used is responsible for the development of leukoplakia.

Hazen<sup>9</sup> probably gives the most conservative opinion when he states that "syphilis represents an incidental rather than a causative factor in this disease."

Leukoplakia is largely confined to male adults. The lesions are rarely found in women and hardly ever begin under twenty or after sixty years of age.

*Pathology.*—Butlin<sup>1</sup> tersely characterizes the lesion as "scar tissue covered by thin epidermis." The papillæ are early involved in a chronic, inflammatory process which tends to obliterate them, leaving a smooth tongue. The hyperkeratosis is secondary. As these changes progress, the fibrous tissue increases and the lesion may be palpated as a slightly indur-

ated area. The epithelial surface may become fissured and broken, or warty overgrowths or small ulcers may appear. Obviously the dividing line between these stages and carcinoma cannot be determined.

*Symptoms.*—Usually there are no early subjective symptoms—the appearance of the patch on the tongue may be the first warning of the approach of this lesion. Later there may be a slight burning or feeling of irritation on taking hot drinks or food.

*Diagnosis.*—The characteristic feature is the white or bluish-white plaque on the mucosa which Bloodgood<sup>8</sup> compares to “a patch of enamel paint.” Careful palpation is most important, as it reveals induration and is effective in differentiating the hard and leathery area from the surrounding tissue. The progress is usually slow and if not seen until late, ulceration or fissuring may have taken place. The potential relationship between these plaques and carcinoma entitles them to most careful consideration and gives a special importance to the early stages of leukoplakia.

*Treatment.*—The first essential is to remove the source of irritation. The teeth must be given careful attention by a competent dentist. Carious teeth, rough edges, and defective plates, or other sources of irritation must be carefully searched for. Tobacco should be omitted or at least its excessive use prohibited. Not much reliance should be placed upon anti-syphilitic treatment, even though a history of syphilis is obtained or the Wassermann is positive. As long as leukoplakia shows no tendency to crack, peel, or ulcerate there is no indication for radical treatment. Efforts have been made to “cure” by means of the Roentgen rays, radium, and the electric needle, but it is not easy to say what may be expected from any method of treatment. The cautery used at a red heat is said to destroy the deeper process and to leave a functioning membrane. Any lesions involving prolonged irritation of an epithelial surface must be considered as an invitation to malignancy; however, Bloodgood<sup>8</sup> followed 27 cases for a number of years, giving careful attention to irritations and in none did cancer develop.

*Carcinoma.*—Cancer of the lip comprises about 2 to 3 per cent. of all cancers. Ninety-five per cent. of the cases are in men and the lower lip is the seat of the lesion twelve times as often as the upper. Ewing<sup>10</sup> describes two clinical types: (a) Papillary, (b) ulcerative infiltrating.

*Etiology.*—Any irritation or trauma, such as an erosion from the teeth, may be the starting point of a carcinoma. Many cases can be directly traced to the use of tobacco, the lesion developing at the site of a smoker's burn or on the side of the mouth where the cigar or pipe is held.

*Diagnosis.*—Bloodgood<sup>11</sup> believes that the most frequent precancerous lesion of the lip is a smoker's burn. It begins as a small depressed area of dark color and leathery consistency at the mucocutaneous border. Later a scab forms which usually becomes detached, only to re-form. This stage is of variable duration; ultimately the scab grows larger, induration follows, with the formation of an ulcer. The fact that such a lesion heals one or more times or appears to respond to some form of treatment should not mislead the inexperienced. Microscopically such lesions are usually carcinomata. Clinical cases are reported in which the initial lesion remained slight; yet the submental lymphatic glands already contained cancer cells. All warts of the mucous border of the lip should be treated as carcinomata until proved to be non-malignant by a competent microscopist. Usually

the malignant wart is larger than the benign; but Bloodgood<sup>11</sup> warns that while he has never observed a wart larger than the end of the index-finger to be microscopically benign, smaller warts are often malignant. A warty growth or a chronic ulcer, covered by a scab with indurated margins and accompanied by salivation and an odor, presents a clear picture. Enlarged glands and loss of weight are significant evidence of progress. Carcinoma may occasionally be confused with chancre, but the former is more chronic in its course, and the latter is more likely to be seen in younger persons. It must be remembered that a positive Wassermann does not exclude the possibility of cancer.

*Involvement of the Cervical Glands.*—It may be very difficult to palpate small lymph glands. These are often found to be enlarged at operation when they previously could not be detected. The glands which may be involved are a chain from the symphysis of the jaw extending toward the hyoid bone, between the geniohyoid and the mylohyoid muscles. Bloodgood<sup>11</sup> refers to an important gland situated between the body of the lower jaw and the submaxillary salivary gland, and says that it will be left behind in neck dissection if the salivary gland is not removed. Another chain runs from the submental glands to the submaxillary and from this to the tip of the parotid and downward behind the internal jugular. In the majority of cases early lymph-node involvement occurs on the same side as the primary lesion, but cases are reported<sup>12</sup> in which the earliest metastases were on the opposite side.

*Treatment.*—(See Treatment of Oropharyngeal Cancer.)

**Carcinoma of the Tongue.**—Carcinoma of the tongue is of the squamous-cell variety and ranks fifth in the cancer statistics. Males are affected eight or nine times more frequently than females. The greatest incidence is between the ages of forty and sixty. Rarely it occurs in the late twenties, and in these cases seems to follow the general rule that cancer is more malignant in patients under thirty than in older individuals.

The margins of the anterior half of the tongue or the tip are most commonly involved. The under surface of the tongue is seldom affected. If the growth is on the anterior half of the tongue, extension is usually in the direction of the gums and the floor of the mouth; extension from the posterior half is toward the pillars of the fauces and the hypopharynx. On account of the abundant lymphatic and vascular supply, involvement of the glands of the neck takes place early, and it must be borne in mind that cancer cells are frequently present in the nodes before they become palpable. Even after the regional lymphatics are extensively involved, metastasis to distant parts of the body is not common. The average duration of life in untreated cases is about two years.

*Etiology.*—The important etiological feature is chronic local irritation, and this factor is usually easy to trace. Bloodgood<sup>8</sup> presents sound evidence to show that when cancer of the tongue develops in men, they are warned of the danger by the presence of definite local lesions in a precancerous form. Usually a rough or carious tooth will be found to have irritated the border of the tongue. In other cases leukoplakia, due to the excessive use of tobacco, precedes carcinoma by some years. In the case of artisans who are accustomed to holding metal objects in the mouth, cancer may develop as a result of repeated abrasion. A simple ulcer due to a bite or a burn may be the precancerous lesion.

*Symptoms.*—Few patients escape early discomfort, and this is usually increased on taking hot liquids or highly seasoned food. As the lesion progresses the tongue becomes less pliable and movement is limited and painful; there is hypersecretion of mucus and saliva, and later this becomes an almost constant dribble. The breath becomes offensive and bleeding is not uncommon.

*Diagnosis.*—One should assume that all chronic lesions of the tongue in adults are potentially cancerous. Leukoplakia may never progress to the cancerous stage, but its potential tendencies must be recognized. Fissures, ulceration, and any wart-like overgrowth must be regarded as probable cancer. Simple ulcers occur on the tongue from injury, but these will promptly respond to treatment.

Chronicity or any tendency toward induration, ulceration, or warty overgrowth are the danger signals in the development of carcinoma of the tongue in its early and operable stage. As the lesion approaches the inoperable stage, unmistakable characteristics appear and the diagnosis is easy and comparatively valueless.

*Prognosis.*—The mortality is commonly between 75 and 90 per cent. According to Bloodgood this is largely preventable. The etiological factors and the precancerous lesions are well known, and the tongue is easily accessible for observation. When the carcinoma is located on the tip of the tongue, the prognosis is more favorable. In cases requiring extensive surgical procedures, the postoperative mortality is high.

*Treatment.*—(See Treatment of Oropharyngeal Cancer.)

**Carcinoma of the Pharynx.**—Squamous-celled carcinoma is the form met with in this region. It may appear on the uvula, soft palate, faucial or lingual tonsils. When on the tonsils, it commonly begins in the lower portion near the base of the tongue. Growth is usually rapid and the true pharynx, tongue, epiglottis, and larynx are involved by extension. The earliest evidence may be that of a definite growth with an indurated base; more often when first seen an ulcer with a hard and irregular edge is already present. The question of induration is most important and must be determined by careful palpation. The glands behind the angle of the jaw may be enlarged and matted together. Other symptoms will depend on the location of the lesion. Movement of the tongue is restricted and swallowing is painful, the pain radiating to the ear. Salivation is common; slight bleeding and an offensive odor accompany ulceration.

*Prognosis.*—Unless the diagnosis is made early and there is prompt response to some form of treatment, the outlook is hopeless. If the tonsil is the seat of the primary lesion and treatment is begun before the surrounding structures are invaded, good results may be obtained. Quick<sup>13</sup> states that when the tongue is involved by extension, the prognosis is reduced by 50 per cent.

*Treatment.*—(See Treatment of Oropharyngeal Cancer.)

**Sarcoma of the Pharynx.**—Sarcoma may begin in any portion of the pharyngeal wall, but the tonsil is the most common site. Lymphosarcoma of the tonsil is most frequent during midlife but may occur in childhood.<sup>14</sup>

*Diagnosis.*—The growth is not always distinctive, but the unilateral location, lobulated appearance, and thickened mucous membrane will aid in identification. The tumor is frequently assumed to be an enlarged tonsil and a tonsillectomy is done, only to have the fossa rapidly fill

with sarcomatous tissue. In other instances a swelling is encountered extending upward into the supratonsillar fossa and simulating in appearance and in the characteristic speech a peritonsillar abscess.

Enlarged glands back of the angle of the jaw may appear, but these are not a constant finding. Fixation of the jaw may be an early symptom. As the growth enlarges a feeling of fullness, difficulty in swallowing, constant clearing of the throat, pain, and bleeding develop. Salivation, ulceration and offensive odor are later symptoms.

Lymphosarcoma of the tonsil is too often mistaken for and treated as chronic inflammatory tissue or a chronic ulcer. It may be difficult to differentiate from carcinoma; but the distinction is of little practical value. The fact that it may occur earlier in life than carcinoma makes it more likely to be confused with syphilis than is carcinoma. This would be a serious error, as valuable time might be lost in administering antisyphilitic treatment.

*Prognosis.*—The prognosis is extremely grave; cachexia comes on rapidly, especially if there is painful deglutition; death may take place from hemorrhage due to an eroded vessel, or the patient may die from exhaustion or intercurrent disease.

*Treatment.*—(See Treatment of Oropharyngeal Cancer.)

#### TREATMENT OF OROPHARYNGEAL CANCER

**General Considerations.**—The name of J. C. Bloodgood, of Baltimore, stands out pre-eminently as that of one who is devoting a lifetime in earnest endeavor to master this dread disease. No one who attempts a survey of this subject could do better than to quote freely the results of his wide experience. He states<sup>8</sup> that there is no doubt that cancer of the mouth is a preventable disease, and that the element of time is the largest single factor in its cure. For cancer of the tongue he presents statistics showing that educational propaganda has decreased the percentage of the hopeless and inoperable cases from 47 to 20 per cent.; and he emphasizes the fact that teaching is more important than surgery, since delay in proper treatment after the onset of a malignant lesion reduces the chance of a cure in operable cases from 62 to 12 per cent., and increases the risk of postoperative death from 5 to 30 per cent. He deduces from his analysis that the literature on the clinical pictures of early cancer is inadequate. Even in the presence of a positive Wassermann reaction, he asserts it is unwise to depend on antisyphilitic treatment for any local lesion on the lip or in the oral cavity until the patient is seen in consultation with one capable of making the differential diagnosis.

The value and safety of biopsy is not entirely settled at the present time. Bloodgood believes that the safest method of performing biopsy is to have it done by the surgeon who is responsible for the treatment. Broders,<sup>15</sup> of the Mayo Clinic, proposes as a prognostic aid of apparent value the grading of the degree of malignancy according to the cell differentiation.

Thorough excision of suspicious lesions in the earliest stage will give cures in almost 100 per cent. of the cases, while there is no known cure for inoperable cancer. The cases in which cancer grows slowly are extremely rare, therefore the physician or dentist who has not had special training in this line should spend no time in temporizing or in treating innocent looking growths with caustics, but should avail himself of the advice and judgment of those of wide experience. Bloodgood states that in one series of 27 cases,

fifty-four physicians and thirty-three dentists had given treatments without recognizing the nature of the lesion.

Treatment must necessarily fall under three heads: (1) Surgery, (2) radiation with radium or Roentgen ray, (3) surgical diathermy.

**Surgery.**—The following general surgical principles are advocated in the treatment of carcinoma of the jaws, tongue, cheek, and lips:

1. The primary focus may be removed by surgical diathermy, cautery, excision, or radium.

2. No cancer tissue should be cut or handled.<sup>16</sup>

3. In operations for cancer of the buccal mucous membrane, a "platter" of underlying bone should be removed together with the intact growth.

4. The local lesion should be removed first and the glands should be removed after a certain period has elapsed, as the delay will diminish the possibility that any of the glands may be overlooked.

5. Cancer of the lip is best treated by maxillary dissection.

6. For cancer of the tongue, block dissection is the method of choice.

7. For electrocoagulation a general anesthetic is required. The rectal administration of ether is very satisfactory. A proctoscopic examination should be made first to rule out any possibility of a diseased condition of the bowel.

8. A single treatment with deep, accurately measured Roentgen rays, should be given from two to three weeks before operation.

I am indebted to Dr. Thomas E. Jones, of the Cleveland Clinic, for the following valuable suggestion in regard to the use of radium in the treatment of cancer of the lip: It is just as efficacious as surgery, and more patients with early suspicious lesions will submit to its use than to surgical removal. If the latter treatment is advised, a considerable number will refuse treatment and therefore will go untreated until it is too late.

For the surgical removal of growths from the lip a quadrilateral incision is preferable to the formerly advocated V-shaped incision, as such an incision would not include all the lymphatics which drain the affected area and some cancer cells might be left.

The surgical outlook is apparently poor in pharyngeal cancer. Beck<sup>17</sup> states that the majority of his cases had already advanced to such a degree when he saw them that even with radical surgery, including a dissection of the glands followed by the use of radium and Roentgen rays, he neither saved the patient nor retarded the growth of the neoplasm.

**Roentgen Ray and Radium.**—Radiation in some form, either alone or in combination with surgical measures, plays an important part in the treatment of oropharyngeal cancer. To bury radium emanations in the tumor and to leave them *in situ* is the method advocated by Quick.<sup>18</sup>

The experience of Dr. Douglas Quick<sup>13</sup> with the use of radium in cancer of the buccal cavity is an extensive one. He states that after he adopted the technic of Janeway,<sup>19</sup> using buried emanations instead of surface application, his results improved. He attributes the good results achieved in treating cancer of lip and cheek to the fact that it is possible to treat the lesion from both surfaces. His procedure is to radiate the neck first with either filtered radium or with Roentgen rays, whether or not the nodes are palpable. No glands are operated upon either by the removal of nodes or by the insertion of radium without preliminary radiation. He advocates conservatism in the treatment of the cervical nodes on the following grounds:

(1) The cervical lymphatics have a more conservative and useful function than that of a filter; (2) extension of disease to the node is by embolism rather than by lymphatic extension.

In making a general comparison he found that there had been fewer recurrences after the use of radium alone on the primary lesion, and of a combination of radium and surgery in the neck, than after surgery alone, notwithstanding the fact that his statistics are based on a group of unselected cases. He believes that for all malignant growths of the tonsil, radium should be the agent of choice, since experience has shown that neoplasms of the tonsil are particularly susceptible to radium. He makes the distinction that cervical metastases from carcinoma of the tonsil are best managed by a combination of radium, Roentgen rays, and conservative surgery, but that in lymphosarcoma surgery plays no part at all; the primary growth and metastases should be treated entirely by a combination of radium and Roentgen rays. Pfahler,<sup>20</sup> Robinson,<sup>21</sup> and others have all published good results from the use of radium. Robinson believes that the best results will be obtained by the co-operation of the radium therapist and the skilled surgeon. Tilley<sup>22</sup> is of the opinion that no sarcoma should be surgically treated until radium has been tried.

**Surgical Diathermy.**—Very good reports from the use of diathermy come from the British authors. The principle of this method as described by Tilley<sup>22</sup> is as follows: "If a high-frequency current is led into the body through a narrow metal rod or needle, the concentration of the current at the point of entry is so great that intense heat is developed in the tissue for a small distance around the electrode. The current is led out by a large flat metal electrode placed on a distant part of the body and at this site very little heat is developed. The heat developed in the small electrode is so intense that the tissues immediately around it are coagulated, the blood-vessels and the lymphatics are sealed, and the risks of dissemination from malignant tumors are lessened." McKenzie<sup>23</sup> relates his experience with this agent in pharyngeal cancer over a period of ten years and thinks that no remedy can equal it in that it is applicable both to operable and inoperable cancers. It has a twofold action: (1) Cauterization combined with coagulation by which cancer tissues can be destroyed; (2) an antiseptic effect by which the tissues some distance beyond the range of its destroying powers are sterilized of bacteria. McKenzie removes the initial lesion by surgical diathermy and the lymphatic glands draining the area by a combination of surgical dissection and diathermy. He recommends that the operation be done in two sittings and prefers treating the neck first, including the ligation of the external carotid artery. The subsequent operation on the pharynx can then be done with more precision and less bleeding. Before dissecting an enlarged gland, he punctures it with a diathermy needle to destroy the living cancer cells. After the removal of all the glands the wound is treated by diathermy. Deep puncture should be made at the angle of the jaw. The initial lesion is removed with a diathermy knife, allowing  $\frac{1}{4}$  inch outside the growth after the surface ulcer has been coagulated. A sharp rise of temperature may come a few days after the operation, but there is usually no shock. The slough separates in ten to fourteen days. There is little or no evidence of infection. Considerable scar tissue ensues, but McKenzie thinks this is desirable.

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## NEUROSES OF THE NOSE AND PHARYNX

## NEUROSES OF THE NOSE

The matter comprising this section does not attempt to deal with reflex neuroses, such as hydrorrhea, rhinorrhea, hay-fever, and asthma. It has to do largely with the sense of smell and its modification in various abnormal conditions. Neuroses of the pharynx will be considered from the standpoint of sensation and motion.

In order that the neurosis involving smell may be better understood, a short description of the anatomy and physiology is here given. The physiology of olfaction is intensely interesting and many points have yet to be settled. The industrious student will find much to interest him in Parker's recent monograph on Smell and Taste, and Dan McKenzie's book on Aromatics

and the Soul. In man, while evolutionary changes have occurred they do not result in such elaboration as is seen in the eye or ear. The essential structure on which smell depends is still primitive, and reaches its highest development not in man, but in terrestrial mammals otherwise inferior to him—in the dog for example.

**Anatomy and Physiology.**—Between the nasal septum and the series of folds known as the conchæ, ordinarily three in number and called the inferior, middle, and superior turbinates, there exists a slit-like space known as the common meatus. It extends from the posterior part of the vestibule to the choanæ. The posterior portion of the space in its uppermost part, where it lies between the superior or such concha as happens to exist, and the space becomes a narrow slit and is called the olfactory cleft.

The nasal mucosa in the main or respiratory portion of the nasal meatuses is reddish in color and consists of a pseudostratified epithelium containing ciliated cells and basal cells with a well-developed tunica propria. Numerous branches of alveolotubular glands containing both mucous and serous cells supply it with moisture. In the olfactory area the membrane is yellowish or light brown in color and is composed of three classes of cells—basal cells, epithelial or sustentacular cells, and sense cells from which the olfactory fibers take their origin. The olfactory cells are the most numerous of these three classes of cells. The nuclei are looped in an oval cell body and the central ends of the cell bodies taper rapidly to become olfactory nerve fibers which pass up through the cribriform plate to the olfactory bulb. Here they end by forming synapses around mitral cells, and the axons of the latter run backward through the olfactory tract, some entering the anterior commissure by means of the mesial olfactory stria and the remainder entering the lateral olfactory stria which leads them to the cortical center for smell in the cornu ammonis. These fibers do not pass through the internal capsule as do the fibers of all the other organs of sense, the gustatory fibers excepted (Henry Hun). The peripheral extremity of the olfactory cell extends as a rod-like structure to the outer surface of olfactory epithelium in a small enlargement called the olfactory vesicle of Van der Stricht. On this vesicle is a number of protoplasmic filaments—the olfactory hairs or lashes. In the submucous tissue of the olfactory area are found Bowman's glands, whose ducts open through the olfactory epithelium and their secretion is largely the cause of the moist condition of this area. There are other nerve-fibers found in this area which are derived from the trigeminal nerve, but they are not so numerous as in the respiratory area. The area covered by the olfactory epithelium is according to Read, equal to a 25 mm. square. It extends from the dorsal portion of the olfactory cleft over the superior turbinate almost to its free edge and correspondingly over the septum to about one-third of its extent.

In order that we may become conscious of odors (*i. e.*, to smell) there must be some stimulation of the distal terminations of the olfactory cell that is carried up to the olfactory center in the brain. The olfactory lashes act the part of receptors and respond to the stimulation carried to them by the air-currents. In inspiration and expiration the air-currents are limited to what we call the respiratory region of the nose, the olfactory region being undisturbed. These currents are accompanied by eddies which make for variations of pressure within the upper nasal meatuses and thereby is a change of air resulting in stimulation of the special epithelium

and initiation of olfaction. Sniffing naturally causes a rapid interchange of air and the appreciation of the full effect of odors.

Durant claims that olfaction is more or less dependent upon an appropriate hygrometric state in the olfactory atmosphere, and whatever facilitates the condensation of watery vapor there facilitates olfaction. Odors are perceived more by inspiration than by expiration, because the expired air passes over the lower nasal meatuses and has lost in the lower pharyngeal mucosa some of its odorous particles.

The olfactory acuity is very highly developed. Means of measuring it have been devised by Zwaardemaker, Allison, Katz, and Valentine. The latter found that oil of roses could be appreciated in 1/2,000,000 of a milligram. Arthur W. Proetz has proposed a method by which definite standardization of odors may be made. Professor Malan (Turin) has devised a method of recording objectively the olfactory powers by tracings made on a revolving drum which show the effects of odoriferous substances on the respiratory and cardiovascular centers. Malingering and Workmen's Compensation cases might be better studied in this manner. In the lower animals the acuteness of smell is well known; ants, dogs, and sharks being particularly keen. In people of dark skin there is evidence to show that the sense of smell is more highly developed than in those of lighter pigment. Mammals with many conchæ (the *Orycteropus*) are unusually keen scented and are termed "macrosmatic." In man, monkey, seals, and some whales where the conchæ are not more than four, there is lessening of acuteness of smell, and they are called microsmatic. Animals with a degenerated olfactory organ are called anosmatic; porpoises, and toothed whales being examples.

The method by which the special epithelium best receives the odors is a disputed point. The odor appears to be conveyed in a gaseous state but the gas may be absorbed by the secretion of the mucosa and so act in a watery medium. Some gases are more soluble in water than in oil, and if the olfactory cells are lipoidal in structure (Backman) they may alter some gases to an oily state when they become more active. Normal butyl alcohol has a strong odor and is soluble to the extent of 8 per cent. in water, while it is indefinitely soluble in oil; on the other hand, ethyl alcohol and methyl alcohol have very slight odor and are freely soluble in water, but very slightly in oil. Care must be taken in testing the reaction of the nasal epithelium to vapors like ammonia which stimulate the trigeminal nerve endings. The minimum perceptibility of odors has been termed by Proetz one in fact and is expressed in terms of grams per liter in the minimum concentration of a substance in solution which can be perceived by the sense of smell. The olfactory organ can perceive an odor too weak to be identified.

Hennings' classification of odors is as follows:

1. Spicy odors: Fennel, the oil of sassafras, anise, and cloves.
2. Flower odors: Heliotrope, coumarin, and Geranium oil.
3. Fruity odors: Oil of orange, citronella, oil of bergamot, and acetic ether.
4. Resinous or balsamic odors: Turpentine, Canada balsam, and Eucalyptus oil.
5. Burn odors: Tar pyridine.
6. Foul odors: Carbon bisulphide, hydrogen sulphide.

**Anosmia** or loss of sense of smell is a symptom rather than a disease and

as such may be partial or complete, temporary or permanent. The loss of taste complained of by patients is due to abeyance of the perception of flavors which depend on the sense of smell. It is convenient to divide anosmia into two classes—respiratory and essential.

*Respiratory.*—Any structural abnormality resulting in preventing air access to the olfactory area will lessen the sense of smell in proportion to the completeness of the obstruction, though obstruction may not be sufficient to prevent nasal respiration. An alteration of the inspiratory air path owing to severe wounds or disfiguring ulceration and contraction of the alæ may result in the inspired air passing along its lower nasal meatuses and thereby not reaching the olfactory area at all or very imperfectly. Septal deflections of themselves rarely produce complete loss of smell. A very bad high deviation might tend to unilateral defect. A definite unilateral anosmia was the only neurological finding in a case of brain tumor seen by Dr. K. McKenzie in Cushing's service. The tumor was found on the same side. Another instance of an intracranial lesion is presented in the case history of a patient of Dr. K. McKenzie's in the Toronto General Hospital:

*Bilateral olfactory groove meningioma with a syndrome of anosmia, homolateral optic atrophy, and contralateral papilledema: Complete removal in two stages by the electrosurgical method. Recovery.*

On January 23, 1928, Mrs. E. J. H., age thirty years, presented herself for examination. She had been perfectly well until two years ago when she noticed that she could not smell things cooking, and, on testing herself with perfumes, discovered that she had completely lost her sense of smell. For the past four months she has suffered from severe frontal headaches. At times she feels as though some one were digging out her eyes. Her vision has been steadily getting worse for the past six months and is much worse on the left side. There has been a good deal of nausea and vomiting since her headaches commenced. The vomiting is not projectile.

*Positive findings on examination:*

1. Quite unable to appreciate any test odors. Primary atrophy of the left disk with the field constricted down to a central vision of 20/100. The right disk shows a papilledema of 4 diopters with the field and blind spot normal. Vision is 20/60.

2. Slight memory deterioration.

3. The Roentgen ray shows some flattening of both supra-orbital plates, with a depression and spreading out of the sella turcica.

*Note.*—A Roentgen-ray diagnosis of pituitary tumor had been made on this patient because of the enlargement of the sella, but the absence of all other pituitary signs, especially the fact that the menstrual periods had remained normal, made one feel that this was not the correct diagnosis. The complete loss of smell enabled one to make an accurate preoperative diagnosis of an olfactory groove meningioma.

Acute inflammatory changes in the nasal mucosa may rapidly shut off the olfactory area and complete anosmia result, but recovery is usually rapid. This is seen in the common head cold, but it is somewhat different in some types of influenza where anosmia is due not alone to the rhinitis obstructing the air-way so much as to a direct toxemia of the olfactory hairs resembling a peripheral neuritis. Chronic hyperplastic changes of the turbinate covering seldom cause much loss of smell but atrophic changes as seen in atrophic rhinitis, with or without fetor, are almost invariably associated with some anosmia. Rhinitis sicca, if of long standing, also causes some loss of smell as also may fifth nerve paralysis by causing dryness of the nasal mucosa. Accessory sinus disease seems to bear a definite relation to defects of smell. Chronic pansinusitis is often associated with the formation of multiple nasal polypi. These are always associated with ethmoidal or antral disease and readily block off as it were the olfactory area. Obstruction in these cases is not the sole cause, as acute and especially chronic

sinus suppuration is, in the majority of cases, associated with some defect of smell.

The olfactory mucosa may be impaired by sniffing cold salt solution, snuff, or by the use of cocaine solution over a considerable time, or the use of nasal douches or sprays containing carbolic acid or astringents like zinc, alum, or a strong solution of iron. New growths may also inhibit or destroy the sense of smell. The obstruction to the air-way may be in the nasopharynx, viz., stenosis of the choanæ, large adenoid mass, nasopharyngeal growths, simple or malignant, and complete adhesions of the soft palate to the posterior wall of the pharynx.

*Temporary anosmia* is not infrequently observed without the occurrence of any acute nasal inflammation or obstruction. Some people cease to be conscious of certain strong odors though identifying others well. Chronic invalids with foul-smelling emanations soon lose their smell for those odors, while workers in glue, soap, and bone factories are not troubled with foul odors, probably due to the occurrence of olfactory fatigue.

*Essential anosmia* is a condition in which there is a loss of smell, but no nasal condition is visible to account for it. The diagnosis should only be made after very thorough nasal examination and the elimination of the factor causing the respiratory type of anosmia.

It may result from injuries of the head when the cribriform plate is fractured. Rarely is it due to congenital defects such as absence of olfactory bulb and nerve. Intracranial lesions are sometimes the cause, such as meningitis, tumors, syphilitic disease, embolism or thrombosis of the middle cerebral artery, epilepsy, tabes, general paralysis, hysteria, and insanity. When due to intracranial lesions the sense of taste is not infrequently also involved. Lesions involving the periphery of the olfactory nerve should also be considered. A peripheral neuritis may be due to influenza, lead, tobacco, or alcohol.

*Prognosis.*—The more one is able to find in nasal examination to prevent proper exchange of air in the upper nasal meatuses, the greater the probability of benefit. Time alone is not the only consideration. Cases of obstructive anosmia have recovered the sense of smell after even thirty or forty years, but the defect was probably only partial. If there are temporary remissions, the prognosis is better. Operations should only be undertaken for a very pronounced obstruction; still the drainage of a chronically inflamed sinus is sometimes followed by very marked improvement in the sense of smell. Patients who have ozena or whose defect in smelling is associated with injury to the olfactory mucosa and nerve-endings, are not likely to receive much benefit if these defects have existed for more than a few months. In essential anosmia the prognosis is nearly always bad, yet occasionally patients recover some of their lost sense after long periods and one has no explanation to offer for it. The toxic type, such as influenza, lead, etc., may respond if the condition has not existed too long.

*Treatment.*—**OPERATIVE.**—Seldom is an operation performed to restore the sense of smell alone. The associated obstructive symptoms are usually the most pronounced and the operation should be directed to relieving the nasal obstruction; it will in many cases at the same time restore the sense of smell. It does not always do so, and too much must not be promised. After clearing the air-way the use of slightly stimulating vapors may assist the olfactory nerve-ends in resuming their full functional activity. Edema

of the nasal mucosa may require snaring or the cautery point; similar changes in the accessory sinuses require, in the main, ventilation and drainage.

**MEDICAL.**—Nasal sprays or douches should be of the blandest variety and used in great moderation. In the essential group nerve tonics and electricity are all we have to offer our patients. Usually these are of little avail. The hysterical patient responds to suggestion in some of its forms. General tonics such as strychnia, arsenic, quinine, and phosphorus may be tried. Lermoyez recommends the following used as a snuff twice daily: Strychnia sulph., 1.10; pulv. iridis, 0.50; sacch. lactis pulv., 10. In syphilitic cases appropriate treatment should be carried out. Tilley has found the use of tincture of iodine applied daily for three times to the mucosa of the olfactory cleft useful in some cases of anosmia. He also advises counterirritation in the form of a blister to the neck. When the anosmia is associated with a chronic edematous change in the mucosa a rigid supervision of the patient's diet, exercise and habits is very essential. Attention to the state of the bowels is in such cases more important than local treatment of the nasal mucosa.

**Hyperosmia** is a condition in which the patient is unduly sensitive to odors. He has hyperesthesia of his olfactory nerve. It must not be confounded with that of patients who have a keen scent for they are not concerned about themselves or inconvenienced. In some cases of hysteria and insanity, and in neurasthenic women where there is exaggeration of nervous impressions, odors may be complained about which, if the patient were in good health, would probably be passed over without observation. Irritative lesions of the olfactory bulbs are also said to be associated with hyperosmia.

**Parosmia** indicates a perversion of the olfactory sense. When imaginary or subjective odors are present the condition is usually of central origin—in fact, it may be a very early sign of mental derangement, constituting the so-called olfactory hallucinations which may also be found in tumors involving the cornu ammonis. The *aura* of epileptics may be referred to the olfactory sense. Parosmia occurs also in hysteria, influenza, organic or functional derangement of the temporosphenoidal lobe, the climacteric period, and hypochondriasis.

**Cacosmia**, the perception of a foul smell, is usually associated with some fetid area in the upper respiratory or the alimentary tract. Very rarely it is a subjective symptom of disease of the nerve tract or an aura of epilepsy.

When a patient complains of a foul smell one should suspect a local cause. A very thorough nasal examination may disclose an empyema of some of the accessory air cells, especially the antrum. The posterior ethmoidal cells and sphenoid come next in order of frequency. A foul-smelling discharge from the frontal sinuses usually does not exist alone, but may prevent clearing up of the suppurative process in the lower cells. Foreign bodies and specific and malignant disease of the nose are found as causative factors. In *ozena* patients do not perceive the smell, but their friends do. Syphilitic necrosis within the nose has a very characteristic penetrating odor perceived by both the patient and his friends. Foul ulceration in the pharynx and nasopharynx may be found. Tumors, such as a breaking-down gumma and suppuration in a cancerous or retropharyngeal abscess, may be present. Tonsillar ulcerations from syphilis and cancer or Vincent's angina are often associated with foul emanations. Foul caseous masses are not infrequently found in the supratonsillar fossa. Abscess in connection

with teeth, especially old crowned teeth, must not be overlooked. It must not be forgotten that foul odors may be produced by lesions in the pharynx, esophagus, larynx, lungs, and stomach.

*Treatment.*—Hyperosmia and parosmia may be benefited by strychnin, arsenic, bromides, and iodides, but, on the whole, very little improvement can be expected in those patients of hysterical type who may be easily impressed by any new or novel remedy or procedure. Cacosmia offers much better prognosis, and the treatment is that indicated by causative lesion.

### NEUROSES OF THE PHARYNX

Pharyngeal neuroses will be considered under two divisions, viz., (1) sensory disturbance; (2) disorders of motility.

#### SENSORY DISTURBANCES

1. **Anesthesia.**—The pharyngeal mucosa is innervated by the glossopharyngeal, the pneumogastric, and the sympathetic system of nerves and the causes of lack of normal sensation may be of central or peripheral origin. Anesthesia due to central causes is usually a part of a neurological picture seen in patients suffering from disseminated sclerosis, bulbar paralysis, locomotor ataxia, tumors, basal meningitis, intracranial hemorrhage, syphilis of the meninges, and gumma of the cranial base. Hysteria too must be considered, but Hurst has shown that the pharyngeal sensibility varies greatly in healthy people. Tickling the posterior pharyngeal wall may produce marked reaction in some people, while in others, quite normal too in every way, similar procedures produce no sensation. If the anesthesia is unilateral, hysteria is more probable, but in testing for this errors easily occur.

The peripheral causes of anesthesia are diphtheria, use of certain drugs, such as cocaine and similarly acting substitutes, carbolic acid, bromides, chloral, morphin, anesthesin, and orthoform, the last only on broken mucosa. Very cold or hot fluids may produce a very short anesthetic effect. Anesthesia of the lower part of the pharynx, *i. e.*, the laryngopharynx, may be associated with lesions involving the glossopharyngeal or in connection with the lower fibers of the constrictors, the superior laryngeal. Professional singers are frequently found to be very tolerant to pharyngeal manipulations; but this is not true anesthesia, but rather an acquired tolerance due to practised control of the pharyngeal muscles. Chronic inflammatory conditions of the pharyngeal mucosa where there is a dry or glazed mucous membrane are usually less sensitive than normal, though they have passed through an earlier stage where excessive irritability was present.

*Symptoms.*—There may be little or no complaint, but a “peculiar” sensation of a little roughness may be elicited by careful cross-examination. If the pharyngeal or laryngeal muscles are also involved the subjective symptoms may be much more prominent. In such patients the danger of misswallowing, with possibly pneumonia, is always present. The superior laryngeal nerve supplies the larynx with sensation but filaments of the nerve also reach the lowest part of the pharyngeal mucosa.

2. **Hyperesthesia.**—Irritability of the pharyngeal mucosa varies so much in individuals who have no obvious local lesions that it is difficult to say when the reactions are normal. We have no standard to guide us. Many people will show marked irritability of the pharynx simply on the

use of a tongue depressor in a widely opened mouth. They volunteer the information that great distress has always followed efforts to see the back of the throat, and also that they are unable to swallow a pill or tablet because of this irritability. Such conditions are normal with these people and are not necessarily evidence of disease, though it may be present in "highly strung" individuals, especially neurotic women. Excessive nasal secretion, unaccompanied by change in the pharyngeal mucosa, may produce in this type of individual considerable discomfort, while in others of more stable nervous mechanism no complaint is made. If one says the former has hyperesthesia of the pharynx, then on the same grounds he should say that the latter has pharyngeal anesthesia or dysesthesia, but in both instances a normal condition exists.

Acute and chronic inflammation of the pharyngeal mucosa is largely the cause of abnormal irritability of the pharynx. Perverse sensations are very frequently present in the symptomatology of chronic pharyngitis. The most common are the feeling of a lump or foreign body, tickling, burning, itching, etc. It must not be forgotten that the so-called globus hystericus may depend on spasm of the cricopharyngeus due to ulceration, simple or malignant, at the mouth of the esophagus. Inflammation of the lymphoid tissue in the pharyngeal vault, palatine tonsil, or lingual tonsils is a frequent cause of excessive pharyngeal irritability; as are concretions in the faucial tonsils, an excessively long styloid process, or even a rigid or ossified stylohyoid ligament. In a similar way various aural symptoms may arise, even earache through the tympanic branch of the glossopharyngeal nerve. Excessive scarring of the posterior wall, due to scarlet fever or other ulcerative conditions, is another cause, while one might mention, too, the anesthetic lesions of the pharynx in leprosy. Enlarged lymph-nodes on the posterior wall of the pharynx may be present and produce no symptoms, especially in anemic girls. When, however, they are present in patients with diffuse chronic pharyngeal hyperesthesia, they may be the sensitive spots from which the irritability arises. Marked thickening of the posterior pillars is a frequent cause of pharyngeal discomfort. Prolonged seepage from the mucosa of the accessory nasal sinuses is often a cause of persistent irritation of the upper and lower pharynx. Crusts in the nasopharynx due to posterior ethmoidal or sphenoidal disease and chronic inflammation of Thornwaldt's bursa may also be the cause, and efforts to remove these crusts enhance the irritable effect. Crusts due to atrophic change produce much less discomfort. Probably the most common of all the causes of hyperesthesia of the pharynx is due to a simple chronic pharyngitis without any particular area exciting attention. It is frequently observed in chronic alcoholics and those who smoke excessively. Chronic nephritis, chronic diabetes, pemphigus, thrush, hepatic cirrhosis, and uncompensated heart disease all tend to produce pharyngeal irritability.

An elderly gentleman consulted the writer, not long since, complaining of a hot, dry sensation of his throat. Nothing beyond a slightly inflamed mucosa was to be seen, but his pharynx seemed unduly sensitive. Further examination showed the patient to have diabetes and only when his blood-sugar was definitely reduced did his pharyngeal distress improve.

Another very frequent cause of excessive pharyngeal irritability is abnormal gastro-intestinal conditions associated with dietetic errors. Those of full habit of body, who eat and drink too fast and too much, and

take far too little exercise, are very prone to excessive irritability of the pharynx.

Excessive use of voice by public speakers in poor atmospheric conditions produces probably its greatest ill-effect in the larynx, but excessive irritability of the pharynx is a very frequent association. An elongated uvula may cause a great deal of pharyngeal discomfort, but the cases are uncommon and in the past have had a great deal of unnecessary surgery.

Patients who have various abnormalities of the thyroid gland or other lesions in the neck frequently describe their symptoms as being situated in the throat and the physician is liable to err unless his cross-examination is searching. Large pulsating vessels are sometimes seen in the lateral part of the posterior pharyngeal wall. Brown Kelly of Glasgow has reported several, while I have seen two. Various abnormal sensations may be present. Lingual thyroid too may be found to cause similar discomfort with undue irritability. I had one case in which the thyroid tumor was removed and myxedema followed, requiring the continued use of thyroid gland to keep the patient well.

Moure in his book on the pharynx and larynx writes of hyperesthesia and hypesthesia, in which he says the former is manifested by a sensation of heat and pain in the pharynx and reflexes are accompanied by excessive contractions of the pharyngeal cavity which accentuates the pharyngeal congestion; while the latter is associated with the sensation of a foreign body, ball, thick saliva or fruit peel of which he continually tries to rid himself.

The general disturbances vary according as the affection is observed in neuro-arthritic or psychopathic subjects. Patients with hypesthesia are easily examined, as they are in the habit of frequently inspecting their own pharynx, and are particularly prone to attach grave importance to small epithelial plugs seen on the tonsils, thinking of these in terms of pus, with all the grave consequences of hidden infection. Mothers are prone to become unduly alarmed in examining their children's throats, and may initiate neurotic disturbances in their offsprings. Moure says hypesthesia usually originates in an emotion, sorrow or overwork, or in the fact that the patient has known someone who had succumbed to a disease of the throat. Pharyngomycosis, a condition in which horny epithelial plugs are present, may produce various discomforts, and if seen by the patient causes grave misgivings.

*Treatment.*—The treatment of anesthesia of the pharynx depends on the underlying condition causing the complaint. The symptoms may be so slight as not to require any local or general treatment. Medical treatment in the form of nerve stimulants, such as arsenic, strychnin or phosphorus, may be of value. When there is a marked dryness of the mucosa the use of wild mountain ash berry is said by its sialagogue action to be helpful. Electricity and massage may also be tried, and mercury in patients with a syphilitic cause. The treatment of the hyperesthetic form deals largely with the pharyngeal condition present, usually some form of chronic pharyngitis. The reader is directed to those sections dealing with these conditions. General treatment must not be neglected. Sometimes assuring the patient that he has no serious disease, such as cancer or tuberculosis, will allay his fear and greatly alleviate his distress. I have in many instances, where there was a good deal of complaining and little or nothing to be

seen to account for it, succeeded in reducing the undue irritability of the pharynx by giving the patient a postnasal applicator and with a small hand mirror show him how to paint his own nasopharynx and pharynx. Any medicament will do, but a mixture of menthol, 10 grains; tannic acid, 60 grains; and glycerine, 1 ounce, is a useful formula. The patient applies this to his pharynx twice daily and the massage he uses makes his pharyngeal mucosa very much more tolerant to slight irritation and he ceases to complain. Bromide and valerian may well be given in addition. Many patients are given an iodine pigment to apply to their pharynx when they complain of pharyngeal irritability with excessive secretion. As iodine stimulates secretion there seems to be no rational reason for its use. When there is atrophy of glandular structures and the mucus is tenacious, iodine may increase its fluidity and give relief. In the hypesthetic form Moure advises against antispasmodic treatment such as bromides. He prefers stimulating the sensibility of the pharyngeal mucosa by means of mentholated pigments, douches, and local electrization. Lozenges are frequently of use and tabloids of *ulmus fulva* (the red or slippery elm), with or without menthol or mucin or carbolic acid, are often found grateful to the patients. In all cases of pharyngeal neuroses it must be strongly impressed that frequent office treatments and local applications at home are prone to keep the patient thinking about himself and his pharynx. Some patients require local treatment, many are better without it. Regulation of the patient's habits, diet, excretions, and exercise will form a basis of most of the rational treatment for pharyngeal neuroses. A study of the patient's nervous system and digestive tract will yield on the whole more information that can be obtained by an inspection of the pharynx.

#### DISORDERS OF MOTILITY

The soft palate, uvula, levator palati, and the pharyngeal constrictors are supplied by the accessory fibers of the spinal accessory nerve distributed through the branches of the vagus. Knowledge of the innervation of the pharyngeal muscles has followed the experimental work of Sir Victor Horsley and Dr. Beevor. The motor neuroses of the pharynx are conveniently considered under two divisions: (a) spasmodic and (b) paralytic.

(a) **Spasmodic Neuroses.**—Spasm of the pharyngeal muscles is rare and is generally met with in patients with an unstable nervous system. Sir St. Clair Thomson divides the spasmodic neuroses into tonic and clonic types. The former occurs in hydrophobia, tetanus, tumors pressing on the bulb, tabes, acute local affections such as tonsillitis and quinsy, and functional disorders, usually hysteria (*globus hystericus*). Tonic spasm associated with pain is called cramp.

Clonic spasm of the levator palati (nystagmus of the palate) shows itself in a peculiar clicking sound audible to the patient and his friends. The movement may effect the soft palate or posterior pharyngeal wall, and be associated with synchronous twitchings of the arytenoid or rapid movements of adduction of the vocal cord. The contractions may be unilateral and recur at the rate of 100 to 200 per minute. The patient may localize them in the ear and the observer hearing them there may be tempted to locate the phenomenon as originating in the tensor tympani or the stapedius muscles, but in reality the sounds are caused by the opening and closing of the eustachian tube.

Insufficient action of the muscles acting as dilators of the eustachian tube may produce ear complications.

Spasmodic contractions are also met with in organic disease of the brain and may be associated with similar rhythmical clonic contractions of the tongue, pharyngeal muscles, esophagus, ear, and larynx, and with nystagmus of the eyes. Clonic spasm may be associated with fifth nerve irritation, poisoning by lead, mercury, alcohol, and strychnin, paralysis agitans, disseminated sclerosis, and is occasionally seen in facial cramp (convulsive tic) and in mild epilepsy. Tonsil irritation especially of the lingual tonsils is occasionally intimately related to the spasm. Transient tonic spasms may follow the use of the eustachian catheter, or a morsel of food swallowed too hastily, or be associated with pharyngitis lateralis and acute lingual tonsillitis. They do not last as long as clonic spasms but if persistent may be mistaken for gullet stricture.

(b) **Paralytic Neuroses.**—It is important to distinguish between true paralysis of the palate and the similar appearances resulting from inflammatory and mechanical interferences with movements due to syphilis and other forms of local disturbances in the pharynx and nasopharynx. The author was consulted by a young woman who had difficulty in swallowing with occasional regurgitation of fluid through the nose and a somewhat muffled voice. A form of pharyngeal paralysis had been diagnosed but the real trouble was mechanical due to a lingual thyroid at the base of the tongue.

The complete removal of the faucial tonsils not infrequently results in contraction and even distortion of the soft palate. Insufficiency of the soft palate is a rare condition that may produce some symptoms very much like a true palsy, such as fluids regurgitating in the nose on swallowing and a muffled voice. Brown Kelly has reported 4 cases of this rare condition. The hard palate was normal but the soft palate was too short to permit deglutition. True palsy is often due to postdiphtheritic neuritis and comes on during convalescence. Severe pharyngeal anginas without Klebs-Löffler's bacillus may produce a similar neuritis, and influenza too may produce a similar change.

Paralysis due to central lesions are not at all uncommon. It is found in bulbar lesions, glossolabiolaryngeal paralysis, apoplexy, embolism, tumors pressing on the bulb, syringomyelia of the bulb (syringobulbia) gumma, and endarteritis obliterans. When the lesion is central there is frequent interference with movements of the tongue, larynx, sternomastoid or trapezius. Basal meningitis due to syphilis or associated with new growths of the cranial base may implicate the nerve-roots before their emergence from the jugular foramen and so cause paralysis. In the glossolabiolaryngeal paralysis (chronic bulbar disease) the paralysis generally begins in the tongue, subsequently involving the lips, velum palati, and pharyngeal constrictors, and the abductors and internal tensors of the vocal cords. In tabes a form of pharyngeal crisis has been noticed in which repeated swallowing movements are made.

Encephalitis lethargica now appears to explain many obscure cases of palsy and one could hardly expect the pharynx to be immune. Tumors in the neck pressing on the branch of the vagus before the pharyngeal branches are given off are occasionally a factor. Diseases of the mouth of the esophagus cause pharyngeal spasm, and painful lesions in the laryngopharynx

may produce symptoms suggestive of pharyngeal paralysis. In the old or very feeble muscular weakness preventing the grasping action of the pharynx from propelling the bolus of food is occasionally present, but is not a true paralysis. The paralysis of the soft palate may be unilateral and in such patients Moure uses the diagnostic term of "velopalatine hemiplegia." It may occur in fractures or tumors at the base of the skull, tuberculous or cancerous adenitis or be a sequel to bulbar paralysis or syringomyelia. Diphtheritic paresis may be on one side and gradually pass to the other half of the palate.

Intracranial lesions involving the hyoglossus and other nerve-roots at the base of the brain may cause Avellis' syndrome—pharyngolaryngeal or glossopharyngolaryngeal paralysis—combined with paralysis of the soft palate and if the hyoglossus nucleus is involved one-half of the tongue as well. In the syndrome of Avellis the lesion is usually a hemorrhage in the area of the nucleus ambiguus and the spinal fillet in the medulla, but may be produced by cerebrospinal syphilis, tuberculosis, or syringomyelia. Charles J. Imperator has recorded in the Transactions of the American Laryngological Association in 1924, 3 cases in which there was paralysis of the soft palate and the left vocal cord, and partial paralysis of the constrictor of the pharynx. No isolated lesion of the glossopharyngeus has been recorded.

*Symptoms.*—If the paralysis is slight only little discomfort follows, but if the palate movement is seriously curtailed the voice is altered and fluid tends to regurgitate through the nose owing to the failure of the palate to contract, and the patient may be unable to whistle, suck or blow out his cheeks. Vocalization is altered in that he cannot pronounce the word "wrong," "rub" becomes "rum," and "egg" is pronounced "eng," a *b* becomes an *m* and a *d* an *n*. Closing the nostrils temporarily improves the voice. On examination the palate hangs limply downward and forward. No movements take place when the patient is asked to say "ah," and no reflex action follows on touching the velum. Sensation may or may not be altered. If the paresis is unilateral, the unaffected side is drawn up with the uvula directed to the same side while the other half hangs limply. Escat describes symptoms of tachycardia due to insufficiency of the vagospinal and a cough of a whooping nature. When the paralysis is unilateral and due to a motor nerve lesion, and not a pharyngeal inflammation, Moure says, similar changes are always found in the larynx. Hysterical cases can usually blow out well and contraction of the palate follows stimulation. St. Clair Thomson refers to hysterical patients thrown in contact with those having palate paralysis, reproducing their cleft-palate voice very successfully.

*Duration and Prognosis.*—When the disease is progressive and involves the pharyngeal constrictors deglutition becomes more difficult, especially for fluids which may overflow into the larynx and produce a spasm or even inhalation pneumonia. In progressive muscular atrophy and bulbar paralysis the epiglottis does not shut down and food may reach the larynx, but if sensation is not impaired and the ventricular bands act promptly very little will reach the glottis.

Diphtheritic paralysis usually recovers after a time, but if not at the same rate in both sides it simulates a unilateral paresis at one stage of the disease. When the disease is of central origin it follows the course of that

disease which in most cases is one of very grave consequence to the patient.

*Treatment.*—In only a small proportion of cases can much hope be offered by therapeutic measures. Postdiphtheria cases should have strychnin and local faradization. Gargling, humming, and vocal exercises are also beneficial. Hysterical patients respond best to measures in which suggestion plays the most prominent part. Attention to the general health is essential. Syphilitic patients require appropriate treatment, and if the disease has not already produced too much damage to the intracranial pathway, pronounced benefit may follow. Tumors and destructive lesions of the bulb offer very little hope and such patients do not usually survive very long. When the pharyngeal constrictors are involved the stomach-tube may be required. If the lesion is in the neck and admits of surgical interference, benefit may follow operative treatment, but such instances would be rare surgical experiences.

PERRY GLADSTONE GOLDSMITH

### DISEASES OF THE NASOPHARYNX

**Examination** of the nasopharynx is made by reflecting light upon a rhinoscopic mirror held back of the soft palate while the person examined breathes quietly through the nose. The image on the mirror shows the posterior

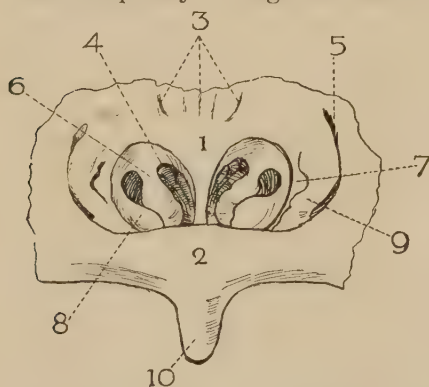


Fig. 187.—Posterior rhinoscopic image: 1, Vomer or nasal septum. 2, Velum or soft palate. 3, Pharyngeal tonsil. 4, Superior turbinate. 5, Rosenmüller's fossa. 6, Middle turbinate. 7, Eustachian orifice. 8, Inferior turbinate. 9, Eustachian eminence. 10, Uvula. (Mortiz Schmidt.)

narres with the septum in the middle line, the posterior ends of the superior, middle, and inferior turbinated bones covered with their mucous membrane; back of this in childhood, the pharyngeal tonsil with its medium fold and possibly the bursa pharyngea and at each side the pharyngeal opening of the eustachian tube, the eustachian eminence, and Rosenmüller's fossa (Fig. 187). Some portion of the nasopharynx may be seen by anterior rhinoscopy. Palpation of the nasopharynx with the finger in young children is often necessary to determine the condition of the pharyngeal tonsil; in adults, to determine the density or origin of a postnasal growth. Examination

may also be made with a pharyngoscope passed into the nasopharynx through the nose.

#### ACUTE NASOPHARYNGITIS

An acute inflammation of the nasopharyngeal mucous membrane, and as this is continuous with that of the nose and of the pharynx proper an acute inflammation of the nasal or pharyngeal mucous membrane, or both, is likely to be accompanied by the involvement of that of the nasopharynx.

An acute nasopharyngitis often precedes an acute rhinitis for a couple of days when it extends into the nasal cavity. In its milder form it may be confined to the nasopharynx and sometimes precedes an acute pharyngitis. As the nasopharynx of children contains adenoid tissue, we have with them in addition, a condition of adenoiditis which plays an important part in childhood. Acute inflammation of the nasopharyngeal mucous membrane in children is frequently a true tonsillitis of the pharyngeal tonsil. The temperature may be as high as 103° or 104° F., and the glands at the angle of the jaw may be enlarged. This condition is known as glandular fever.

A form of acute nasopharyngitis plays an important part in cerebrospinal meningitis. The microbe of this disease sets up a specific inflammation in the nasopharynx and secondarily a meningitis.

Acute exanthemata also produce an acute inflammation of the nasopharyngeal mucous membrane, especially of the lymphoid tissue in children.

**Etiology.**—The etiology of the simple acute nasopharyngitis of adult life is the same as that of acute rhinitis. People who lead sedentary lives, who overeat and indulge in alcohol and live in overheated houses, or who wear too much or too heavy clothing, are apt to be more susceptible to colds than are those who are much in the open air and have their houses properly ventilated and not overheated, who avoid fur coats and too heavy underwear. Exposure to drafts, having wet feet, cooling off too quickly after exercise are apt to produce sudden attacks of "cold." Those who live in dry cold climates are not susceptible to colds until they move to warmer climates. There seems to be a certain contagiousness about taking colds, seen frequently in the rapid spread of colds in the midst of families, although no germ has been isolated which is supposed to produce coryza. Chlorine, bromine, and other irritating gases often produce a traumatic rhinitis and nasopharyngitis.

**Pathology.**—The mucous membrane is thickened, red, and dry at first after which the swelling diminishes and there is increased secretion which contains a large amount of desquamating epithelium.

**Symptoms.**—In the simple form of acute nasopharyngitis, there is a burning and smarting sensation back of the palate which is intensified by swallowing.

**Prognosis.**—It subsides at about the same time as the accompanying acute rhinitis, in one to four weeks. Infection of the middle ear must be guarded against in acute inflammation of the nasopharynx, especially in children where the pharyngeal tonsil is implicated. Young children also swallow the secretion which often causes infection of the alimentary canal.

**Treatment.**—The treatment of acute nasopharyngitis is that of acute rhinitis. If seen early the patient should be put to bed and free perspiration induced. This is usually accomplished by giving a hot foot-bath, hot drinks, and 10 grains of aspirin or of Dover's powder. This treatment will not accomplish anything after the condition is well established, that is, after the second day. Internally, some form of rhinitis tablets should be given every two hours to control the amount of secretion, those which contain  $\frac{1}{16}$  grain each of camphor, opium, aconite, and nitrate of potash are very good. The nose should not be irrigated during an acute obstruction of the nose. Irrigation when the nose is obstructed often produces middle-ear infection. To overcome the hyperemia of the nasal mucous membrane

and to encourage drainage, a weak solution of adrenaline (1 : 10000) or ephedrine inhalant should be sprayed into the nose with a hand atomizer, after which applications can be more safely made to the nasal and nasopharyngeal mucous membrane. Soothing oil sprays may be used to protect the nasopharyngeal mucous membrane. A pleasant oil spray is the following:

		Gm. or c.c.
R. Ol. menthæ viridis.....	℥xx.....	.60
Camphor.....	gr. xx.....	1.30
Eucalyptol.....	℥xx.....	1.30
Fld. albolene ad.....	℥iv.....	120.00

Argyrol and silvol, each in a fresh 25 per cent. solution, are recommended as a local application in these cases. As the treatment of acute rhinitis has been taken up in another chapter, more detail of its treatment cannot be given here. Autogenous vaccines appear to benefit some cases, others they appear to make worse. The use of prophylactic vaccines has not proved to be as beneficial as claimed by enthusiasts.

#### CHRONIC NASOPHARYNGITIS OR CHRONIC HYPERTROPHIC NASOPHARYNGITIS

**Definition.**—Like the acute inflammation of this region, chronic nasopharyngitis is seldom a distinct disease, but a part of a chronic inflammation of the nasal mucous membrane (hypertrophic rhinitis) or a part of a chronic pharyngitis. The condition may, however, be confined to the nasopharyngeal mucous membrane, or it may involve the pharyngeal tonsil and the pharyngeal bursa if the lymphoid tissue has not atrophied.

**Etiology.**—Deflection of the septum, hypertrophic rhinitis, in fact, all obstructive and inflammatory conditions of the nose and paranasal sinuses, predispose to chronic nasopharyngitis. Unhygienic surroundings and poor nourishment seem to develop this condition, especially when the patients inhale large quantities of dust. Workers in tobacco, machinists, clothing cutters, and stone cutters suffer frequently from a chronic nasopharyngitis.

**Pathology.**—The mucous membrane is swollen, is usually red, with increase of connective tissue.

**Symptoms.**—There is constant hacking and attempts to clear the throat of a very tenacious mucus which often produces retching and vomiting, especially in the morning.

**Diagnosis.**—Examination shows the membrane to be covered with thick mucus which has lost much of its moisture. The mucous membrane is very red and thickened.

Chronic postnasal catarrh, chronic catarrh of the nasopharynx or American catarrh, are terms which came into use years ago when all droppings of mucus, or pus, from behind the soft palate were supposed to have their origin in the nasopharynx, and it was called catarrh because the mucus dropped into the throat. Most such secretion, we know now, has its origin somewhere else and the diagnosis of nasopharyngitis consists in eliminating all other diseases which cause the discharge of mucus or pus from or through the nasopharynx. These diseases are hypertrophic rhinitis, paranasal sinusitis, especially maxillary sinusitis, posterior ethmoiditis, and sphenoiditis. It is often difficult to find the source of a postnasal discharge.

**Prognosis.**—When the chronic condition of the nasopharynx depends upon the condition of the nasal mucous membrane, improvement generally

follows treatment of the nose with proper local treatment of the nasopharynx. If the condition is due to, or aggravated by, the occupation of the patient, no improvement can be expected until the patient is induced to change his occupation. Excessive use of alcohol and tobacco should be discontinued.

**Treatment.**—All nasal and paranasal sources of the condition should be corrected. When there is a remnant of the pharyngeal tonsil it should be removed and all adhesions in the nasopharynx destroyed. The catarrh itself should be treated daily by thoroughly cleansing the nasopharynx by postnasal irrigation with normal salt solution, after which the nasopharynx should be swabbed with some stimulating pigment such as the following:

		Gm. or c.c.
R. Iodi .....	gr. iv .....	.26
Potassii iodidi .....	gr. xl .....	2.60
Glycerini .....	℥j .....	.30.00

This should be applied to the membrane on a cotton-wound applicator of which about  $1\frac{1}{2}$  inches are bent at right angles to the shaft of the applicator.

#### ATROPHIC NASOPHARYNGITIS (Nasopharyngitis Sicca)

**Definition.**—A chronic inflammation of the nasopharyngeal mucous membrane in which the membrane is atrophied.

**Etiology.**—Various theories have been advanced from time to time, but no definite cause can be assigned for it. It is usually a part of an atrophic rhinitis and starts in childhood, as a rule, never after the twenty-fifth year. It may be the last stage of an hypertrophic nasopharyngitis. Atrophic rhinitis is frequently associated with a paranasal sinusitis.

**Pathology.**—The mucous membrane is atrophied and almost completely replaced by connective tissue. The ciliated epithelium is replaced by squamous epithelium.

**Symptoms** are much the same as those of the chronic hypertrophic nasopharyngitis, except that they are more severe because the mucus is deposited upon the roof and side walls of the nasopharynx in thick dry crusts which produce the feeling of a foreign body in the throat. These crusts often come away in large pieces and leave an excoriated surface which is soon covered with similar crusts which, like those of atrophic rhinitis, have a very offensive odor.

**Diagnosis.**—On examination of the nasopharynx the crusts are seen, and the condition is generally found to be part of an atrophic rhinitis and can often be seen extending into the pharynx and larynx. A healing tertiary syphilitic ulcer on the posterior pharynx wall covered with dry secretion may be taken for atrophic nasopharyngitis. This ulcer, however, has a characteristic raised border which can be seen after the crusts have been removed; and if the ulcer has already healed at the time of examination a characteristic stellar scar will be found. If there is any doubt about the diagnosis, a Wassermann test should be made and other evidence of syphilis sought for in other parts of the body.

**Prognosis.**—Careful attention to cleansing and stimulating the nasopharyngeal mucous membrane will make the patient comparatively comfortable. A cure cannot be expected. The tendency to crust formation grows less as age advances.

**Treatment.**—The treatment of atrophic nasopharyngitis is that carried

out for the treatment of atrophic rhinitis with special attention to the removal of crusts from the nasopharynx and stimulating the nasopharyngeal mucous membrane as well as that of the nose. By means of a wash-bottle or fountain syringe, the nose should be thoroughly irrigated with an alkaline solution made of a couple of teaspoonfuls of equal parts of biborate and bicarbonate of soda added to a pint of warm water and should be used twice daily until all crusts are washed away. If the discharge is very offensive, as it usually is, the irrigation should be followed once daily, at least, by a solution of permanganate of potash, 2 grains to a pint of water used in the same manner. The nose and nasopharynx, following this, should be swabbed with the iodine preparation mentioned when speaking of the treatment of chronic hypertrophic nasopharyngitis. The patient can be taught to do this after a time. The object of the treatment is to keep the mucous membrane moist and free from crusts, as the atrophied mucous membrane cannot be restored to its normal state. Various vaccines have been recommended for the treatment of nasal and nasopharyngeal atrophic conditions. Various operations have been and are being performed to narrow the internasal space. Sugar has been suggested as a very beneficial form of treatment for atrophic rhinitis. The sugar is used either as an insufflation five or six times daily or a 25 per cent. solution of glucose in glycerine is applied to the nasal and nasopharyngeal mucous membrane.

#### ADENOIDS

**Synonyms.**—Adenoid vegetations, enlarged third or Luschka's tonsil.

**Definition.**—Adenoids appear in two forms, either as a simple enlargement of the pharyngeal tonsil, or as an hypertrophy of all the lymphoid tissue of the nasopharynx. The term "adenoids" is used whenever the pharyngeal tonsil is large enough to produce pathological symptoms. This often depends upon the relation of the size of the lymphoid mass to the size of the nasopharynx. A medium-sized pharyngeal tonsil might produce no symptoms in the spacious nasopharynx of an adult but would produce marked obstruction to nasal breathing in a child with a small nasopharynx.

**Etiology.**—The causes which are active in the development of adenoid tissue are not definitely known. A normal increase in the size of the pharyngeal tonsil commences at about the fifth year of child life and continues until puberty when the normal tonsil usually atrophies, but when diseased it persists until the twentieth year, in some cases throughout life. There may, however, be an abnormal amount of tissue in the nasopharynx of children as early as the third month. The normal pharyngeal tonsil is present at birth. Pathological enlargement of the pharyngeal tonsil is usually associated with chronic enlargement of the faucial tonsils.

Adenoids are also present as a part of the lymphoid hypertrophy in the state known as status lymphaticus when all the lymphoid tissues and glands of the body are enlarged. There is no known hereditary influence in the production of adenoids, but they are often seen in abundance in several members of the same family. They appear in all races but are more prevalent in northern climates than in warm southern countries. They are abundant in children with deflected nasal septi, or high arched palates where the adenoids probably are the cause and not the result of the condition. The pharyngeal tonsil, like the faucial tonsils, takes part in all

acute inflammations of the nose and throat. They are acutely inflamed after the exanthemata like measles, scarlet fever, diphtheria, whooping-cough, and during an attack of influenza. These acute inflammations seem to be a prominent feature in their chronic enlargement.

**Pathology.**—Adenoid vegetations are an hypertrophy of the lymphoid tissue of the nasopharynx. This hypertrophy may be confined to an enlargement of the pharyngeal tonsil, or it may extend to all the lymphoid tissue found in the nasopharynx and occurs as a lobulated mass with several furrows extending anteroposteriorly and may be large enough to fill the nasopharynx and extend below the soft palate. The lymphoid tissue may be acutely inflamed and attain an enlargement several times its usual size and may have in its folds yellow and white exudate similar to that found in the crypts during a follicular inflammation of the faucial tonsils and consisting of various bacteria, epithelium, etc. The pathological changes producing the usual enlargement are not from increase of the connective tissue, but are due to extensive lymphoid development, except when the growth is more dense when the lymphoid tissue is associated with increase of connective tissue. After an acute inflammation of the pharyngeal tonsil, the infectious material remains in the fissures of the tonsils the same as it does in the crypts of faucial tonsils but it does not seem to cause focal infection to the same extent.

**Symptoms.**—The symptoms of adenoid vegetations are due first, to nasal obstruction and mouth breathing, second, to eustachian obstruction, third, to subacute and acute inflammation of the lymphoid tissue and extension of this inflammation to other organs.

The symptoms of nasal obstruction and mouth breathing in children are characteristic. The dull facial expression with the open mouth, thick lips, pinched nose, and dead toneless voice all point in childhood to adenoids. These children are restless in their sleep, snore and wake up frequently. They are listless, their memories are bad which interferes with their progress at school. The slightest mental effort produces a dull headache which incapacitates them for mental work. This mental lethargy has been named, nasal neurasthenia or aprosexia by Guye of Amsterdam. These obstructive symptoms, of course, vary with the size of the growth. Where there is a marked nasal obstruction for a long time, children often develop a deformity of the chest known as pigeon breast. Young infants developing adenoids from the third to the sixth month are unable to nurse and are consequently undernourished and become emaciated.

*Eustachian obstruction* may be found associated with nasal obstruction, but may be due to a moderate degree of adenoids when this tissue is located at the side of the nasopharynx. Obstruction of the eustachian tube produces catarrhal otitis media. The tympanic membranes are retracted and the patient does not hear well. The loss of hearing may be the first symptom of adenoids and it is an important one.

The symptoms of inflammation and infection occur as frequent colds, and in the more severe forms there is a regular acute adenoiditis with high fever and deposit of secretion in the folds of the pharyngeal tonsil. During these attacks the infection often extends to the middle ear as an acute purulent otitis media and the child comes to the physician with an acute ear trouble or later with a discharging ear. These inflammations, etc., likewise, are the cause of complications such as croup and bronchitis and

young children swallow the secretion which produces various gastrointestinal conditions.

*Indirect Symptoms.*—Children with adenoids often have peculiar facial movements or twitchings. Asthma and nocturnal enuresis have been mentioned by many as symptoms of adenoids.

**Diagnosis.**—When a patient is presented for examination with the open mouth and characteristic facial expression, a provisional diagnosis has already been made and when we look into the oral cavity, we note that the teeth are irregular and that the palate is highly arched, that the faucial tonsils and the follicles on the posterior pharyngeal wall are usually enlarged. A mass of clear mucus is usually seen sliding down the posterior wall of the pharynx. Further examination should be made according to the age of the patient. Digital examination (palpation) is usually necessary in the case of young children and is accomplished by the physician holding the child's head firmly under his left arm and pressing the left cheek between the teeth of the opened jaws with the left forefinger, inserting the right forefinger into the mouth until the posterior wall of the pharynx is reached, then turning it up behind the soft palate into the nasopharynx where adenoids are recognized by their soft consistence. In children over six years old it is usually possible to make a posterior rhinoscopic examination in the usual way. When present, adenoids appear as a rounded mass on the posterior wall with furrows extending anteroposteriorly (Plate V, 1). If of any size they cover the upper part of the septum on the rhinoscopic mirror and if large they may completely hide it and the image of the posterior nares.

**Differential Diagnosis.**—All children complaining of nasal obstruction, where the nasal cavity is normal, are usually found to have adenoid vegetations. It is seldom necessary to differentiate between adenoids and nasopharyngeal fibromata or the pedunculated nasopharyngeal polypi. Both these varieties of tumor occur occasionally in older children and young adults and in the male sex. Fibromata are seen on the posterior wall of the nasopharynx as smooth round tumors and are hard on palpation. They produce complete nasal obstruction which adenoids never do. Nasopharyngeal polypi are seen on inspection in the anterior part of the nasopharyngeal cavity, are semitranslucent, pale blue or white in color, and are pedunculated and soft on palpation. Sarcomata in young children may easily be mistaken for adenoids if seen before they extend below the palate or into the nose as they are of about the same density. If removed by mistake for adenoids they return very rapidly. Sarcomata in older patients are of rapid growth and carcinomata do not develop until after the thirtieth year.

**Prognosis.**—The results following the removal of adenoids are always so favorable that no hesitation should be had in recommending an operation if they are at all enlarged and especially if there is any retraction of the tympanic membranes. The beneficial effects of the operation on mouth breathing are often immediate and normal respiration is restored. There are many cases, however, where it is necessary to remind the patient to breathe through the nose. It is often necessary to close the child's mouth at night by tying a bandage over the head and under the chin to force nasal respiration. Soon after the operation most of the symptoms disappear. The nasal discharge disappears except when the paranasal sinuses are infected. The cough disappears and the patients rest well at night. The effect

upon the hearing is usually noticed at once and the discharging ears generally heal with proper treatment in a short time. The voice loses its dead toneless character and if there are no other causes for nasal obstruction, ill health, and stupidity, the children are in a short time perfectly well.

**Treatment.**—When a diagnosis of adenoid vegetations has once been made, no treatment but surgical removal can accomplish anything. Adenoids should not be removed while the child is suffering from an intercurrent malady or has a recently developed fever because an operation may set up an acute septic condition. They should not be removed during the

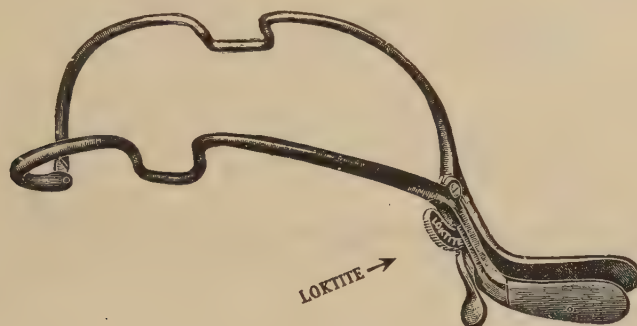


Fig. 188.—Jennings' mouth-gag.

acute stage of a middle-ear infection. The blood coagulation time should be less than four minutes and hemophiliacs and patients presenting signs of status lymphaticus should not be operated upon. In suspected cases of the latter condition a Roentgen-ray examination of the thymus gland should be made.

*Method of Operating.*—The patient is prepared in the usual way for an operation and, if a child, is given a general anesthetic in order to do the operation thoroughly. An adult can be better managed with a local anesthetic applied to the nasopharynx and the procedure is the same as under a general anesthetic.



Fig. 189.—La Force adenotome.

When a general anesthetic is given, the patient is placed in the prone position or on the side, with the head slightly lowered and when sufficiently anesthetized, a mouth-gag (Jennings, Fig. 188) is placed in the mouth and under good illumination of the pharynx the operation is carried out. If a tonsillectomy is to be done at the same time it should precede the adenoid operation. The pharynx should be carefully palpated with the index-finger to determine the size of the growth and any extensions of it which may reach the lateral walls of the nasopharynx, or Rosenmüller's fossa; any adhesions should be separated with the finger. Various instruments have been devised for the removal of the growth including forceps, curets with

and without a cage, and adenotomes such as the La Force adenotome (Fig. 189) which is the safest and most satisfactory instrument to use with a child under general anesthesia. With the box of the La Force adenotome open, it is passed up back of the soft palate until it is felt to touch the nasal septum, when it is closed and the instrument when withdrawn will be found to have removed the whole, or the greater part, of the adenoid mass;



Fig. 190.—Gottstein's curet.

any adenoid tissue still remaining in the pharynx should be removed by again introducing the same instrument or by using a curet. When a curet such as Gottstein's (Fig. 190) or Beckman's (Fig. 191) or a caged curet



Fig. 191.—Beckman's curet.

such as Delastanche's (Fig. 192) is used, it is introduced in the same manner as the adenotome and it is then brought down and forward by a sweeping movement. The handle of the curet should be elevated toward the patient's face as soon as the adenoid mass is severed, so as not to strip the sub-

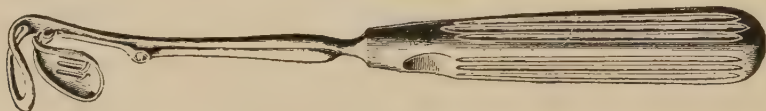


Fig. 192.—Delastanche's curet.

mucous tissue from the posterior pharyngeal wall. If a caged curet has been used, the adenoid mass will be in the cage of the curet and if a Beckman's or Gottstein's is used, the tissue may come away or it must be taken out with a pair of forceps. Sometimes the mass may remain attached to the posterior pharynx wall at the level of the soft palate when it must be separated with a pair of scissors to prevent its being drawn downward. The removal of adenoids with postnasal forceps is not usually satisfactory unless the blades of the forceps are broader than usual so that the whole of the adenoids, or most of them, can be grasped at one time. Brandegees (Fig. 193) and similar forceps are in this respect the most satisfactory. When adenoid forceps are used, care should be taken not to grasp the posterior margin of the nasal septum.

The whole of the vault should be cleared of all lymphoid tissue before the patient comes out of the anesthetic. During the operation, especially if the adenoid operation has been preceded by a tonsillectomy, the pharynx should be kept free of blood by the use of a vacuum suction tube held in the pharynx by a nurse standing on the side of the patient opposite the operator. Bleeding is usually quite brisk for a few seconds following the removal of the adenoid mass but generally subsides promptly, otherwise, pressure

must be made in the nasopharynx with a gauze sponge of proper size grasped by a pair of adenoid forceps; this, however, is rarely necessary. It is seldom necessary to "plug" the nasopharynx after an adenoid operation.

After the operation has been completed, the patients should be rolled on the side with their faces swathed in a towel saturated with ice water and when the operator is satisfied that the blood has ceased to flow, the gag should be removed and the patients sent to the room where they should be placed on the side or stomach.

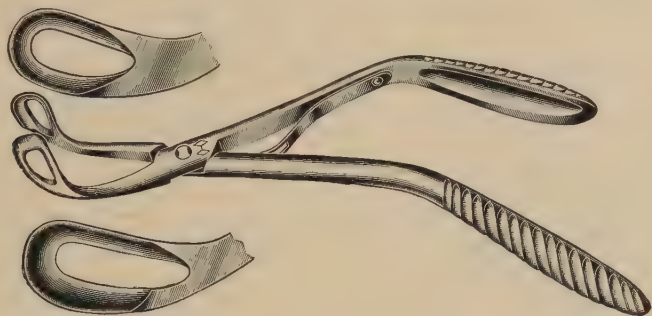


Fig. 193.—Brandegee's forceps.

After-treatment is not usually required as irrigation is likely to set up a middle-ear infection, but the patient should remain in bed for twenty-four hours. Children should not be allowed to go to school until the wound is healed on account of the risk of infection.

#### THORNWALDT'S DISEASE

*Pharyngeal bursitis* and *cysts of the bursa pharyngea* are known collectively as Thornwaldt's disease. They are different manifestations of the same pathological condition.

**Anatomy.**—The bursa pharyngea is a pouch or recess found at times in the middle line of the nasopharynx at the lower part of the pharyngeal tonsil. Some believe it to be the remains of a passage found in fetal life between the nasopharynx and the hypophysis (Rathke's pouch). It is probably a pocket formed by adhesions of the median folds of the pharyngeal tonsil.

#### PHARYNGEAL BURSTITIS

**Definition.**—When the bursa is chronically inflamed there is a discharge of mucus into the nasopharynx with the formation of crusts about the opening constituting a form of chronic nasopharyngitis.

**Symptoms.**—Dropping of offensive mucus into the nasopharynx.

**Diagnosis.**—With the rhinoscopic mirror a depression is seen on the posterior wall from which a discharge is coming.

**Treatment.**—Consists of the application of a strong solution of nitrate of silver to the depression and the removal of any adenoid tissue which may surround it.

#### CYSTS OF THE BURSA PHARYNGEA

**Definition.**—The sides of the bursa often adhere and form a retention cyst. These cysts vary in size and when small, they are often removed as a part of the adenoid tissue and are not recognized before their removal. When allowed to develop in neglected cases, they often attain considerable

size and contain inspissated secretion consisting of cholesterin crystals and degenerating cells.

**Symptoms.**—When large they produce nasal obstruction and the symptoms found with adenoid vegetations.

**Diagnosis.**—On the rhinoscopic mirror larger cysts appear as glistening yellow tumors projecting from the posterior wall of the nasopharynx. They are distinguished from fibromata by their color and glistening surface and want of density.

**Treatment.**—Their removal with an adenoid curet is easily accomplished.

#### FOREIGN BODIES IN THE NASOPHARYNX

**Definition.**—Food sometimes lodges in the nasopharynx during an attack of vomiting, or it may lodge there when food is regurgitated into the nasopharynx when the soft palate is paralyzed (postdiphtheritic paralysis), or metallic substances held in the mouth are partly swallowed many times and regurgitated into postnasal space.

**Symptoms.**—The presence of a foreign body somewhere in the throat is usually known by the patient but he may not be conscious of what part of the throat in which it is located unless it is very large, for the sensation of a foreign body in the nasopharynx may be referred to the larynx. One is, however, usually conscious of something in the upper and back part of the throat if it is large enough to cause nasal obstruction and has come on suddenly during an attack of vomiting.

**Examination.**—If the foreign body is large it may be seen projecting from behind the soft palate into the pharyngeal cavity. With patience and encouragement, an examination of the nasopharynx can be accomplished in a short time. Sometimes it is necessary to spray a weak solution of cocaine into the nasopharynx through the nose.

**Treatment.**—Foreign bodies can generally be removed from the nasopharynx by passing a pair of adenoid forceps through the mouth into the nasopharynx and grasping the object between the blades of the forceps.

#### BENIGN TUMORS OF THE NASOPHARYNX

The benign tumors are nasopharyngeal polypi and nasopharyngeal fibromata. Rarely have papillomata, adenomata, chondromata, and dermoid polypi been found in the nasopharynx.

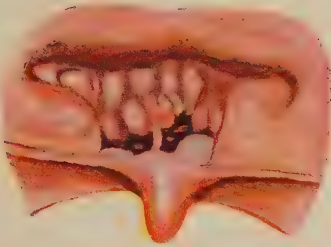
#### NASOPHARYNGEAL POLYPI

**Definition.**—Under this heading are included the pedunculated growths found in the nasopharynx. They originate either from the lateral walls of the nasopharynx or extend into the nasopharynx through the posterior nares. The latter originate either in the maxillary antrum (antrochoanal polypi), from the middle turbinated bone or from the posterior part of the nasal septum.

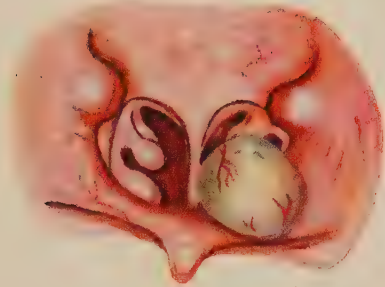
**Etiology.**—The causes of nasopharyngeal polypi are not known and like fibromata occur during adolescence. Those originating in the nose occur during adult life. No definite cause can be found for their occurrence.

**Pathology.**—Those having their origin on the side walls of the nasopharynx differ from those originating in the nose, are firmer and denser, that is, they contain more connective tissue and are not edematous, while those extending from the nose are the edematous nasal polypi. If these

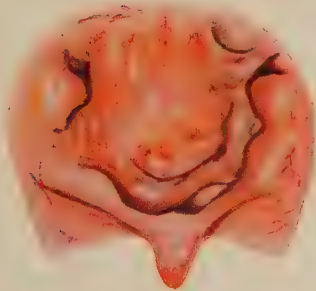
PLATE V



1. Adenoid vegetations in a young man twenty-one years old who had complained since his tenth year of nasal obstruction and loss of hearing. (From Mikulicz and Michilson's Atlas of Diseases of the Mouth and Pharynx.)



2. Nasopharyngeal polypus as seen by posterior rhinoscopy in a young man twenty-three years old. (From Grünwald's Atlas, and Epitome of Diseases of the Mouth, Pharynx and Nose.)



3. Fibroma of the nasopharynx as seen by posterior rhinoscopy in a boy, aged twelve. (From Grünwald's Atlas and Epitome of Diseases of the Mouth, Pharynx and Nose.)



4. Fibrosarcoma of the nasopharynx in a seventeen year old boy. (From Mikulicz and Michilson's Atlas of Diseases of the Mouth and Pharynx.)



edematous nasopharyngeal polypi have attained much size before they come under observation they may have undergone cystic degeneration.

**Symptoms.**—The first symptom manifesting itself is nasal obstruction, usually of one side, generally accompanied by catarrhal discharge. As the growth enlarges and reaches downward, there is considerable discomfort.

**Diagnosis.**—A bluish-white tumor is seen on the rhinoscopical mirror which, more or less, fills the nasopharyngeal cavity (Plate V, 2). Differentiation should be made between those originating in the nasal cavity and those attached to the side walls of the nasopharynx. Both are pale blue or white in color, and both are pedunculated. The origin of the growth in the nose or nasopharynx can usually be made out by inspection. Sometimes it is necessary to make a digital examination. When an antrachoanal polypus is suspected, its origin in the antrum can be determined by transillumination of, or by radiography of, the sinuses. The corresponding antrum will be opaque. Either form of polypus is distinguished from a nasopharyngeal fibroma by the latter's pink or red color, immobility, density, and by frequent attacks of hemorrhage into the mouth. These tumors are easily distinguished from a posterior hypertrophy of the lower turbinate which is seen as a rounded mass with a mulberry-like surface and occupying the position of the posterior end of the lower turbinated bone. Sarcomata are much like fibromata, but are not so dense and bleed frequently. Carcinomata are dark red and accompanied by metastatic glands and do not usually produce nasal obstruction.

**Treatment.**—After thorough local anesthesia of the nasal and pharyngeal mucous membrane has been produced either form of nasopharyngeal polypi can usually be removed by passing the loop of a nasal snare around the polypus. After the loop is passed through the nose, a finger passed into the nasopharynx may be able to push the loop around the polypus, when the growth may be either cut off or avulsed by closing the wire firmly about the growth and using traction on the handle of the snare. If it is found impossible to pass a snare loop around the tumor, it may be removed by passing a pair of adenoid forceps, behind the palate and grasping the growth and removing it by avulsion.

#### FIBROID TUMORS OF THE NASOPHARYNX

**Definition.**—Nasopharyngeal fibromata are comparatively rare and have their origin from the periosteum, usually of the posterior upper wall of the nasopharynx and sometimes from Rosenmüller's fossa. They occur usually as a single sessile tumor.

**Etiology.**—There is no known cause for the development of nasopharyngeal fibromata. They develop between the tenth and fifteenth year and have a tendency to degenerate by the twenty-fifth year of life. They occur more frequently in the male sex.

**Pathology.**—These growths are made up chiefly of fibrous tissue with few cells and an unusual number of blood-vessels, they grow rapidly and cause absorption of bone but not infiltration of the surrounding tissue. They are not complicated by enlarged glands except when there is infection of the surrounding mucous membrane. They are described as microscopic benign tumors which are clinically malignant.

**Symptoms.**—The first symptoms are due to nasal obstruction and the tumor usually attains a large size and produces mechanical difficulties before

it affects the general health. The voice becomes early like that of adenoid vegetations, and there are frequent attacks of epistaxis and expectoration of blood. Erosion from pressure, producing an offensive discharge, may occur later. As the growth increases in size external deformities are produced. When prolongations of the growth extend into the nose they produce the deformity known as "frog face." It may have an extension back of the eye and shove the eye forward. The tumor may extend down and interfere with deglutition and respiration and nearly always produces loss of hearing from pressure upon the eustachian eminence. This loss of hearing is usually an early symptom. When it grows large it produces pain from pressure on the cranial nerves and may extend to the brain and cause meningitis. Death occurs only when caused by hemorrhage, sepsis, exhaustion, or intercranial complications.

**Diagnosis.**—The tumor may press the soft palate forward or the lower end of the tumor may be seen behind and below the soft palate. When the tumor is not large enough to produce these signs it will be seen on the rhinoscopic mirror as a pink or red round tumor filling the postnasal cavity and hiding the usual landmarks (Plate V, 3). On palpation it is found to be of firm consistence and to be immovable and the base, although broad, does not extend to the whole of the nasopharyngeal wall.

As stated before these tumors are distinguishable from all others with the possible exception of the chondromata, which are very rare, by their firm structure. Gumma, on account of the position which it sometimes occupies on the posterior pharyngeal wall, might be mistaken for a fibroma, though it generally breaks down soon into a tumor with a crater-like necrosing cavity in its center. Fibromata differ from the simple polypi of the nasopharynx by their dark color and by their tendency to frequent attacks of hemorrhage. This must be borne in mind in making a digital examination. Malignant tumors have, likewise, the tendency to severe hemorrhage, but are softer in consistence and carcinomata are usually accompanied by metastatic glands.

**Prognosis** is grave especially if these growths attain any size before the twenty-fifth year when they have a tendency to disappear. They have been successfully removed without radical operation.

**Treatment.**—Small fibromata may be removed by the same methods as nasopharyngeal polypi either with a strong cold snare or the galvanic cautery snare through the nose or by avulsion through the mouth. After thoroughly cocaineizing the nasopharynx, the exact attachment of the tumor should be made out by inspection and palpation before attempting the operation. In children the operation should be undertaken under a general anesthetic. When the tumor is too large to remove in this manner, when it extends into the nose or down into the pharynx, its surgical removal becomes a very formidable operation not only on account of its anatomical position, but also on account of the possibility of a serious and sometimes a fatal hemorrhage. To completely remove such a tumor, a preliminary lateral rhinotomy, such as Moure's, is considered the best way to reach it through the nose, or the soft palate may be split to reach it through the mouth. After it is reached by either of these routes it is separated from the bone along with the periosteum from which it grows. Sometimes a preliminary ligature of the external carotid is done before the radical operation to prevent dangerous hemorrhage. The operator should always

be prepared to promptly plug the posterior nares after removal of a post-nasal fibroma.

Many years ago Voltolini in Europe, followed by Rufus Lincoln of New York, introduced the method of reducing the size of these tumors by electrolysis and then removing them with the galvanocautery snare. Other authors advised the injection of various substances directly into the tumor. Since radium and diathermy have come into vogue either as an aid to or as substitutes for surgery, they have been extensively used in the treatment of fibroid and other tumors of the nasopharynx. Radium emanations have been successful in not only reducing the size of these tumors and sometimes causing them to disappear, but especially in preventing the very serious hemorrhage which is so often a dangerous complication. Planting of seeds of radium emanations in a nasopharyngeal tumor can only be carried out by an operator familiar with the dosage of radium and having the technical skill of an operator in the nasopharynx. Radium emanations are sometimes used to reduce the size of the tumor and prevent hemorrhage before removing it surgically.

#### MALIGNANT TUMORS OF THE NASOPHARYNX

**Definition.**—Sarcomata, lymphosarcomata, endotheliomata, and carcinomata may occur primarily in the nasopharynx, and malignant growths in the nose, antrum, orbit, mouth, tongue or tonsils may secondarily involve the nasopharynx. Sarcomata are occasionally met with primarily in the nasopharynx and at all ages from childhood to old age. They usually have their origin from the posterior superior wall of the nasopharynx. Sarcomata grow very rapidly and soon fill the nasopharyngeal cavity producing much nasal obstruction and are apt to invade the adjacent nose and paranasal sinuses, do not break down but may have superficial ulceration.

Carcinomata originate usually from the side wall of the nasopharynx, especially from Rosenmüller's fossa and do not grow into the nasopharyngeal cavity until late, therefore, do not usually produce nasal obstruction but infiltrate the neighboring organs and are not usually found until after the thirtieth year and are apt to bleed freely and ulcerate or break down easily.

**Etiology.**—No known cause can be given for the development of these malignant growths in the nasopharynx.

**Symptoms of Sarcomata.**—On account of its rapid growth and central position in the nasopharynx, the chief symptom of sarcomata is rapidly developing nasal obstruction. Hemorrhage is another frequent symptom. Enlarged cervical glands are not an early manifestation except in the case of lymphosarcoma which is always accompanied by marked enlargement of the cervical glands, often with no symptoms pointing to the nasopharynx.

**Symptoms of Carcinomata.**—On account of their location in the tissues in the side wall of the nasopharynx, carcinomata produce symptoms due to pressure and infiltration of organs and nerves located in that part of the body. As the eustachian tube is the most superficially located organ found in this region, the first symptoms produced by a nasopharyngeal carcinoma are disturbance of hearing (catarrhal otitis media), and pain in the ear and mastoid. Carcinomata usually occur after the thirtieth year and if patients over thirty years present themselves with middle-ear catarrh, otalgia, and mastoid pain, without any visible evidence of inflammation of the middle ear, especially if these two symptoms are accompanied by hem-

orrhage from the throat, they have most probably a carcinoma of the corresponding side of the nasopharynx. These symptoms are, however, soon followed by enlargement of the cervical glands. All these symptoms may occur before an appreciable nasal obstruction is found.

Many symptoms produced by pressure on the organs in the nasopharynx may occur which do not point to the pathological condition being in the nasopharynx, as these symptoms may occur before there is any hemorrhage from the throat.

A nasopharyngeal carcinoma may involve the second, third, and fourth nerves, both the second and third branches of the fifth and the sixth cranial nerves, the gasserian ganglion and the sella turcica producing pain in the eye, trifacial neuralgia, and other symptoms depending on the nerve or organ involved (Gordon B. New's syndrome), or it may press on the nerves passing through the jugular foramen involving the ninth, tenth, eleventh, and twelfth cranial nerves, producing paralysis of the corresponding half of the palate, tongue, pharynx, larynx, and the sternocleidomastoid and the trapezius muscles (Jackson's syndrome). These symptoms may occur in patients without any nasal obstruction and before there is any hemorrhage into the throat and, consequently, the patients consult the ophthalmologists, neurologists, or internists; therefore, the tumors are not discovered unless a routine examination of the nasopharynx is made.<sup>1</sup>

**Diagnosis.**—Sarcomata are distinguished from fibromata by their being softer in consistence (Plate V, 4) and by making a microscopical examination of a section of the growth. Carcinomata should be suspected where patients present themselves after the thirtieth year complaining of pain in the ear and mastoid with loss of hearing without any visible middle-ear inflammation, especially if hemorrhage from the throat occurs. Carcinomata of the nasopharynx are easily overlooked in their earlier stages of development, unless it is recognized that where loss of hearing with pain in the ear and mastoid occurs in a middle-aged person where there is no acute inflammation of the middle ear, it usually means that a carcinoma or lymphosarcoma is developing in the nasopharynx. This fact should direct one's attention to this part of the throat even before the cervical glands become enlarged or before there is hemorrhage from the throat, which are both usually early symptoms and are found before the cranial nerves become involved. Severe pain either in the eye or ear, or trifacial neuralgia, is always found in carcinomata of the nasopharynx. The diagnosis should be confirmed by microscopical examination of a specimen removed from the tumor.<sup>2</sup>

**Prognosis.**—Sarcomata usually run a fatal course and when successfully removed they return either in the nasopharynx or some other part of the body. Carcinomata are usually fatal in a very short time and usually end in erosion of a blood-vessel or the patient dies of sepsis.

**Treatment.**—Sarcomata have been successfully removed surgically but they always return. The operations recommended for the treatment of sarcomata are the same as for nasopharyngeal fibromata. Sarcomata may be destroyed by radium emanations or by diathermy, but they return as after surgical removal. Radium emanations have proved useful in preventing hemorrhage and reducing and destroying the growth. As carcinomata are always fatal in a very short time, treatment must necessarily be only palliative. The columnar and basal-cell types are said to be more

favorable for irradiation than the squamous-cell type. The symptoms—pain, hemorrhage and discharge—are said to be relieved for a time by irradiation. Roentgen-ray treatment of the glands of the neck is being used along with the radium emanations introduced into the tumor in both carcinoma and lymphosarcoma. Diathermy has found considerable favor in the treatment of malignant tumors. The tumors are destroyed by coagulation, and the glands are dissected out with the diathermy knife. However, where a carcinoma of the nasopharynx is well advanced, which takes place in a very short time, the patient can only be relieved of the severe pain by morphine.

W. SCOTT RENNER.

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#### DIPHTHERIA

**Definition.**—An acute communicable disease usually characterized by the formation of a pseudomembrane and severe toxemia. It is caused by the Klebs-Löffler bacillus. The lesions are most frequently on the tonsils, in the nose, nasopharynx, uvula, pillars, and larynx, although other mucous membranes and wounded surfaces may be affected.

**History.**—Man has developed diphtheria from the earliest days. Pythagoras and Aretæus, Galen in the second century; Asclepiades in the second century and Paul of Ægina about 660 have written more or less extensively about the closing of the throat and the various clinical manifestations under the name of "Malum Egyptiacum." Epidemics in Rome in 856 and 1004 are described by Byronicos. Van Woerd reports one in Germany in 1517. Spain and France were invaded by diphtheria in the sixteenth century. A malignant septic form under the name of Garootillo spread through Spain and Italy about 1600. The sequelæ of paralysis and severe contagion are described by Villarial and Herrera.

Tracheotomy was performed in Italy and Spain about this time according to the writings of Francotte. In Britain, Jossilin described the disease in 1638, and it was subsequently commented upon by Home, Fothergill, Huxham, and Starr. An international congress under the decree of Napoleon in 1807 gave the first serious consideration to croup. Douglas, Bard, Colden, and others noted the advent of diphtheria epidemics in North America during 1735 to 1770. The literature up to the discovery of the Klebs-Löffler bacillus was elaborated by Virchow in 1847, who pointed out distinctions between varieties of pseudomembranous inflammation, and also by Bretonneau and Trousseau of the French School.

A new era of understanding began with the revolutionary discoveries of Klebs-Löffler, Behring, Roux, Yersin, Brieger, and Fränkel, beginning in 1883. Löffler confirmed the observations of Klebs regarding the presence of the bacillus in the false membranes of diphtheria and demonstrated the toxins in 1888. The researches of Roux and Yersin were exhaustive and

important factors in the study that led up to the discovery of antitoxin, consummated by von Behring in 1893.

**Etiology.**—It is absolutely and definitely established that the Klebs-Löffler bacillus is the cause of diphtheria. Its site of selection is most frequently the fauces and larynx. The micro-organism varies in appearance and size. This small germ is about the length of the tubercle bacillus 2.5–3 mm., Gram-positive, and stains irregularly with Löffler's alkaline methylene-blue. It may be taken by swab or sterile glass-slide or cover, from the affected mucous membrane and grown on blood-serum or ordinary culture-media in twelve to sixteen hours. It does not form spores. The ends of the organisms are frequently clubbed, although the size and form may vary

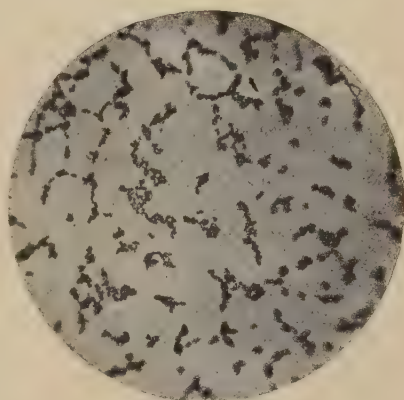
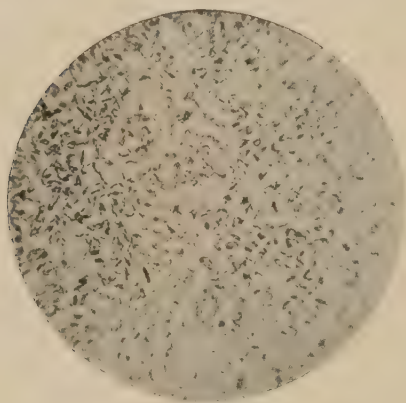


Fig. 194.—Löffler bacilli ( $\times 650$ ) (Koplik).      Fig. 195.—Pseudobacilli ( $\times 650$ ) (Koplik).

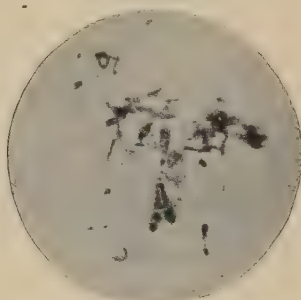


Fig. 196.—Involution forms of the Löffler bacillus ( $\times 650$ ) (Koplik).

greatly. Under favorable conditions of growth it may remain alive for months in fragments of dried membrane. The significance of its presence in healthy throats is not entirely understood. Carriers may often be eliminated by removing the tonsils and adenoids or treating a purulent ear. Nearly 70 per cent. of those attending diphtheria patients show a positive culture.

**Contagiousness.**—Epidemics vary in virulence, type, and contagiousness. The influence of individual resistance, mixed infection, atmospheric conditions, season, prevailing winds, dampness, and overcrowding of school and sleeping rooms, are important factors. Laryngeal invasion is frequently transmitted in its peculiar type. Infection is generally by direct contact.

It may come from milk or food, contaminated dishes or cooking utensils. Animal carriers as cats, dogs, pigeons, cows, and turkeys are occasionally the source. Playthings, clothing, slates, pencils, erasers, books, bedding, must be safely guarded and disinfected or destroyed. As the disease is particularly common from September to June in the north temperate zone the schools are the frequent disseminators of infection and require scientific regulation. Endemics or sporadic cases occasionally occur without scientific explanation. Children recovering from measles and scarlatina are especially susceptible.

**Predisposing Causes.**—Enlarged tonsils and adenoids, chronic pharyngitis and sinus disease render the individual much more susceptible. Certain families exhibit a lowered resistance. Ravolli claims a certain similarity, in this regard, between diphtheria and tuberculosis. He gives a record of

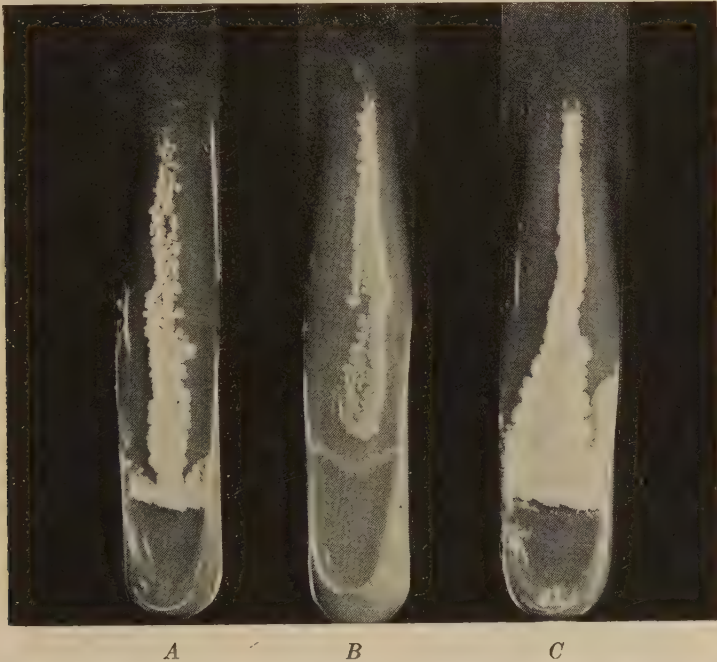


Fig. 197.—A, Pseudobacillus. B, True bacillus. C, Pseudobacillus (natural size). (Koplik.)

observations in a series of families who were predisposed to tuberculosis, where diphtheria seemed to appear without a recognizable source of contagion. Five exhibited positive tuberculosis at the time of infection. Hagenbeck and Hoppe-Seyler support the claims of Ravolli. The study of our case records at the children's pavilion of the Detroit Tuberculosis Sanatorium and elsewhere would show these observations were in error.

**Age.**—Before the advent of antitoxin, diphtheria was one of the most fatal diseases of early life with a mortality of 25 to 75 per cent. According to reports of Boards of Health, three to five years is the most susceptible age. In early infancy it is rare and the suckling babe is almost immune. A relative immunity rapidly supervenes after five years and increases to adult life although it is variable to conditions of its appearances as evidenced by its prevalence in army camps during the World War. Hensch in 1403

cases found none under three months. Freer in 4250 cases at Basle found 7 under three months, and 2.6 per cent. under one year. It is estimated that at least 75 per cent. occur before the tenth year.

*Sex.*—Sir Clair Thomson supports the idea of a preponderance among females on account of household duties and the habit of kissing. Our observations would show no distinction with regard to sex, although more boys are affected after seven years from increased opportunity for exposure.

*Incubation.*—This period is usually two to five days. Cases are recorded that developed within twenty-four hours to eight days after exposure.

*Season.*—The undulatory character of Klebs-Löffler infection has been noted for centuries. In our climate (Detroit) July and August show the least number of cases annually. The number gradually increases until the

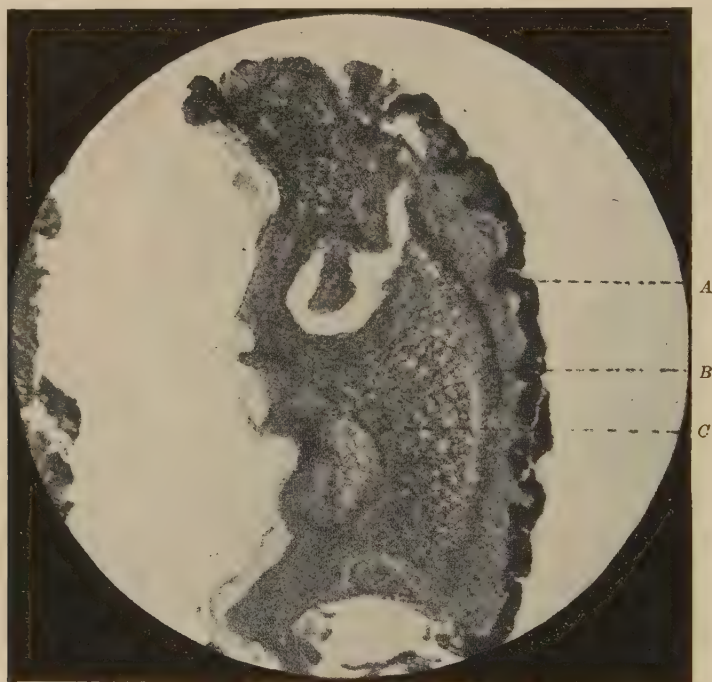


Fig. 198.—Photomicrograph of exudate from a case of pharyngeal diphtheria ( $\times 60$ ). A, Squamous epithelium undergoing necrosis; B, zone of leukocytes; C, fibrin. (From Shurly, *Diseases of the Nose and Throat*, published by D. Appleton & Co.)

opening of school gives an added impetus with January, February and March showing the maximum. The number then steadily declines until July.\* The virulence and character of the endemic may take on epidemic waves. The severity of the infection in spring may be worse than that of the fall or vice versa. The seasonal effects of high winds, dampness and rain, unpaved streets, crowded living quarters, and diminished sunshine are well-noted factors. Endemics of influenza and measles are frequently accompanied by diphtheria. Previous acute tonsillitis and other mixed infections often invite an attack. The social condition of childhood *per se* is not a marked influence as the children of the poor transferred to conditions of

\* Statistics Detroit Board of Health.

proper isolation and hygiene are not more susceptible than the children of the well to do.

**Pathology** (Fig. 198).—Laycock of Edinburgh, in 1858, predicted the parasitic origin of diphtheria but it required the discovery of the specific microbe and its toxins to demonstrate the true etiological and pathological sequence. The pathology is determined by three principal conditions: First, preparing the soil or part for attack associated with lowered biochemical resistance or local predisposing pathology. Second, lodgment of the specific germ. Third, biochemical changes in the presence of Klebs-Löffler bacilli resulting in the development of diphtheria toxins carried into the lymphatic and blood-streams. It is essentially primarily a local disease with later systemic manifestations.

The immediate pathological changes are found on the mucous membrane or wounded skin area in the form of hyperemia and swelling, giving the picture of so-called catarrhal inflammation. A layer of whitish-yellow pseudomembrane develops as a fibrous exudate. This earliest formation may be easily removed by peeling or with soda bicarbonate solution as in acute tonsillitis, or it may quickly undergo a necrotic change involving the mucous membrane and separated from it with difficulty and hemorrhage. The proliferated epithelial cells may be held by a fibrinous network appearing as a coagulation of fibrin which it resembles in microscopic properties. The inflammation may take on all degrees of severity and include ulceration. It most frequently invades the tonsils, larynx, and trachea, and nose. Rarely it is a primary infection of the bronchi, esophagus, ears, or gastro-intestinal tract. It is observed on the conjunctiva and anus, also the vagina. The adjacent lymphatic glands are usually swollen, tender, and inflamed.

The pathology of extension and systematic absorption may seriously or fatally affect the muscle of the heart with or without endocarditis and exhibit small or large hemorrhages with granular and fatty degeneration of the tissue substance. The pericardium also may undergo the same change with the development of an increase in fluid. The lungs may show various degrees and allocations of great congestion with small or extensive hemorrhage into the tissue. The pseudomembrane may follow the minute ramification of the bronchial tree and develop the pathology of edema, bronchopneumonia, infarctious emphysema, or atelectasis. Pleuritis is occasionally a complication with effusion of serum, blood, or pus. It produces the pathology of hemorrhagic parenchymatous, or interstitial nephritis with fatty and hyaline degeneration of the epithelial layers of the tubules and glomeruli. Hyaline, waxy, or hemorrhagic lesions may be observed in the liver and spleen. In the severe septic type the blood is sticky, brown, and slightly coagulable with an increase in leukocytes. In diphtheritic paralysis the nerves of the soft palate exhibit a granular degeneration with extravasation into their substances.

**Inoculability** can be proved by experiment and reported cases. Riesenman cites one of a bacteriologist while using a pipet drew a few drops of the Klebs-Löffler culture into his mouth. The period of incubation was forty-eight hours. Recovery took place with antitoxin. Cultures from the tonsils were positive for three weeks.

**Immunization.**—According to the theory of Ehrlich the contaminating toxin overcomes, under varying conditions, the natural antitoxin of the

blood. Many individuals are naturally immune through life or at different periods of their existence. This may be demonstrated by the Schick test. Gladys Ward employed this method in 150 children suffering from various diseases, 150 scarlet fever patients, and 50 maternity cases. The greatest number of positives occurred between the ages of six months and five years; 56 per cent. among scarlet fever patients, and 37 per cent. in general diseases. In the maternity cases 90 per cent. of the mothers and 96 per cent. of the babies gave a negative reaction. Ward regards the test of great value in deciding whether the patient is a carrier or really suffering from active diphtheria. Thus a negative reaction in a case of tonsillitis or nasal discharge would indicate that a patient was merely a carrier and not suffering from active diphtheria. It is claimed that the test is of value in showing what persons exposed to infection may be spared prophylactic antitoxin. It is recommended that only nurses with a negative Schick test should be employed in diphtheria wards. Weaver operated, for tonsils and adenoids, forty carriers in whom local washes failed to remove Klebs-Löffler bacilli and the bacilli disappeared very promptly. Gray and Meyer report 88 of 90 carriers free of diphtheria bacilli after twelve days treatment of the nose and throat using mercurochrome 1 to 2 per cent. solution.

The technic of the Schick test is as follows: The test is given intradermally with a No. 27 gauge needle after the skin has been cleansed with alcohol; 1/50 minimal lethal dose of toxin so diluted in physiological salt solution that this amount is given in 0.1 c.c. solution. Diluted toxin deteriorates rapidly at room temperature and should be freshly prepared within a week. The test is given in the upper arm and the control, which consists of the same material as the toxin except that it has been heated to 75° C. for five minutes, is given on the same arm 1 inch below the toxin. Weaver and Maher found that those having less than 0.04 unit of antitoxin per cubic centimeter of blood gave a positive Schick reaction. Crooks tested 808 persons of whom 69.6 per cent. gave positive reactions.

Immunization may be attained in most individuals by the use of a course of toxin-antitoxin. The diphtheria rate was reduced from 13 to 0.53 per cent. among nurses in the Durand Hospital. The value of 0.11+ mixture is equal to the 3L.+ mixture without the severe reactions. Children do not react as severely as adults. In 156 cases given a Schick test, Crooks reports 144 immune, Schick negative, three to six months later. Four and one-half months is the average time required to develop immunity from the first course of three injections. Diphtheria toxin-antitoxin mixture as usually supplied contains 0.11+ L. dose of diphtheria toxin neutralized with the required amount of diphtheria antitoxin in packages of 3 ampules each containing 1 c.c.

Park asserts that diphtheria toxoid which contains only a minute amount of antitoxin is nearly equal in immunizing power to toxin-antitoxin with the danger of anaphylaxis largely eliminated. The dose should not be larger than 0.5 c.c. because of pseudoreaction. Immunization may be produced by 1000 units of antitoxin, but this is not reliable for more than thirty days.

**Symptoms.**—Many of the onsetting signs of diphtheria vary according to the grade from mild to moderate, severe or septic, with mixed infections. We usually find myalgia, headache, nausea, sore throat, and temperature between 99° and 101° F. or higher. These complaints may be augmented by

chill, weakness, swelling of the cervical and submaxillary glands. Odynphagia may be slight to severe depending on the local inflammation. Pain in the lumbar region may be more severe than that in the fauces. The exudate or false membrane may occur on the tonsils at the site of bacterial choice. It may remain here or extend to the pillars, soft palate, or pharynx, and sometimes to the nose. The earliest appearance may be a film increasing hourly to a yellowish-white patch turning to gray or grayish brown. It grows heavier, firmer, and more like leather with an attachment to the membrane below that produces bleeding and a raw surface if the pseudomembrane is pulled away. This re-forms quickly over the denuded surface. With septic absorption the pulse may become more frequent and small. The expression takes on a more anxious picture with a moist or clammy skin, open mouth, cyanosis of fingers and lips. The septic picture of lethargy with an indisposition to talk or move may supervene. The heart should be carefully observed as the pulse may become feeble, intermittent, or slow as a toxic effect is produced on the pneumogastric nerve and the cardiac muscle, with vomiting and heart failure. Hibbard claims that when the pulse-rate remained above 150 the mortality was 50 per cent. When the nose or nasopharynx is invaded the symptoms are manifested by nasal stenosis, and efforts are made to clear the passage of the ichorous discharge, by blowing, snuffing, or hawking. The kidney involvement is marked by albuminuria or casts. The ear and systemic extensions, with greater cervical gland enlargement, take their origin from the nose or particularly the nasopharynx.

Primary nasal diphtheria is rare, but even the unilateral variety may develop as the writer can testify from personal experience as a sufferer for ten to fourteen days. Rhinorrhea may be a prodromal symptom, or a nasal hemorrhage may appear at the onset. Paralysis may occur from the seventh day to six weeks or later, although the third week is the more frequent time of appearance—usually motor it may be sensory also. One of the first symptoms noted may be that fluids come through the nose. The soft palate is the first region affected. The lesions of the nervous system were recognized by the older writers, although in 1863 they were particularly observed by Charcot, Vulpian, and Oertel. In 1876 Rieman showed lesions of the medulla oblongata following diphtheritic meningitis. The pharyngeal and laryngeal muscles may be attacked in order. The upper and then the lower extremities may follow. A case under observation in the Detroit, Eye, Ear, Nose, and Throat Hospital, showed all the voluntary muscles affected. This patient was fed by the stomach-tube until recovery. The ciliary muscle may be involved. Entire recovery is the rule under the hypodermic use of arsenic and strychnine, and the galvanic current.

**Diagnosis.**—Diphtheria must be differentiated particularly from follicular tonsillitis, Vincent's angina, quinsy, pharyngeal mycosis, syphilitic ulcer, cauterizations, pneumo- and streptococcic infections such as scarlatina anginosa. A thorough clinical examination of the nose and throat and nasopharynx, in the daylight when possible, offers the best means of determination. This should be confirmed by a careful bacteriological examination of material taken direct from the throat and stained, or when in doubt preferably by culture.

Follicular tonsillitis resembles diphtheria more nearly than other infec-

tions. It may be quite difficult to differentiate at the onset. The exudate, however, in tonsillitis is found at the mouth of the follicles or crypts. The material is soft, removable with a swab or soda solution, and of a white or whitish-gray color. The exudate may be more tenacious, however, and may exhibit even a fibrous character. In the presence of a diphtheria epidemic the appearance may be confusing.

The difference may be summarized as follows:

*Follicular tonsillitis.*

Fever 100° to 105° F.  
Onset sudden.  
No Klebs-Löffler present.  
Membrane easily removed.  
Tonsils inflamed and swollen.  
Exudate over crypts, and not usually spreading over pillars.  
Glands equally enlarge.  
Constitutional disturbances more marked early.  
Vomiting rare.  
Albumin rare.  
No paralysis.  
No croupy cough.

*Diphtheria.*

Fever 99° to 101° F.  
Onset more gradual.  
Klebs-Löffler present.  
Membrane adherent.  
Tonsils not acutely swollen.  
Exudate extending to uvula and anterior pillars to pharynx and palate.  
Glands enlarged more on one side.  
General symptoms, slighter in mild case.  
Vomiting more common.  
Albumin more frequent.  
Paralysis indicates diphtheria.  
Croup more frequent.

The differential diagnosis in pharyngeal mycoses may be made from the following facts: No constitutional phenomena of any importance attend the condition. The inflammation of the mucous membrane of the fauces and particularly the tonsils is little or nothing in comparison to that of acute tonsillitis or diphtheria. An examination of the throat shows that the white patches of mycosis are not exudate or pseudomembrane, but adhering fungus growth as determined by the microscope. Secondary syphilis may be mistaken for diphtheria while the condylomata are present in the pharynx. To differentiate by culture the technic must be properly carried out. As McCollom says: "A great source of error is the fact that when taking the culture the swab or the platinum needle is rubbed over the surface of the membrane in the very position where the organisms are most likely to die. The edge and if possible the under surface of the diphtheritic membrane are the proper places from which to take the cultures. A second culture may be taken with advantage from the secretion of the mouth or nose. No antiseptic gargle should be used a short time previously. A culture placed in an incubator will be ready eighteen to twenty-four hours later. The diagnostician must not permit himself to be misled because of negative findings. The clinical examination is of the greatest value, and promptness in the use of antitoxin saves lives. The antidiphtheritic serum should be administered when the clinical findings are positive. Puzzling border-line cases with negative bacterial findings occur even in epidemics."

**Mixed Infection.**—Atypical varieties of diphtheria develop in the presence of mixed infection particularly with the various forms of streptococcus, staphylococcus, and pneumococcus. These cases may exhibit clinical features of irregularity in behavior under treatment, in temperature range, in the course and complications of the disease. The puzzling cases without Klebs-Löffler findings and with a positive clinical picture may lead to a fatal termination unless antitoxin is used early. Too much importance must not be placed on the bacteriological examination which may fail to be positive on account of faulty technic. True diphtheria as a typical

clinical exhibit may be greatly modified by the predomination of micro-organisms of the streptococcus family. In pseudomembranous laryngitis 85 to 95 per cent. is due to Klebs-Löffler infection, and must invariably be treated as such. Error in this regard is dangerous and leads to unnecessary mortality. Following its invasion, no doubt each pathogenic micro-organism has a definite train of clinical phenomena characteristic of it and comparative ease of differentiation by clinical instead of microscopical and cultural methods. It is seldom the case that a pure culture of any one organism exists.

In every mouth we find pathogenic germs that remain impotent except when favorable conditions are presented for their growth. When resistance is lowered to any one of these strains they may become the exciting cause of the inflammation at the proper stage, and dependent on the virulence and variety of the germ these accessories may take on their special phase in modifying or increasing the variety and character of the progress of the disease. It is, therefore, apparent that the influences of mixed infection is hourly or daily in close relation with the resistance of the individual, the virulence of the germ, and the variety and attenuation of the micro-organism. The key to the condition must ever remain in the antibacterial power of the individual. This axiom in the treatment is paramount. It must never be forgotten. Any method that will raise the resistance early is of great value. Treatment with this in mind must be given promptly and empirically until we can attain a scientific measure of antitoxic power. In diphtheria we have the Schick test and toxin-antitoxin. They must be used extensively.

The varying malignancy of mixed infection may be illustrated by the following case: In a child the infection was diagnosed as Klebs-Löffler, and antitoxin given followed by recovery. Three weeks later a typical streptococcus angina developed with cultural findings positive for diphtheria. Antitoxin was again administered, although the patient was probably immunized. The course was that of the mixed infection, with the Klebs-Löffler germs apparently without influence in the second attack.

The staphylococcus may cause simple spasmodic laryngitis. The streptococcus stenosis may be of longer duration requiring re-intubation. In the Klebs-Löffler laryngitis the antitoxin is a specific with a positive action. The ratio of mortality is changed in intubation cases from 80 to 90 per cent. to 5 to 20 per cent. The staphylococcus is usually found in the fauces and especially in mild tonsillitis. They may be obtained from the crypts. A severe form may develop, however, with high temperature and constitutional symptoms.

Mixed infection with the influenza bacilli as reported by Wyncoop may show marked redness, swelling of the tonsils, with a well-developed membrane, yet the culture may show an approximately pure finding of the so-called influenza bacillus.

**Prognosis.**—This may vary greatly with the epidemic and its virulence. It is influenced by the age of the patient. The mortality is greatest under five years and diminishes rapidly after puberty. The toxic type with cardiac degeneration is often suddenly fatal. The complications vary with the extent and location of the exudate. The hemorrhagic form with nasal or throat bleeding may be quickly fatal. The stage at which a diagnosis is made and treatment begun greatly influences the mortality. The value of antitoxin after the third day diminishes rapidly, even in mild cases as

cardiac paralysis may supervene. Enlarged tonsils and adenoids increase the mortality. Cottier found 50 per cent. hypertrophied tonsils and adenoids in 38 postmortems. A grave prognosis is indicated in cases with anuria, toxic slow pulse, bronchopneumonia, hemorrhagic collapse, vomiting, and extensive and extending pseudomembrane. Secondary laryngeal involvement with membrane occluding the tube and toxemia are highly fatal, although paralysis from membrane in the larynx alone is quite different.

Without antitoxin, in Boston, statistics in 24,813 cases showed a death-rate varying from 30.7 to 35.7 per cent. Epidemics showed as high as 50 per cent. mortality. Ecchymoses and sepsis with or without operation are usually forerunners of death. High dosage of antitoxin (40,000 to 100,000) may save some apparently fatal cases. Its general use has reduced the mortality in large cities to 8 to 15 per cent.

**Treatment** may be prophylactic, local, constitutional, and operative. The prevention of diphtheria is possible and practical by efficient scientific methods. They embrace immunization, quarantine, and sanitary measures. The Schick test, toxin-antitoxin, and antidiphtheritic serum represent the armamentarium. They may be used extensively by Boards of Health, in schools, and by the family physician. The problem of immunization is solved most completely in this disease if the technic of prevention receives widespread application. All children should be tested and given a card of the condition of their immunity. Epidemics may be stopped, and even a second case in a family, controlled by efficiency in these methods. In the presence of exposure a 2000-unit dose of antitoxin gives immunity for thirty days in almost every case.

As the disease is contagious it follows obviously that the more perfect the quarantine the less chance of spreading the disease. The laity have learned to dread diphtheria and they will co-operate with the Board of Health in most cases. The management of the quarantine is important and based on scientific facts. We know that sunlight and free ventilation are of the greatest value. The carrying of the disease by clothing, furniture, food, playthings, books, pencils, and animals is usually appreciated. All discharges should be disinfected by burning or corrosive sublimate (1 : 500). The mild or walking cases with nasal discharge are a frequent source of danger. School inspection has done much in early diagnosis and prophylaxis. Gauze should be used in the place of handkerchiefs and destroyed. The visiting physician and nurse should wear a cotton gown and proper head protection for the hair. The hands should be frequently washed with mercurial soap or bichloride of mercury solution.

The after-care of the room is of great prophylactic importance. Steam sterilization is best for clothes, mattresses, and other bedding; 1 : 500 bichloride should be used on the woodwork and floors. Sunlight and painting are efficient wherever possible. Although practically a winter disease, fly and insect infection must be avoided. Sterilization above 212° F. for an hour will destroy all bacteria. Local prophylaxis is of little value except as a measure of cleanliness. Many practitioners have faith in applications, gargles, or sprays of silver, alcohol, carbolic acid, hydrogen peroxide, boric acid, and the like. It must be remembered, however, that antitoxin is the only genuine prophylactic to be depended upon.

*Local Treatment.*—The therapeutic idea of washing and disinfecting the

area invaded by the pseudomembrane is ancient and logical only within proper limitations. Common sense and judgment require that the use



Fig. 199.—Position of child, operator, and assistants for intubation. (From Shurly, *Diseases of the Nose and Throat*, D. Appleton & Co., publishers.)

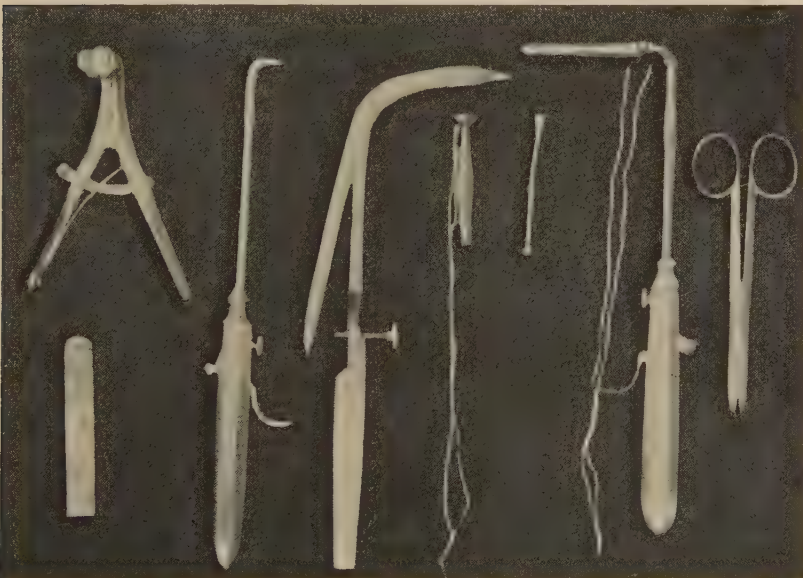


Fig. 200.—Showing the scale, the gag, the obturator, and the introducer with a tube attached (McCollom).

of local measures shall not be over strenuous and sap the vitality of the patient by too frequently waking with disturbance of rest. Astringents and escharotics may be harmful as well as useless. There is practical as well as theoretical value in some of the long list of local disinfectants. In mild cases an alkaline lotion of salt and soda such as compound antiseptic powder may be sufficient. Seiler's or Dobell's solution furnish a cleansing wash. In children solutions may be used with a large soft-rubber ear syringe, flushing the throat and gently irrigating the nose. A stronger preparation may be made with hydrogen peroxide 1 to 4 or 7 in lime-water. Following these irrigations in the presence of thick membrane, Löffler's solution may

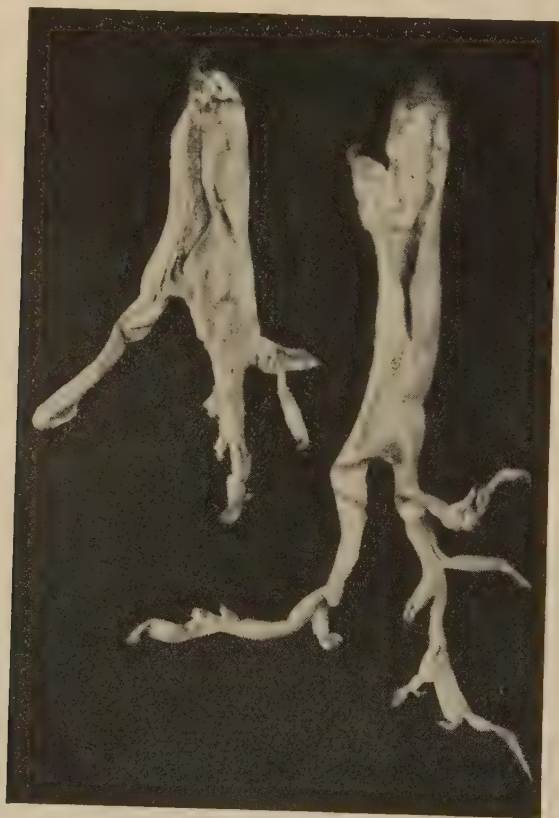


Fig. 201.—Casts of the trachea and bronchi (McCollom).

be applied in very small quantity for ten seconds to the fauces and tonsils. Many hundred formulæ have been recommended for local treatment, but most of them are unnecessary or inefficient with a sufficient dosage of antitoxin.

In septic throats Sir St. Clair Thomson suggests as the best local treatment an acid solution of chlorate of potash containing free chlorine. It is prepared by 5 minims of strong hydrochloric acid on 9 grains of powdered chlorate of potash and shaking up with an ounce of water gradually added. This is mixed with an equal quantity of hot water and used for syringing the throat every two hours. In severe cases thorough local treatment is

necessary and should be carried on night and day at regular intervals. The danger of infecting the sinuses and eustachian tubes is spoken of as a deterrent and yet this occurs in point of fact only occasionally and this drainage of the nose and the reduction of toxemia are so important that these features of the case must be considered.

The constitutional or general treatment means antitoxin 5000 units at once, and more repeated every twelve to twenty-four hours as indicated. The patient is given absolute bed rest. A full dose of calomel followed by a saline is recommended for its action on the bowels and the additional value of mercury on the lymphatic and glandular apparatus.



Fig. 202.—Insertion of finger to locate epiglottis and arytenoids. (From Shurly, *Diseases of the Nose and Throat*, D. Appleton & Co., publishers.)

In septic cases or those with any question of cardiac failure, alcohol, digitalis, and strychnine are of the greatest value. A young child may take a surprisingly large dose of whisky or brandy. One to 2 drams every four hours may be given to a child one to two years with a careful observation of the effect on the pulse. The increase may be made gradually. The heart must be watched with great care for indications for the use of strychnine and digitalis. The former is of value through the period of convalescence and earlier as a prophylactic aid in threatened paralysis.

Antitoxin is a specific in this disease that needs no argument in support of its use. It rests on its merits, it requires only the watchful eye of the family physician to give it early, often, and in large doses, to all cases that offer any question as to the differential diagnosis as well as those with Klebs-Löffler findings. Many lives are lost by procrastination or waiting for a culture. A fatal issue may be avoided by giving 5000 to 2000 units in the first twenty-four hours of invasion.

In health there is practically no physiological action except in a few cases susceptible to horse serum. It cannot be held responsible for com-

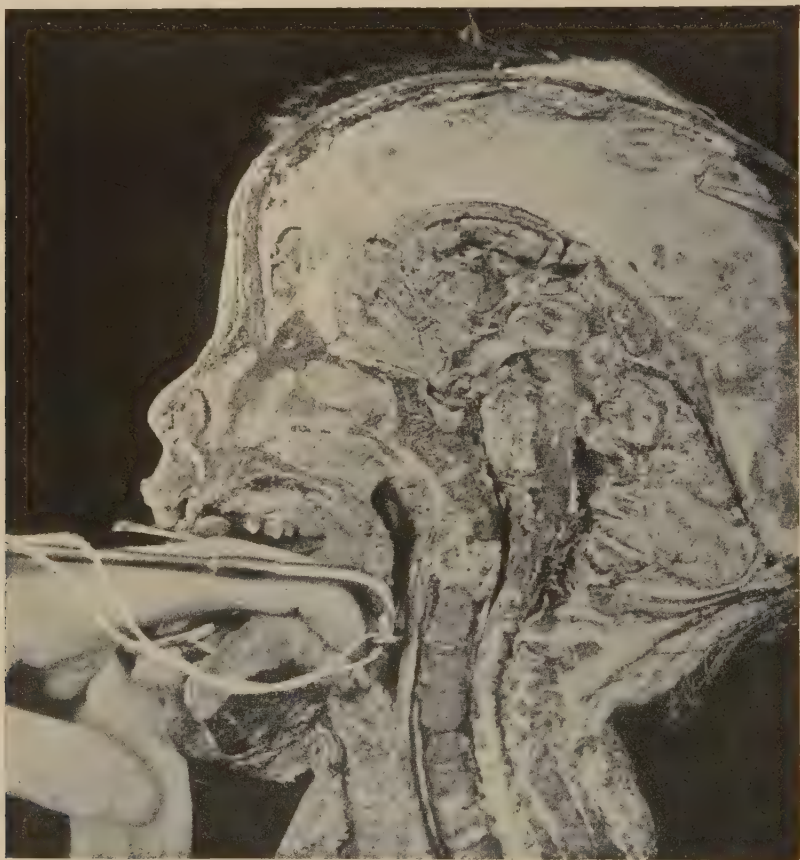


Fig. 203.—Tube entering larynx. (From Shurly, *Diseases of the Nose and Throat*, D. Appleton & Co., publishers.)

plications such as albuminuria, nephritis, or cardiac pathology according to the best accumulated clinical evidence. It is practically harmless. It is true that eruptions such as urticaria erythema, simplex multiforma, and arthritic edema may follow its use particularly about the ninth day after the initial dose. The effect on the exudate in from six to twenty-four hours may be startling and definite. A red line of demarcation forms around the exudate followed by a clearer line between the mucous membrane and the false membrane. The exudate loosens at its edges and its folds. The separation extends over the entire exudate. The patch

becomes soft and exfoliates with coughing and irrigation. The mucous membrane remains red and granular. The pseudomembrane may recur over the area as a finer and softer film. The action may be more prompt in laryngeal cases and an entire cast of the tracheal picture and bronchial tree may be exfoliated. The general effect of antitoxin often produces a reactionary rise of temperature. It may even be quite a marked hyperpyrexia. This is usually of short duration (twenty-four to thirty-six hours). As the body temperature returns a feeling of well-being supplants that of malaise with headache and backache. These phenomena sometimes occur in milder cases with the early administration of the serum.

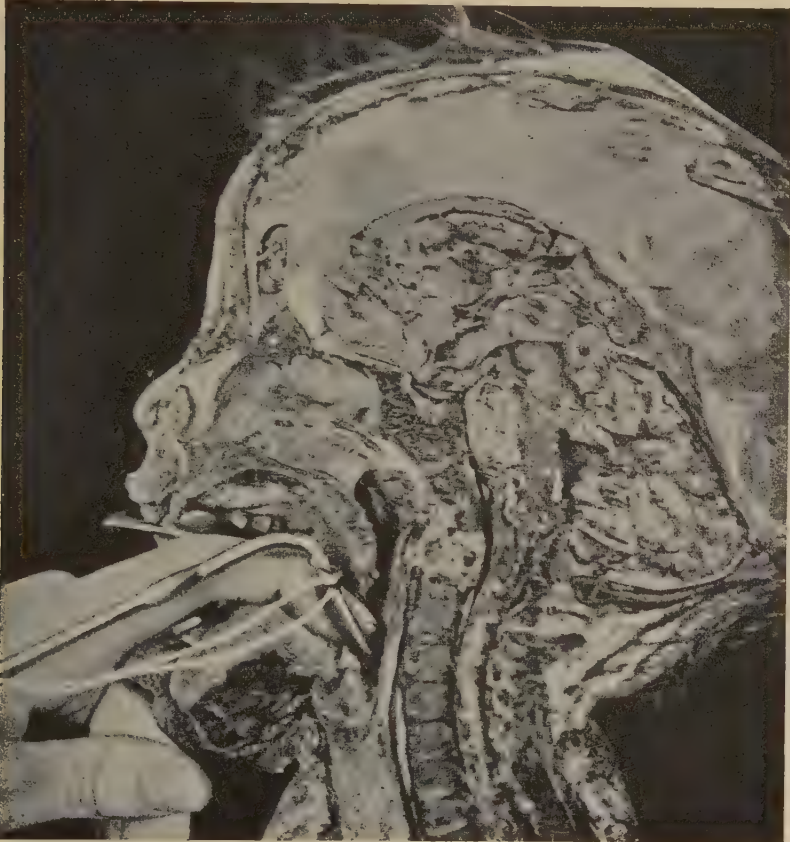


Fig. 204.—Illustrating the error of depressing the handle of the introducer, resulting in the tube entering the esophagus instead of the larynx. (From Shurly, *Diseases of the Nose and Throat*, D. Appleton & Co., publishers.)

The dosage must vary with the judgment of the physician and with the age of the patient and severity of the symptoms. Procrastination and misleading early diagnosis must be avoided. The loss of even a few hours in severe or laryngeal cases may be fatal. Five thousand to 40,000 units may be used during the twelve to twenty-four-hour intervals. Given within twenty-four hours the death-rate should not exceed 5 per cent. This mortality increases rapidly until it reaches 12 to 20 per cent. if not given before the fifth day. In severe cases the number of injections may vary from three

to five. There are no contraindications to the use of antitoxin, except in subjects sensitive to horse-serum for which an injection of adrenaline is the logical antidote.

Laryngeal diphtheria may require intubation or tracheotomy. The operation of choice is usually intubation.

**Intubation.**—*History.*—At the beginning of the Christian era Hippocrates mentions the passing of sounds into the larynx and trachea for the relief of difficult respiration. Bouchut in 1858 produced silver tubes almost cylindrical that were carried into the larynx by a sound and held by a silk thread for extraction. Modifications of tubage or catheterization of the

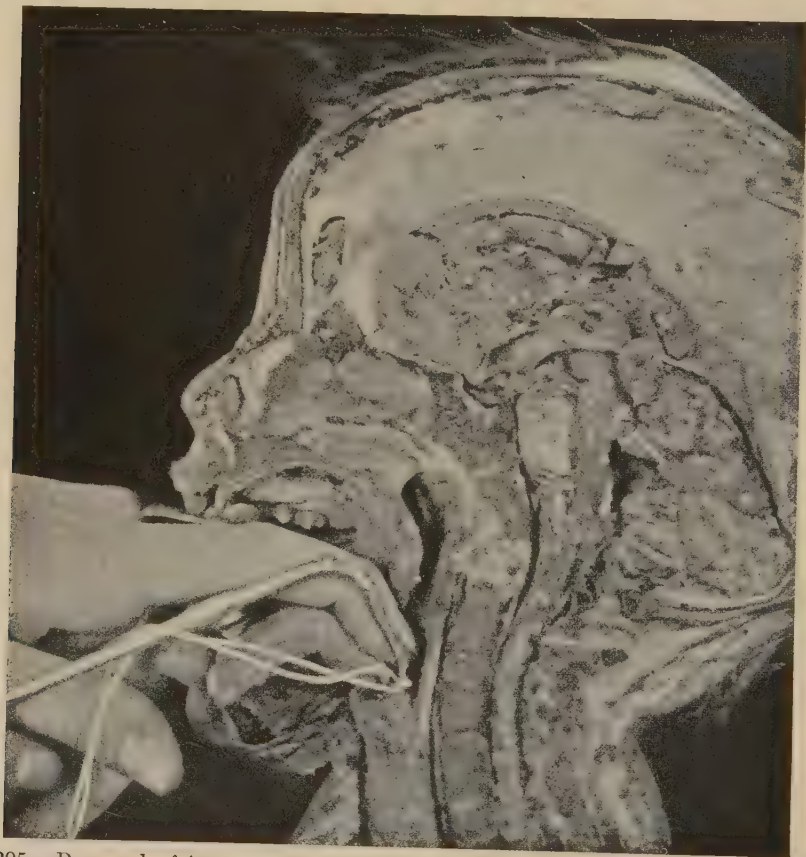


Fig. 205.—Removal of introducer, with the finger holding tube in the larynx. (From Shurly, *Diseases of the Nose and Throat*, D. Appleton & Co., publishers.)

larynx were reported by Chiari, Brown-Séquard, Bergman, and others. In 1885 Frank Waxham intubated a sixteen-month child. The discovery of the laryngoscope added impetus to tubage for various forms of stenosis.

The great work of O'Dwyer of New York stands out pre-eminently as the scientific contribution that perfected the technic and instruments for successful results. His tubes and method of introduction and extraction offered a safe and quick method which has not been surpassed up to the present time. Mosher has devised instruments for direct intubation under illumination that are useful in selected cases. The indications should

be considered early and the operation not used as a last resort. When difficult breathing begins with retraction of the chest muscles, restlessness, sweats, and slight cyanosis, intubation should not be delayed. Dangerous obstruction may occur without the complete loss of voice. Intubation has the advantages of requiring no anesthetic, leaving no scar, maintaining respiration through the usual air-tract, rapidity of relief, ease of application in the hands of experts, with co-operation of the relatives, less danger of pneumonia, and less after-care required, and for a shorter period. It has a great advantage over tracheotomy in the fact



Fig. 206.—Tube in the larynx, with the string attached. (From Shurly, *Diseases of the Nose and Throat*, D. Appleton & Co., publishers.)

that when skilfully performed the resulting mortality is less. Secondary tracheotomy can be performed if necessary in rare cases.

*Operation.*—The child may be placed in a sitting posture in the lap of an assistant or in a horizontal position on a table with the head slightly raised. The arms and elbows especially are tightly pinioned to the body by means of a sheet thoroughly wrapped around the thorax. The instruments—consisting of a suitable threaded tube, introducer attached; a mouth-gag; an extractor; and tongue-depressor—may be sterilized and placed on a chair or table in immediate reach. If the sitting position is the choice,

the operator sits in front of the patient, introduces the mouth-gag well back on the molar teeth, while a second assistant holds the head and gag firmly. The position of the head should be erect as if the patient were hanging by the head. The extended forefinger of the left hand, palmar side down, is carried through the mouth to the base of the tongue and the epiglottis picked up and gently moved forward. The right hand conducts the introducer and tube horizontally along the tongue in the median line. The handle of the introducer should be started well down on the chest of the patient. When the lower end of the tube comes in contact with the

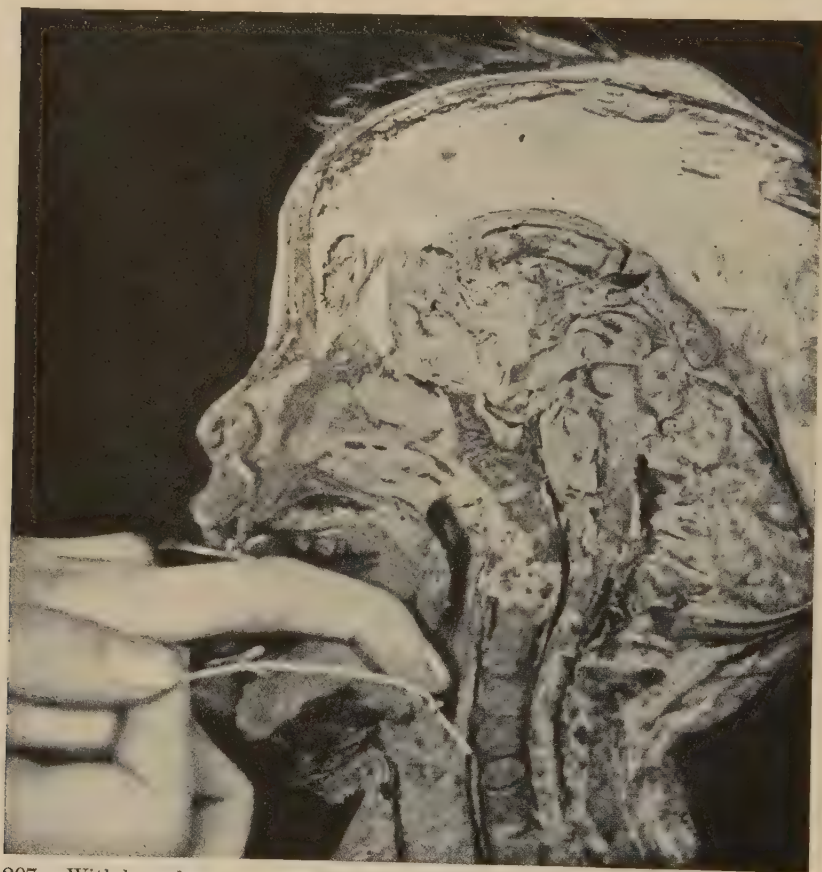


Fig. 207.—Withdrawal of the string, with the finger on the tube to hold it in position.  
(From Shurly, *Diseases of the Nose and Throat*, D. Appleton & Co., publishers.)

left index-finger at the entrance to the larynx the handle of the introducer should be given an abrupt turn to bring the tube into a vertical or beyond a vertical position to meet the slightly forward angle of the larynx and not slip into the esophagus. The critical point of junction is here. The tube must be kept in the median line. The left index-finger must be a guide and the angle forward must be quickly and accurately executed to meet the swollen and membranous entrance to the larynx. When the tube reaches the larynx the applicator is hurriedly removed, the left index-finger then gently guides the tube deeper into place. The loop of string or

silk thread about 2 feet long which passes through the eyelet of the tube will remain hanging out of the mouth. It may be passed over the ear and covered with adhesive tape and left until proper observations of the effect are made.

A successful intubation will usually be followed by a deeper respiration and a cough with the characteristic tubular metallic sound of the air rushing through. If the tube is in the esophagus no cough will be heard and the breathing will be worse or not relieved. The tube may be vomited with

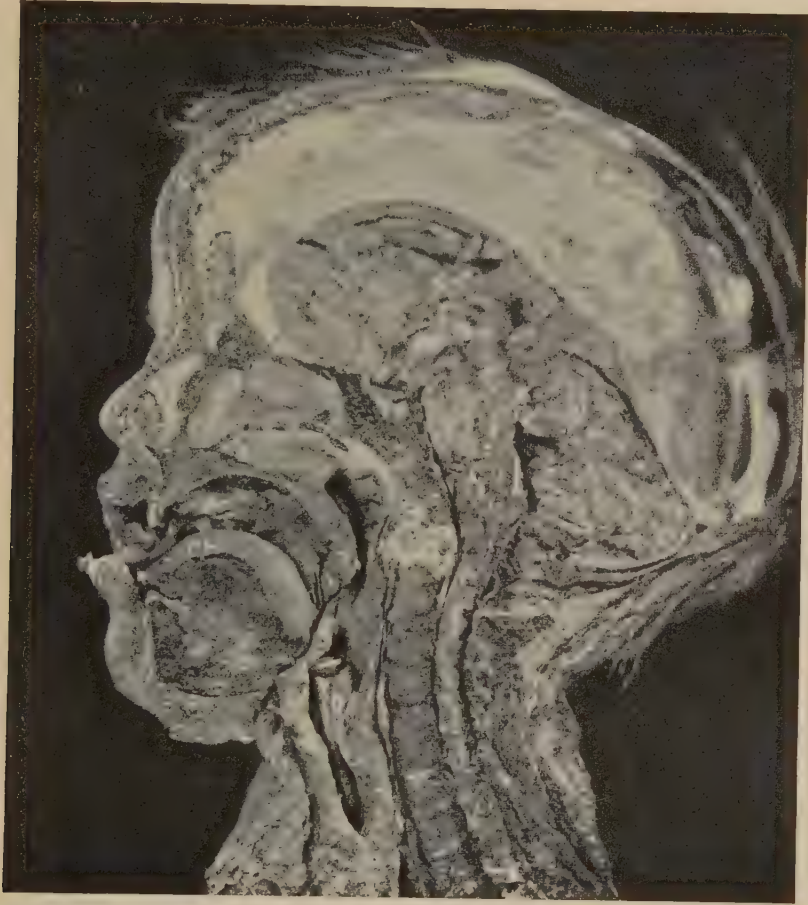


Fig. 208.—Tube *in situ*. Showing the head of the tube upon the vocal cords, and the position of its distal end in the trachea. (From Shurly, Diseases of the Nose and Throat, D. Appleton & Co., publishers.)

gagging. If membrane has been pushed down ahead of the tube it must be quickly removed and an effort made to cough out the loosened membrane. The tube is then reinserted. With respiration quiet the thread is removed by cutting and placing the left index-finger on the head of the tube.

Intubation should be done without force as trauma is a frequent cause of retained or prolonged use of tubes. Leaving the thread increases the danger of coughing out the tube. In removing the tube the left index-finger becomes the guide. The first steps are similar, the preparation, position,

and assistants are the same. The extractor is passed deeply into the lumen of the tube, the jaws are spread and the tube is gently pulled upward. It is wise to prepare a tube for introduction at this time as asphyxia may follow collapse of the larynx. The tube is removed on the third or fourth day. Slowly increasing edema in two or three hours may supervene, and the tube must be reinserted. In my experience with more than 500 cases a few required reintubation several times during a period covering seventeen days as a maximum.

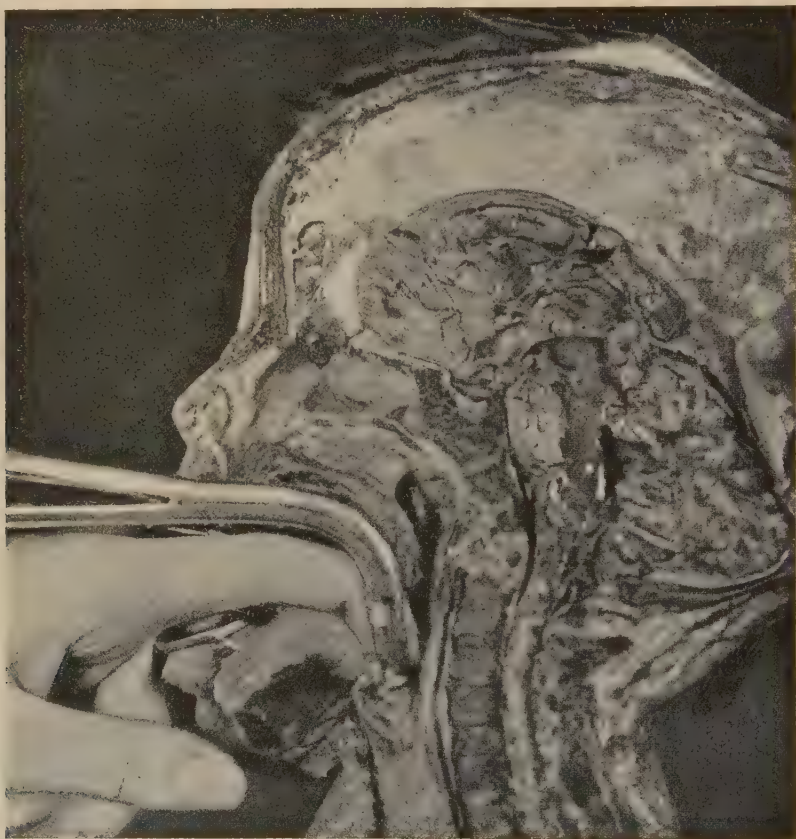


Fig. 209.—Method of removing the tube. The beak of the extractor, guided by the finger, is just entering the tube. (From Shurly, *Diseases of the Nose and Throat*, D. Appleton & Co., publishers.)

The principal difficulties in practising intubation may be met by learning the operation on the manikin, dog, or cadaver. The limited space afforded in the pharynx of the young child necessary in timing and making the abrupt turn seems impossible for some to learn. The art of selecting a tube to fit is not all in the measuring scale. Individual differences of the child, small for its age, must be met by smaller sized tube. A tube too large passes into the esophagus or pyriform sinus more easily. A tube too small will be coughed out readily especially as the edema subsides, and the next size may have to be inserted, or a larger retaining swell may be indicated. A tube swallowed usually passes the alimentary canal without difficulty.

Retained or prolonged tubes require special study as to the cause. The writer has removed a number by coating the tube at the last introduction with vaseline saturated with alum.

These cases of retention are the result of traumatism in performing the operation or choosing an unsuitable tube which by too great pressure produces ulceration, which is frequently found where the end of the tube rests and the direction of pressure may be changed or relieved entirely by frequently changing the tube or by using one with larger retaining swell or one that has been shortened to remove the pressure below. These modifications have been suggested in splendid articles by O'Dwyer and Lynah.



Fig. 210.—Feeding intubated child with nursing bottle; the head being lower than the shoulder (the Casselberry position). (From Shurly, *Diseases of the Nose and Throat*, D. Appleton & Co., publishers.)

*Feeding.*—The problem of feeding after intubation must be given careful attention. It may be met in four ways. First—liquid food may be swallowed with difficulty, while semisolids are taken readily. The writer remembers a patient two years of age who sat at the table eating the usual food without difficulty. Some may refuse food at first from discomfort only. The mother or nurse should be instructed how to feed the patient by an actual demonstration before leaving the house or hospital. Second—the Casselberry position (Fig. 210): The patient is placed on the back with head inclined, lowering the head at an angle of 45 degrees or more

may, be very satisfactory. The feeding is given slowly with a spoon or nursing bottle and the liquid follows the roof of the mouth. Third—the patient may adopt the prone position with the head over the bed facing a glass of liquid nourishment placed on the floor into which a catheter is inserted. In this manner the patient feeds slowly by suction. Fourth—a nasal feeding is given through a small rubber catheter which is inserted into a larger rubber tube and in turn attached to a glass funnel. The lubricated



Fig. 211.—Feeding intubated child by suction of fluids through rubber catheter. (From Shurly, *Diseases of the Nose and Throat*, D. Appleton & Co., publishers.)

catheter is passed slowly and gently through the nose and into the esophagus. A few ounces of water is passed followed by milk, drugs, and stimulants as indicated, and followed by water. The child readily becomes accustomed to this procedure after a few trials. This method is of value in postdiphtheritic paralysis.

BURT R. SHURLY.

## PART III—EAR

### ANATOMY OF THE EAR

**Introduction.**—Before one can understand the diseases of the ear, it is important to know something of the essentials in the anatomy of this part. Only a brief presentation of these structures is permissible here. The organ of hearing comprises a *central* part, that within the cranial cavity, made up of the central pathways of the eighth nerve and the cortical area in the second temporal convolution, and a *peripheral* part located within the temporal bone. It is with the latter mechanism alone that we are chiefly interested. This peripheral mechanism is subdivided into an outer, a middle, and an internal ear.

#### THE OUTER EAR

The outer ear consists of the auricle and the external meatus. The auricle has a supporting framework of flexible cartilage covered with integument. On the anterior surface this covering of skin is closely adherent to the perichondrium. On the posterior surface this attachment is much looser. This anatomical relation accounts for the frequent occurrence of hematomas from traumatism over the anterior surface of the auricle. The pendulous lower part of the auricle known as the lobule is made up of connective tissue and fat with its covering of skin. The outer rim of the auricle is known as the helix. Parallel with this outer rim is an elevation called the antihelix. In front of the meatus is a cartilaginous prominence, the tragus, and directly back of this is a similar prominence, the antitragus, the two being separated by the incisura intertragica. The flaring, trumpet-like opening of the meatus itself is called the *cavum conchæ*, and the depression above this is known as the *cymba conchæ*.

The **meatus auditorius externus**, the external meatus, has an outer cartilaginous and an inner osseous portion. In the newborn the osseous portion is entirely wanting, and it is not until the head is full grown that the osseous meatus is fully formed. The direction of the canal in the cartilaginous portion is upward, inward, and forward, while that of the osseous portion is downward, inward, and backward. In order, therefore, to gain a satisfactory inspection of the fundus of the meatus in the adult, it is necessary to straighten out this angle by pulling the auricle upward and backward.

The **cartilaginous meatus** is made up from a tongue-like prolongation of the cartilage forming the auricle. From this prolongation of cartilage is formed the lower and anterior meatus wall which in the cartilaginous canal is considerably longer than the upper posterior meatus wall. In order to give mobility to the auricle, this cartilaginous tongue is split by two horizontal breaks called the *incisuræ santorini*. These breaks have the clinical importance that through them an abscess of the parotid occasionally discharges into the external meatus and a deep-seated furuncle of the meatus may discharge into the parotid region. The skin covering the cartilaginous meatus is beset with a growth of stiff hairs and carries the serum in secreting glands.

In the newborn the membranous cartilaginous meatus is attached to the os tympanum, a narrow rim of bone to which the drum membrane is attached. There is no osseous meatus. As the head expands the bony meatus is formed in a large part by accretions to this os tympanum. In this manner is formed the whole of the anterior, the lower and part of the posterior meatus wall. The upper wall is formed from the squamous bone and the upper part of the posterior wall from the petrous bone, as it expands into formation of the mastoid process. It is only this upper posterior portion of the meatus that is encroached on by the mastoid cells. Along this wall just external to the attachment of the drum membrane lies the tympanic antrum. These relations of the pneumatic spaces of the middle ear to the external meatus wall are of clinical importance. In the first place cholesteatoma of the antrum often breaks through this wall into the external meatus. In the same way an infiltration of the periosteum along this segment of the external meatus may result from mastoiditis.

At the upper margin of the opening of the osseous meatus is a conspicuous landmark, the *spina suprameatum*, to which the superior ligament of the auricle is attached. The outer part of the osseous meatus is oval in outline with a long diameter perpendicular. The membrana tympani which closes the fundus of the meatus is placed at an angle so that the membrane lies as much horizontal as perpendicular; the lower anterior segment lies deeper than does the upper posterior segment. The floor of the osseous meatus is convex, and at the junction of the middle and inner thirds there is a conspicuous narrowing. The part lying internal to this constriction is known as the sulcus of the external meatus. Foreign bodies entering the external meatus usually lodge at this *isthmus* of the meatus until forced through, when they sink into the sulcus from which they are removed with difficulty.

#### THE MIDDLE EAR

The middle ear is made up of three parts, the *tuba auditiva* (t. eustachii), the *cavum tympani*, and the *cellulæ mastoideæ*. The **eustachian tube** begins in the lateral wall of the nasopharynx, takes a course upward, backward, and outward, and opens in the anterior wall of the tympanic cavity. The inner two-thirds is cartilaginous and the outer third is bony. At the junction of the two is a constriction known as the *isthmus*. The pharyngeal orifice is a wide flaring opening with a prominent upper posterior lip known as the *torus tubarius*. The tympanic orifice of the tube is also flaring. The only narrow part of the eustachian tube is at the isthmus. From the osseous portion pneumatic spaces occasionally develop, forming the *cellulæ tubarium*, a condition which in middle-ear infection may account for persistent discharge from the tube. The internal carotid lies just internal to the tympanic orifice, separated by a very thin bony plate. The apex of the cochlea lies at this point.

The **cavum tympani**, commonly known as the drum, lies just internal to the membrana tympani. The cavity of the tympanum consists of four parts, the *cavum tympani* proper, a *recessus hypotympanicus*, a *recessus epitympanicus*, and the *antrum tympanicum*. The tympanic cavity contains a chain of three ossicles which join the drum membrane with the internal ear. The first of this chain, the *manubrium*, is attached to the membrana tympani. It articulates with the second ossicle called the *incus*, which in turn articulates with the stapes, the foot-plate of which

fits into the fenestra vestibuli. The articulations between the ossicles are such that they constitute a series of levers by which the movements of the drum membrane are transmitted to the foot-plate of the stapes. The resulting excursions of the stapes are less extensive and, therefore, more forcible than are the movements imparted to the handle of the hammer as it lies embedded in the drum membrane. Two small muscles regulate the tension between the ossicles, the tensor tympani, pulling the handle of the malleus inward and thus forcing the foot-plate of the stapes into the oval window, and the stapedius muscle which tilts the stapes out of the oval window.

In addition to the chain of ossicles the cavum tympani contains various folds of mucous membrane extending from the inner wall to these ossicles. These folds often form the basis for the formation of stiffened adhesive

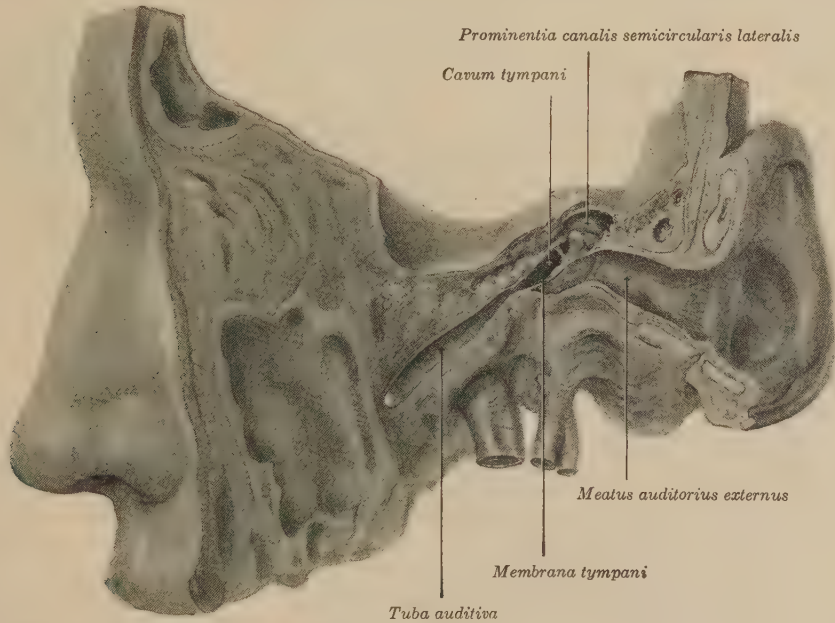


Fig. 212.—Section showing relation of the tuba auditiva (eustachii) to cavum tympani and the meatus auditorius externus.

bands, the result of inflammatory processes in the tympanum. In this way they may constitute the basis for serious impairment of hearing. These folds of mucous membrane are found most commonly passing from the body of the incus and the long process to the inner wall of the tympanum. The folds are especially common also in the niche of the fenestra vestibuli, passing from the cruræ of the stapes to the walls of this depression. Occasionally similar folds of membrane are found passing from the incus to the walls of the attic and spreading fan-like over into the antrum.

The *outer wall* of the tympanum is formed in part by the membrana tympani and in part by the segment of the squamous bone which goes to make up the upper wall of the external meatus.

The membrana tympani is more important because of the information to be derived from its examination in diseases of the ear than for its importance as a physiological structure in sound perception. It forms an incom-

plete oval with a longer diameter perpendicular. In its upper margin is a break called the *incisura tympani* (Rivini's segment) which represents the cleft not closed by the *os tympanum*. The entire drum membrane with the exception of the part at the upper pole attached to the margin of the *incisura tympani* is known as the *pars tensa* because in addition to the skin forming the outer covering and the mucous membrane forming the inner covering, there is placed between these two layers a firm middle layer composed of radiating and circular fibers. The segment of the upper pole is known as the *pars flaccida* because it lacks the firm fibrous middle layer. The *upper wall* of the tympanum is a thin plate from the squamous bone in which dehiscence often occurs. This plate of bone is called the *tegmen* and continues posterior as the roof of the antrum. Passing diagonally

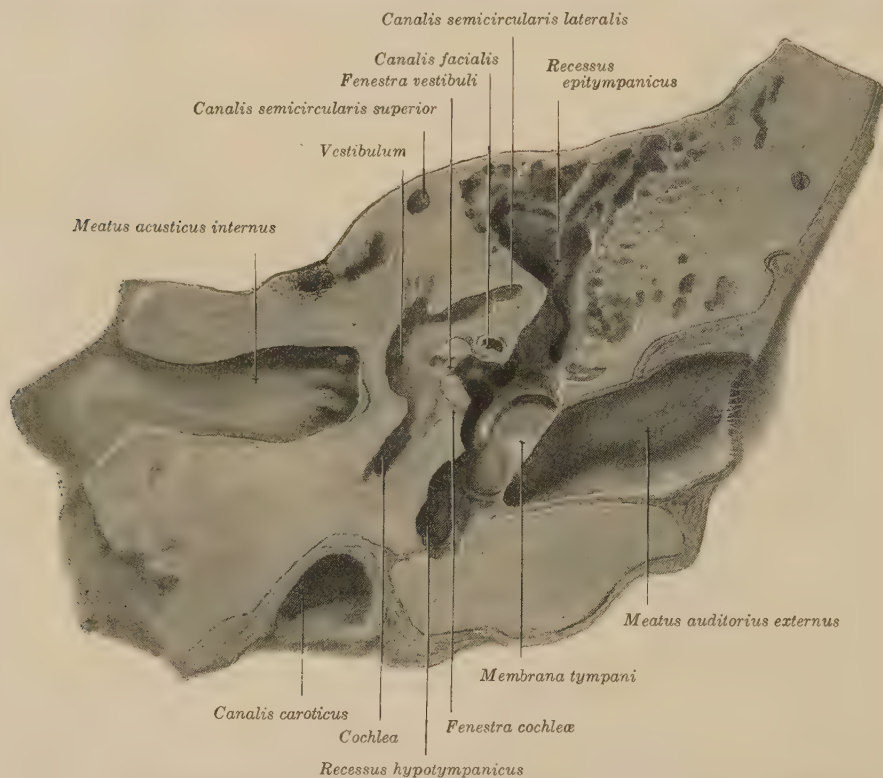


Fig. 213.—Section through the meatus auditorius externus, cavum tympani, vestibule and meatus auditorius internus, right side. Section viewed from posterior.

through the tegmen is the petrosquamosal suture which in the young transmits blood-vessels and lymphatics from the middle ear to the middle brain fossa. The *floor* sinks below the line of the attachment of the *membrana tympani*, thus forming the *recessus hypotympanicus*. Pneumatic spaces occasionally lead off from this region and form the *cellulæ tympani*. Not infrequently the bulb of the jugular encroaches on the floor of the tympanum where it produces a dome-like elevation in which occasionally dehiscence occurs. The *posterior wall* contains a large opening which occupies the upper half and leads to the antrum. This passage is known as the *aditus* and it is part of the space included in the general and more

extensive term "recessus epitympanicus." The *anterior wall* contains the large bell-like opening of the tuba auditiva. The internal carotid encroaches on the tympanum in this region, being separated by only a thin plate of bone. The inner wall is formed in large part by the capsule of the labyrinth. A conspicuous rounded elevation lies about the middle of the inner wall. This is the promontory formed by the first turn of the cochlea. Two openings in this wall lead to the labyrinth. The upper opening lying opposite the upper posterior quadrant of the membrana tympani is the fenestra vestibuli (oval window) into which the foot-plate of the stapes fits. Directly below and separated only by a couple of millimeters is the second opening, the fenestra cochleæ (round window), enclosed by a strong membrane, the membrana tympani secundaria, which separates the tympanum from the scala tympani of the cochlea. Just above the oval window lies the canal for the facial nerve as it passes horizontally through the

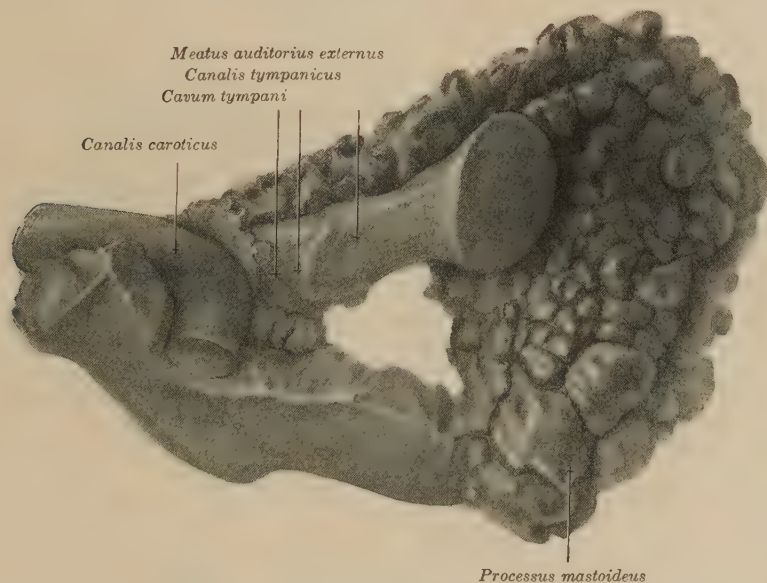


Fig. 214.—Wood's metal cast of left temporal bone showing relations of pneumatic cells of processus mastoideus and meatus auditorius externus and the canal for carotid artery. Note relations of the cellulæ mastoideæ to external meatus.

tympanum. Anterior to this is the canal for the tensor tympani muscle, which terminates posteriorly in a conspicuous bony ledge called the *processus cochleariformis*. Directly back of the promontory is a small bony elevation springing from the posterior wall called the *eminentia pyramidalis*, which is hollowed to transmit the tendon of the stapedius muscle. A small pitting in the bony wall in this region is known as the *sinus tympanicus*.

**Cellulæ Mastoideæ.**—The pneumatic spaces of the mastoid process constitute the remaining part of the middle ear. These cells do not exist in the newborn since the mastoid process is still undeveloped. As the child develops the mastoid process is gradually formed, and as it forms pneumatic spaces develop, leading off from the antrum. These mastoid cells tend to become larger as the margin of the process is approached, the tip usually being occupied by one or several large cells. Pneumatization

of the mastoid process is often incomplete or even entirely arrested, a condition apparently due to early infection in the tympanum and results in lowered resistance of these parts.

The surgical relations of the mastoid process are of the greatest interest because of the necessity of rather extensive operations in this region. The location of the lateral sinus is of importance. This sinus lies along the inner wall of the process at a varying distance from the external meatus and from the outer wall of the mastoid. This anatomical variation in the location of the sinus necessitates great care on the part of the surgeon when operating on the mastoid. The course of the facial nerve through the temporal bone is also of great importance to the surgeon. This nerve enters the temporal bone along with the eighth nerve through the meatus acusticus

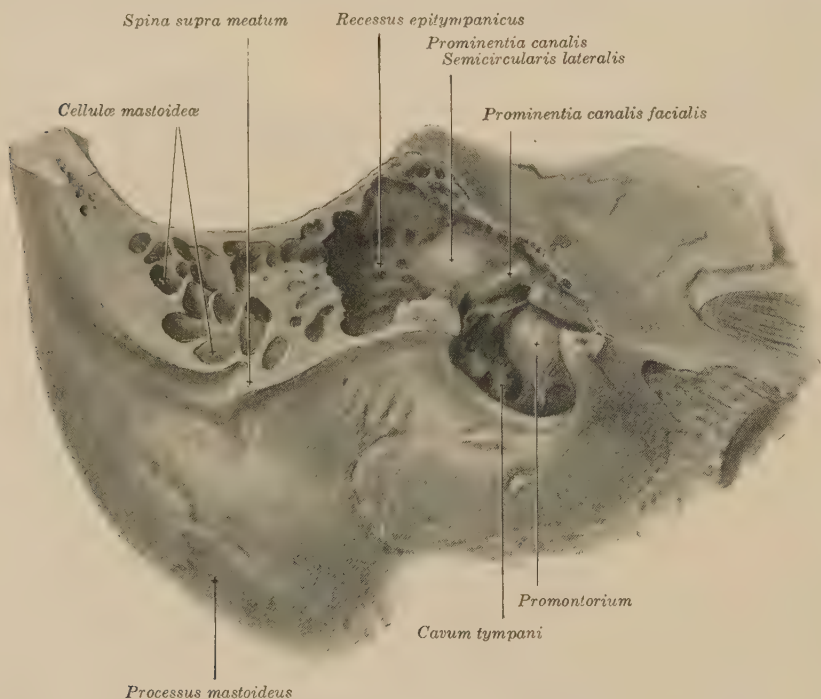


Fig. 215.—Section through upper posterior wall of the meatus auditorius externus and showing relation of cellulae mastoideae and antrum to meatus. Note also relation of antrum to cavum tympani.

internus. It enters the tympanum just above and in front of the oval window. Through the tympanum it takes a horizontal course, lying just above the oval window. When it reaches the posterior wall of the tympanum, it turns abruptly downward and lies along the posterior margin of this cavity in the wall of the external meatus. Opposite the oval window it lies on a level with the inner wall of the tympanum, but as it passes downward toward the stylomastoid opening it lies in the posterior wall of the external meatus and is separated further and further from the level of the inner wall of the tympanum. This latter relation makes it impossible to remove the whole of the posterior wall of the meatus without injuring the nerve.

The *horizontal semicircular canal* lies in the floor of the passage from the tympanum to the antrum where it forms the *prominentia canalis semicircularis lateralis*. Above the mastoid process, the tympanum, and the antrum lies the middle brain fossa; the shelf of bone separating these spaces is very thin and not infrequently contains dehiscence through which infection readily extends to the brain cavity. The petrosquamosal suture passes directly through the tegmen. Atypical pneumatization is also of surgical importance. Such cells as have been pointed out may develop from the osseous portion of the tuba auditiva where they form the *cellulæ tubarium*, from the floor of the tympanum where they constitute the *cellulæ tympani*. They invade more or less extensively the root of the zygoma lying above and in front of the *cavum tympani* and extending occasionally as far forward as the apex of the petrous bone. A large cell occasionally develops internal to the digastric groove.

The outer surface of the mastoid process has several conspicuous markings. At the upper posterior margin of the external meatus is the *spina suprameatum* for the attachment of the superior ligament of the auricle. The upper boundary of the process is marked by a conspicuous ridge, the *linea temporalis*. Not infrequently the surface of the process is indented in the adult by the remains of the petrosquamosal suture which in the newborn opens directly into the antrum. The size of the mastoid process varies considerably in the adult. In the completely pneumatic process it is more or less rounded. When the pneumatization has been arrested the process remains smaller. It is in the latter type where the *sinus lateralis* is usually found lying near the surface and encroaching on the posterior wall of the external meatus.

#### INTERNAL EAR OR LABYRINTH

The labyrinth is the important part of the ear so far as concerns function. It is here that the special sense organs are located. The important advances that have been made in recent years in our knowledge of the diseases of the labyrinth and the fact that operative work now includes the inner ear make it important to include here a description of the anatomy of this part.

**Osseous Labyrinth.**—The bony capsule of the labyrinth is a hard, ivory-like structure which distinguishes it from the surrounding spongy bone. This capsule is 2 or 3 mm. thick and is embedded in the pyramid of the temporal bone. The relations of the osseous labyrinth to the surrounding structures are of importance. The promontory lying on the inner wall of the *cavum tympani* is formed by the first coil of the cochlea. The *fenestra vestibuli* (oval window) is an opening connecting the tympanum with the vestibule. The *fenestra cochlea* (round window) is a similar opening between the tympanum and the *scala tympani* of the cochlea. The apex of the cochlea lies just internal to the tympanic orifice of the eustachian tube. The horizontal canal forms a conspicuous prominence in the floor of the *aditus*. The superior canal forms the *eminentia arcuata* on the superior aspect of the pyramid. On the posterior aspect of the pyramid is the *meatus acusticus internus* for the transmission of the seventh and eighth cranial nerves and the artery of the labyrinth. Near this is the opening of the *aquæductus vestibuli* for the transmission of a tube from the membranous labyrinth known as the *ductus endolymphaticus*. On the lower

margin of the pyramid is the opening of the aquæductus cochleæ, which establishes a communication between the perilymph of the cochlea and the fluid in the subarachnoid space. The osseous capsule receives its blood-supply from the surrounding bone.

The cavities of the osseous labyrinth are best studied by means of Woods' metal casts. In the anterior wall of the vestibule is an oval opening, the fenestra vestibuli, which receives the foot-plate of the stapes and establishes communication with the cavum tympani.

On the posterior wall is the opening for the aquæductus vestibuli. In this wall of the vestibule are two depressions, the recessus sphæricus and the recessus ellipticus for lodging the macula acustica sacculi and the macula acustica utriculi respectively. From the anterior end of the vestibule extends a tubular prolongation which is coiled upon itself like the shell of a snail and is called the cochlea. In man there are  $2\frac{1}{2}$  coils of the cochlea. The central stem of the cochlea is a pyramidal shaped structure

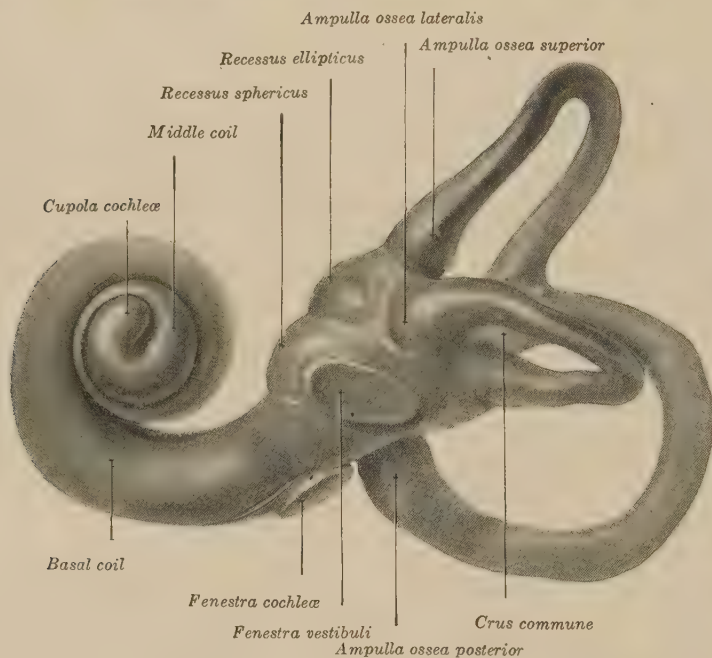


Fig. 216.—Wood's metal cast of the human labyrinth, anterior view.

called the modiolus, which provides channels for the branches of the cochlear nerve and blood-vessels. A bony shelf, the lamina spiralis ossea, extends from the modiolus throughout the length of the cochlea. This bony shelf with a membranous extension, the membrana basilaris, divides the cochlear tube into two spaces: on the one side is the scala vestibuli and on the other the scala tympani. At the apex of the cochlea the two communicate through a funnel-shaped opening called the helicotrema.

From the posterior part of the vestibule spring three *semicircular canals*—the superior, the posterior, and the horizontal. These canals communicate with the vestibule through five openings. One of the cruræ from the superior and one from the posterior canals join to form the crus commune.

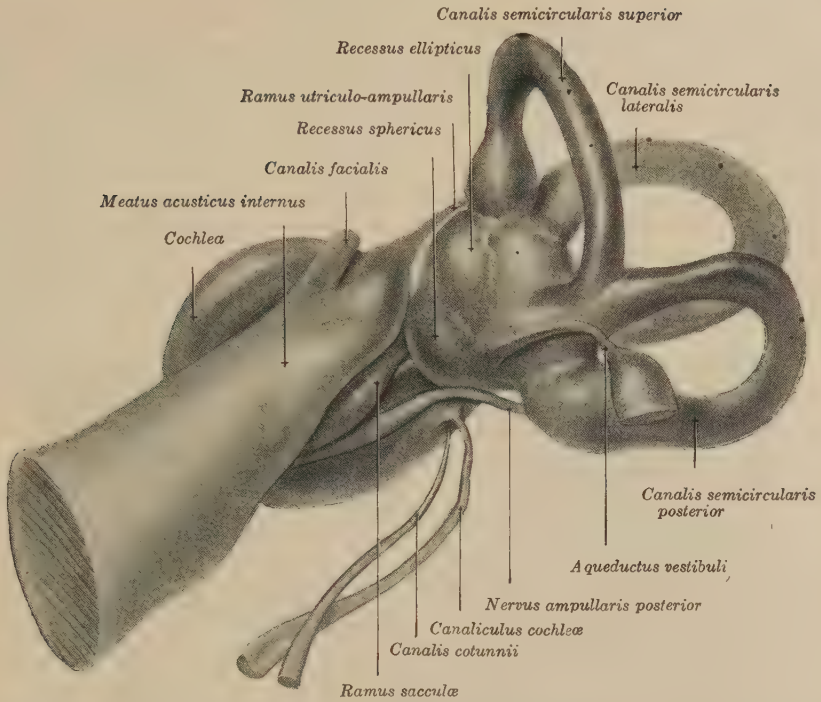


Fig. 217.—Wood's metal cast of the osseous labyrinth, posterior view, showing the internal meatus and the channels for the vestibular and cochlear nerves, also the aqueductus vestibuli and aqueductus cochleæ and the canalis cotunnii for the transmission of the vein of the aqueductus cochleæ.

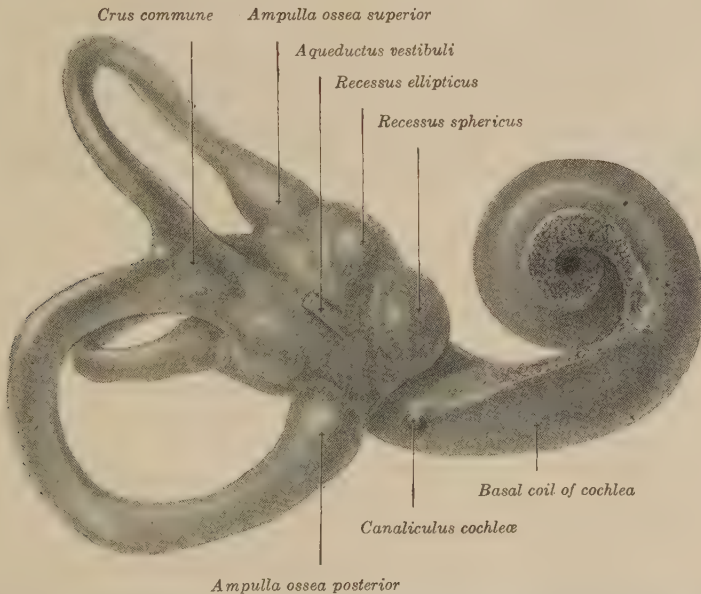


Fig. 218.—Wood's metal cast of the right temporal bone showing meatus acusticus internus and the several canals for branches of the eighth nerve, the aqueductus vestibuli, canaliculus cochleæ, and the canalis cotunnii for the passage of the vein from the labyrinth.

The anterior crus of the horizontal and superior canals and the posterior crus of the posterior canal open into the vestibule through a dilated ending called the ampulla. The three canals occupy the three planes in space, the superior canal on the one side lying in the same plane as the posterior canal of the opposite side.

**The Membranous Labyrinth.**—A membranous labyrinth hangs suspended in the osseous labyrinth surrounded on all sides, except where it is attached to the bony wall, by fluid—the perilymph—the latter filling the osseous labyrinth. In the semicircular canals the membranous labyrinth conforms closely to the shape of the osseous cavity, having its membranous canal and membranous ampulla. In the vestibule there are two mem-



Fig. 219.—Section through oval and round windows showing niche of the oval window with stapes in position, the promontory between the two windows, the niche of the round window and the membrana tympani secundaria separating the cavum tympani from the scala tympani of the cochlea; Corti's organ at the very beginning of the basal coil; the perilymphatic space of the vestibular cavity constituting the scala vestibuli separated from the scala media by Reissner's membrane; the membranous saccule and the macula acustica of the saccule.

branous sacs—the utricle and saccule. The membranous canals communicate with the utricle, while the saccule and utricle are joined through the ductus endolymphaticus.

In the cochlea the membranous labyrinth consists of a tube which follows the coils of the cochlea and is called the ductus cochlearis or scala media. The membranous cochlea or acoustic labyrinth connects with the vestibular or static labyrinth through a tiny tubule—the ductus reuniens (Henseni).

Extending from the saccule and utricle is a membranous tube, the ductus endolymphaticus, which passes out through the aquæductus vestib-

uli to the posterior aspect of the temporal bone where it opens into a closed sac—the saccus endolymphaticus.

The entire membranous labyrinth is developed from the primary otic vesicle, an invagination of the embryonic ectoderm. It forms a closed sac completely filled with endolymph.

The membranous semicircular canal is attached along the convex side of the osseous canal, while the membranous ampulla practically fills the space of the osseous ampulla. Each membranous ampulla contains a prominent transverse ridge, the crista ampullaris, which has a rich vascular supply and which conducts terminal filaments of the vestibular nerve to the layer of sensitive hair-cells with which the whole of the crista is covered. Superimposed above the hair-cells is a peculiar epithelial product, the cupula, into the under surface of which the hairs of the hair-cells can be traced. This is the special sense organ of the semicircular canals called the

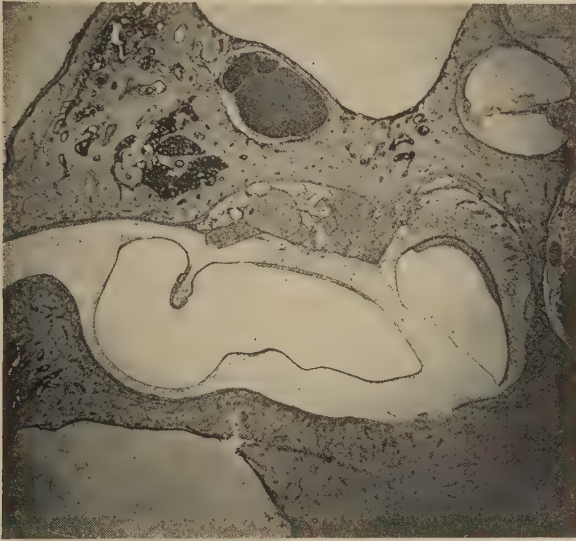


Fig. 220.—Section through the labyrinth showing crista acustica ampullaris, macula acustica of the utricle, also macula acustica of the saccule. Section also through first coil of the cochlea.

crista acustica ampullaris. Movement of endolymph from the canal toward the utricle or from the utricle toward the canal cannot take place without exerting pressure on the cupula and producing an interaction between this structure and the hair-cells, which interaction constitutes the normal stimulation of this end-organ.

In the vestibule the utricle and saccule are attached to the bony walls in the recessus ellipticus and recessus sphæricus respectively. At the points of attachment the epithelium of the membranous labyrinth forms a special sense organ called the macula acustica, which consists of hair-cells similar to those of the crista acustica and a superimposed membrane, the otolith membrane, to the under surface of which the hairs are attached. These two end-organs lie in planes at right angles to each other. The hair-cells of the macula acustica receive the terminal filaments of the vestibular nerve just as do the hair-cells of the crista. Both of these end-organs retain

the term "acoustic," applied to them when they were believed to be a part of the sound-perceiving mechanism, although neither of these end-organs is believed now to have anything to do with hearing.

The structure of the membranous cochlea is even more complicated. We have already seen how the osseous cochlea is practically separated into two chambers by a bony ledge, the lamina spiralis ossea. The membranous extension of this partition, the membrana basilaris, renders the separation complete. The membranous cochlea lies above the membrana basilaris. An extremely delicate membrane, the membrana vestibularis (reissneri), separates the membranous cochlea, the ductus cochlearis, from the scala vestibuli. The outer termination of the basilar membrane is attached to a fan-shaped fibrous band called the spiral ligament.

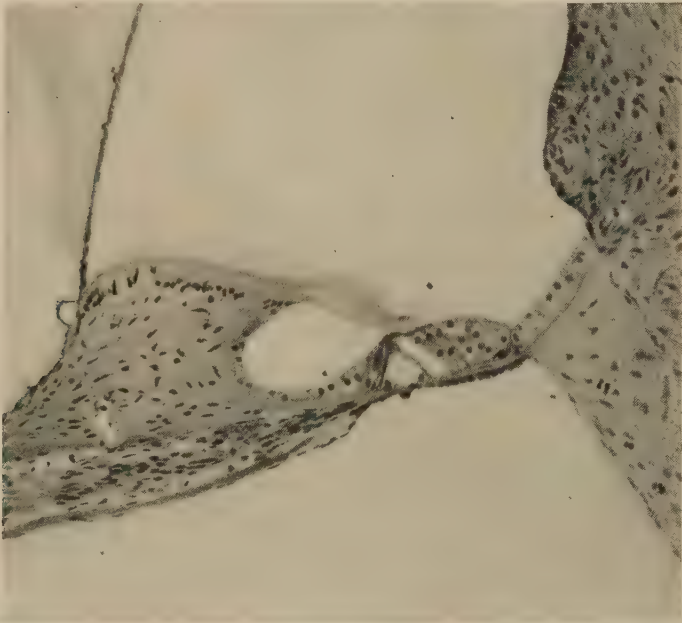


Fig. 221.—High-power magnification of Corti's organ in the middle of the basal coil of the cochlea showing the details of this organ; membrana tectoria in position.

The outer wall of the ductus cochlearis is divided into two unequal segments by a spiral ridge called the prominentia spiralis. The larger segment above is occupied by the stria vascularis. The smaller segment below forms the sulcus spiralis externus. The stria vascularis, as its name implies, has a rich vascular supply, the blood-vessels lying just beneath the superficial layer of epithelium. Several layers of cells form the deeper structure. These deeper cells are derived in part from the superficial layer of epithelium and in part from the connective tissue of the spiral ligament.

The epithelial lining of the sulcus spiralis externus is easily distinguished (by staining qualities) from the cells of Claudius, which rest on the basilar membrane external to Corti's organ. The cells of the sulcus externus are a continuation of those covering the spiral prominence, and are directly continuous with the surface epithelium of the stria vascularis. They moreover resemble the cells of the stria in possessing long fibrillar processes

which penetrate the structure of the spiral ligament. The epithelium of the sulcus externus is peculiar also in that there are scattered throughout the basal coil areas where these cells form clumps which penetrate the structure of the spiral ligament. These clumps of epithelium contain tubules which open on the surface and clearly constitute a glandular mechanism, probably the glands for secreting the endolymph.

On the basilar membrane rests a highly specialized epithelial structure, the organon spirale of Corti, the end-organ of the membranous cochlea. Conspicuous in the formation of this end-organ is the tunnel of Corti formed by the pillars or rods of Corti. The inner of these rods rests on the fixed structure of the lamina spiralis; the outer rod alone rests on the basilar membrane. The important physiological element in Corti's organ is the same peculiar hair-bearing cell already noted in the crista and macula

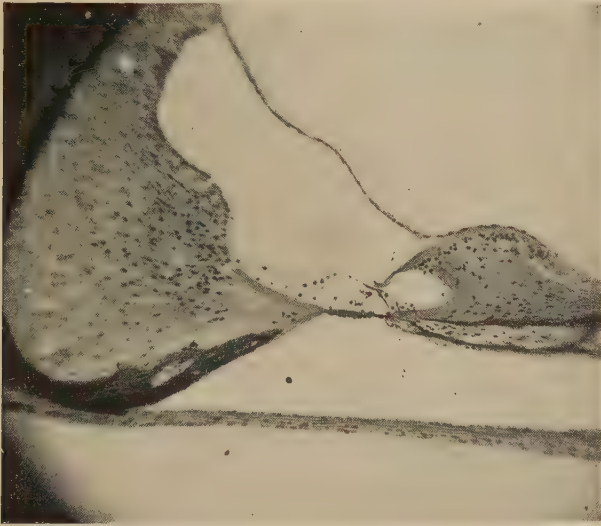


Fig. 222.—High-power magnification of Corti's organ near the beginning of the basal coil showing membrana tympani secundaria, scala tympani, scala media separated from the perilymphatic space of the vestibule by Reissner's membrane.

acustica. There are four rows of these hair-cells. One lies against the inner rod; the other three rows are placed external to the outer rod, from which they are separated by a narrow space called the space of Nuel. The hair-cells do not rest on the basilar membrane, but are separated by a row of supporting cells. These hair-cells receive the terminal endings of the cochlear nerve. The filaments for the outer cells can be seen coursing through the tunnel of Corti and the space of Nuel. Externally the organ of Corti is flanked by a row of tall, clear cells, the cells of Claudius, which extend to and partly cover over the peculiar epithelium already described which occupies the sulcus spiralis externus. Where the basilar membrane joins with the crista of the spiral ligament there is located in the basal coil a small clump of cells covered over in part or completely by the cells of Claudius. These are the cells of Böttcher, the function of which is not known. The outer termination of the lamina spiralis is occupied by a structure made up of connective tissue and epithelium from the top of

which a peculiar membrane, the *membrana tectoria*, takes its origin. This is a structure analogous to the cupula of the crista and the otolith membrane of the macula. The same relation exists between the *membrana tectoria* and the hair-cells of Corti's organ as we have seen existing between the cupula and the otolith membrane and the hair-cells of the other two end-

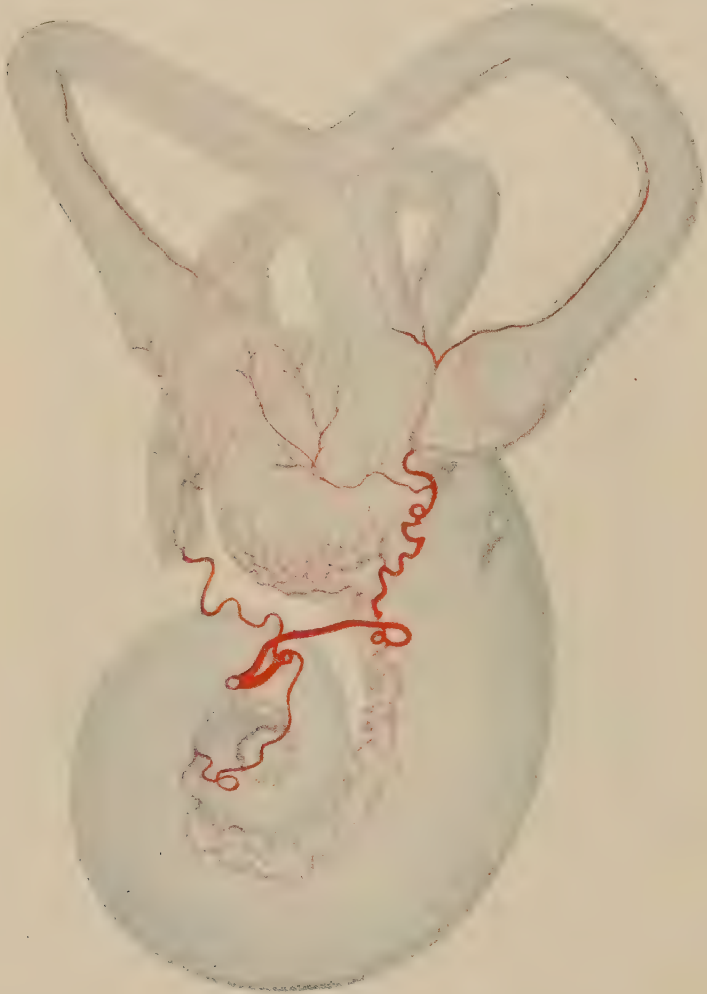


Fig. 223.—Arterial supply of the human labyrinth. Note that a single vessel, the labyrinthine artery, supplies the entire labyrinth, that the first branch, a cochleovestibular branch, supplies the proximal two-thirds of the basal coil, the macula acustica of the sacule and the crista ampullaris of the posterior canal. The next branch divides into two trunks, one following the modiolus to the upper coils of the cochlea, the other the ramus utriculo ampullaris to the macula of the utricle and the cristæ of the horizontal and superior canals.

organs. The hairs of the hair-cells are invariably in contact with and adherent to the under surface of the *membrana tectoria*.

On the under surface of the *membrana tectoria* is a spiral band known as the *streifen* of Hensen. In the older conception of the tectorial membrane this structure was represented as separated from Corti's organ, and

there was no clue to the significance of this *streifen* of Hensen. Recently I have been able to demonstrate that this constitutes a facet for the attachment of the tectorial membrane to the supporting cells just internal to the inner row of hair-cells, an anchorage for holding this membrane in place. The organ of Corti varies in size from one end of the cochlea to the other. At the beginning of the basal coil it is extremely tiny but becomes larger and larger toward the apex of the cochlea.

**Blood-supply of the Labyrinth.**—Interest in the blood-supply of the labyrinth is enhanced because of its bearing on clinical otology. The Ménière syndrome—the sudden occurrence of deafness, tinnitus, and vertigo—although by no means always attributed now to hemorrhages in the labyrinth, is no doubt usually dependent upon some circulatory disturbances. The question of extension of infection from the middle ear to the labyrinth and of extension of labyrinthine infection to the intracranial cavity are questions which have to do in a large measure with blood-vessel communications.

The internal ear has for its sole blood-supply a single vessel, the *labyrinthine artery*. This vessel, according to Siebenmann, is a branch of the basilar artery. The vessel enters the labyrinth through the meatus acusticus internus. It is not feasible here to give a minute description of the coursing of the branches of the labyrinthine artery through the internal ear. There are certain outstanding features of this distribution which have important clinical possibilities, especially when we bear in mind that the branches of the labyrinthine artery are in the nature of end-arteries, that is vessels which constitute the sole supply for definite areas.

In the fundus of the internal auditory meatus the labyrinthine artery gives off as its first branch a vessel which can be termed the “cochleovestibular branch,” since it supplies part of the cochlea and part of the vestibular apparatus. This vessel divides into two trunks: One provides the blood-supply for the proximal two-thirds of the basal coil of the cochlea, the other courses posteriorly under the name of the posterior vestibular artery.

The first important area supplied by this vessel is the macula acustica sacculi. The posterior vestibular artery sends a branch horizontally across the vestibule, supplying, however, only the periosteum and not reaching the membranous labyrinth. The ampulla of the posterior semicircular canal receives its blood-supply from the posterior vestibular artery. The continuation of this vessel supplies the posterior crus of the posterior semicircular canal, the posterior crus of the horizontal, and the crus commune.

After giving off this cochleovestibular vessel, the continuation of the labyrinthine artery again divides into two trunks. One penetrates the modiolus and constitutes the arterial supply for all of the cochlea except the proximal two-thirds of the basal coil. The other branch continues posteriorly following the ramus utriculo-ampullaris as the anterior vestibular artery. This vessel supplies the important capillary areas of the macula acustica utriculi as well as the crista acustica of the superior and horizontal canals. Continuations of the anterior vestibular artery supply the anterior crurae of the superior and horizontal semicircular canals.

It might be well at this point to emphasize again the clinical bearing which this branching of the labyrinthine artery has. In the first place it is possible by the lodging of an embolus in the labyrinthine artery to destroy completely the function of both the vestibular and cochlear mech-

anisms. An embolus lodging in the cochleovestibular vessel would disturb the function of the proximal two-thirds of the basal coil of the cochlea, that is the part of the cochlea which we believe has to do with the perception of tones toward the upper end of the tone scale. At the same time there would arise disturbances of equilibrium because of the suppression of the blood-supply for the macula acustica sacculi and the crista acustica of the posterior canal.

In the same way an embolus lodging in a continuation of the labyrinthine artery should be able to suppress the hearing for the lower tones which we believe are taken up by the organ of Corti in the upper coils of the cochlea. This same embolus should be able to produce a profound disturbance in equilibrium because the function of the macula acustica utriculi and that of the crista acustica of the superior and horizontal canals has been disturbed.

Furthermore, when we bear in mind that the organ of Corti throughout the entire length of the basal coil is supplied by vessels which radiate out from the modiolus along the lamina spiralis ossea, the terminal branches of which are in the nature of end-arteries supplying definite circumscribed areas of Corti's organ, it becomes apparent that disturbances of these separate terminal arterial twigs would be capable of disturbing the function of definite circumscribed areas in Corti's organ.

The venous system of the labyrinth is arranged so that the veins instead of leaving along with the artery, that is through the meatus acusticus internus, leave along an entirely different route. In man there are two veins which drain blood from the internal ear. One is the vein of the aquæductus cochleæ, which passes through the canalis cotunnii. This vessel drains all the blood from the cochlea and part of the blood from the vestibule and semicircular canals. The venous blood which is not collected by this vessel leaves through a vein which passes out along with the aquæductus vestibuli.

There is a decided variation in different individuals regarding the distribution of the veins from the vestibule between the outlet at the aquæductus cochleæ and the outlet along the aquæductus vestibuli.

The blood-supply of the labyrinth constitutes a closed system, that is a system in which there are no communications with the surrounding structures. The bony capsule of the labyrinth itself receives its blood-supply from the structures which surround it. In the tympanum, for example, the blood-supply for the promontory is from the same vessels which supply the mucous membrane lining the middle ear. It is doubtful whether in man there exist any communications between the blood-vessels supplying the osseous labyrinth and the branches of the labyrinthine artery itself. Such communications I have found exist in the labyrinth of the calf.

There are a few points in the distribution of the blood-vessels in the internal ear which have a peculiar interest. In the first place, Reissner's membrane in man has no blood-vessels. In several of the lower species Reissner's membrane is a vascular structure. The membrana basilaris is always a vascular structure. Vessels radiating out along the lamina spiralis ossea terminate in a circular vessel which lies under the tunnel of Corti. Not infrequently there are two circular vessels lying parallel in this region. The zona pectinata of the basilar membrane, that is the part lying between the tunnel of Corti and the spiral ligament, has been described usually as

quite free from blood-vessels. I have found that in the pig, the calf, and the sheep this area has an occasional vessel which connects the vessels under the tunnel of Corti with those which supply the spiral ligament.

GEORGE E. SHAMBAUGH.

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## EXTERNAL EAR

**General Consideration.**—The anatomicophysiological knowledge is of course very important in understanding the management of the diseases of this structure; yet it is not within the province of this chapter to go into any detailed descriptions. However, one will remember that the external ear is made up of both bone and cartilage; lined or covered by skin with all its tributaries. The blood and nerve supply is very essential in this skin covering especially in that part of the external ear where it becomes a part of the middle ear, that is the external or dermal layer of the tympanic membrane. Here are some of the finest nerve-endings, capillaries, lymphatic and particularly that epithelial lining that gives to that structure, the tympanic membrane, the pearly gray appearance and the opportunity for a light reflex. Again in the skin of the external canal at the bony cartilaginous junction some special glandular elements such as the ceruminous glands which supply that wonderful golden-yellow material in the child (ear-wax). There are also located here especially large glands of the sebaceous type which play an important rôle in disease of this part of the ear. At the meatal opening one finds the regional development of hair (vibrissæ) acting with the cerumen and sebum as protection to the delicate tympanic membrane. The outlines, position, curves, and depressions of the specially typed elastic cartilage of the pinna and canal are so constructed as to best serve the special function of hearing. Thus, they catch the sounds, deflect and reflect them in such a manner as to produce the best effect on the more delicate structures of the middle and internal ear. Of greatest interest (at least to the writer) is the embryology of the ear and not least this external part of it. Particularly is this true when we consider the various shapes, sizes, types, curves, points, lines, etc., about the auricle and canal in each individual, differing not only in each person but in the individual. The two ears are never just alike, although more nearly so than the ears of one person and another. The writer believes that ear-casts would be by far more accurate identification than finger-prints. The disturbances as a consequence of some misstep or mishap in development of the ears are productive of congenital malformations, a subject of much speculation (also of great interest to the writer particularly as to the causes of these mishaps of undevelopment and maldevelopment).

In consideration of the diseases of the external ear the otologist comes in close contact with at least two other special branches; namely, dermatology and general surgery. Consequently on several topics the reader will have to be referred to the text in those specialties for a more detailed description. We shall consider the subject of external ear diseases in the following groups: (1) Foreign bodies; (2) inflammatory; (3) traumatic; (4) new growths; (5) congenital deformities; (6) vascular. We shall also

divide their consideration into: (a) The pinna; (b) external auditory canal. The external auditory meatus is not considered separately but is treated in connection with either of the two above-mentioned divisions. The dermal layers of the tympanic membrane will not be considered in connection with the external ear diseases but rather left for him who will treat the subject of the middle ear. However, references will be made to some pertinent changes of this structure whenever there be reasons for so doing.

References appended and appear in alphabetical order.

#### FOREIGN BODIES

Foreign bodies find their way into the meatus in great variety (most frequently in children), of which pebbles, sand, seeds, and buttons are the commonest objects introduced; older children insert little paper balls, and adults cotton worn as protection dropping into the fundus. Once in the meatus, the article may remain because the child is unable to get it out or else it is forgotten. The ears of the insane and the feeble-minded are receptacles of various inanimate objects.

Animated intruders, particularly small flies and bedbugs, sometimes find their way into the meatus, causing great discomfort by movements across the drum membrane.

**Treatment** consists in removal of the foreign body by irrigation or mechanical manipulation. In the removal of an insect from the ear, the introduction of a drop of chloroform followed by washing is advocated.

**Cerumen or Ear-wax.**—Not only must the accumulation of this normal secretion be considered but the excessive formation as well. The cause is not known but it is believed that some pre-existing acute inflammation in adult life such as an erythema, dermatitis, eczema, or some chemico-mechanical irritant persisting for some time is followed by excessive formation of the wax. It becomes inspissated with dust and other foreign material, thus becoming changed in its consistency (especially found in certain occupations, *i. e.*, bakers, street-cleaners). This mass remains lodged at the lower and anterior portion of the canal just above the depression made by the position of the condyle of the lower jaw. Gradually more and more accumulates so as to advance toward the tympanic membrane as well as externally. The fatty part of the cerumen gradually disappears and from the irritation, epithelium of the canal, that has become desquamated, becomes a part of the wax-plug. There still, however, remains a good sized slit above and behind the accumulation, consequently not much disturbance is experienced by the patient. If the wax-plug, through some thermal, mechanical, or chemical influence becomes affected so as to swell, it will at once produce symptoms. The commonest of these is a sense of fullness in the ear and difficulty in hearing. Should the swelling of the plug be great, pain in the ear radiating to the temple or down the neck will be experienced. In case this material reaches inward sufficiently to touch the tympanic membrane, pain from that region as well as tinnitus aurium is experienced. The writer has had more than one case of cerumenal plug causing dizziness, vertigo, and tinnitus, and symptoms of very grave ear diseases, which when the wax was removed, disappeared. The writer has never seen a nystagmus in connection with any of these severe cases. The recognition of the wax-plug is comparatively easy by its brownish dark appearance, being located quite near the external auditory meatus. When patients

present themselves with this complaint, the entire circumference of the canal is usually filled, but if, as most frequently happens, the wax is discovered in the course of a general ear examination, one will see beyond the plug in the posterior and upper part of the canal. This point should be well remembered in connection with the removal of the wax-plug by means of washing, as will be described later.

The differential diagnosis is first, some other foreign substance; second, neoplasm; third, inspissated masses of cholesteatoma of the external auditory meatus; fourth, otolithiasis and osteophytic masses; and fifth and most important are chronic suppurative middle ears with cholesteatomatous formations which become hard and inspissated with dirt and wax. Rarely does a fungoid growth of the aspergillus variety become so solid as to be mistaken for a wax-plug.

**Treatment.**—*Removal.*—The best method and that which is least likely to traumatize the canal is syringing. Warm bicarbonate solution or salt water or even plain water drawn into a pint size syringe with a blunt-pointed nozzle is used. Said nozzle is directed upward and backward, the auricle being lifted up and back, thus straightening the canal. By a steady firm drive of the piston a forcible stream of water passes above the wax-plug toward the tympanic membrane. The solution returning, pushes the wax-plug outward. Usually after one or two attempts the plug is delivered with a splash into the waste dish. Should that not occur, one should again make an examination so as to be certain that one is dealing with a wax-plug and if so to determine whether it has changed its position so as to prevent the current of water from being admitted. By some slight manipulation with a probe, care being taken not to traumatize the canal, the position of the wax-plug may be changed and the next syringeful of water will deliver it. In the event that the plug is not expelled, one may assume that the impaction is so marked and the plug so inspissated that softening is necessary. To this end a prescription of sodium bicarbonate, 20 grains, in glycerine and water  $\bar{a}\bar{a}$   $\frac{1}{2}$  ounce, is given the patient for home use, with direction to fill the canal twice a day for three days, warming the solution preliminary to its use. Following this, the removal of the plug is usually certain and easy. However, should it continue to resist removal and one is certain that it is an inspissated mass of wax, one will proceed to remove it piecemeal and with care, attempt being made during the procedure to remove it with the aid of the syringe. The mass having been removed, one should inspect the ear with special reference to traumatized areas, old perforations in the tympanic membrane, etc. The tympanic membrane is frequently flushed after the removal of the wax-plug, but this is only transient. One will do well, especially in those cases where the ear has been blocked for some time, to thoroughly block the canal with cotton and advise the patient to avoid noisy places because of the possibility of the patient suffering from an attack of vertigo due to irritation of the labyrinth. This precaution is of course only necessary for a few hours until the ear is again accustomed to the external noises. The prevention of re-formation of the wax-plug is in the avoidance of irritants, dusty dirty places, and re-examination of the ears at least twice a year. In cases of more rapid re-formation of the wax-plug, more frequent examination is advisable.

**Otolithiasis** is a concretion formation within the auditory canal usually associated with chronic suppurative otitis media and desquamated otitis

externa. These concretions feel hard to the touch and with a probe are somewhat movable. When removed and examined they will often show a foreign body in the center. The writer had a case of this kind which showed, on close inspection, a small piece of cotton in the center.

### INFLAMMATORY CONDITIONS

**Eczema** of the external ear (*eczema aurium*) may occur as an independent affection by extension from an eczema of the auditory canal or in association with an eczema of the face, especially in infants and young children in which there is an associated involvement of the auricle. In children the eczema, when limited to the auricles, is frequently seen on the posterior aspect and particularly in the crease. Crusting and fissuring are frequently associated. Like eczema elsewhere the process may be acute, subacute, or chronic, and may be associated with single lesions or a combination of the predominant lesions, viz., erythema, papules, pustules, and vesicles with the associated weeping, fissuring, crusting, and the subjective sensations of burning and itching.

The type most frequently seen upon the face is the *erythema type* of eczema which is seen in middle aged and old people, and by extension may involve the ears. It usually consists of one or more hyperemic patches of variable size, pale or bright red in color, and associated with itching and burning. The skin is harsh and dry, of reddish color with violaceous or yellowish tinge. There is some thickening with scaling, and a tendency toward oozing in areas. The process is seen in those subjected to exposure or extremes in temperature, and it may be aggravated by indulgence in rich foods or alcoholic stimulants. The course is usually chronic.

*Papular eczema* is rarely seen in the ears, occurring chiefly on the limbs and occasionally on the trunk.

*Vesicular eczema* may occur on any part of the body; but the face and scalp of infants being most common sites, involvement of ears by extension is therefore frequent. The course is usually chronic with acute exacerbations. The lesions consist of vesicles aggregated or closely crowded with scattered discrete lesions in which may be found papules and pustules. There is usually noted considerable inflammation with edema and swelling. When the vesicles rupture a raw, weeping, crusted surface may result.

The *pustular type* of eczema is seen in the scalps of infants; although not a frequent type, it may develop from the vesicular type just mentioned to which it is similar except that the lesions contain pus instead of serum. This type is essentially chronic and is seen particularly in ill-nourished individuals especially children and young people. A pustular eczema of the scalp and region behind the ears is frequently seen due to pediculi, and the associated presence of nits in the hair serve to establish the etiology.

*Etiology*.—The etiology of eczema involves a study of many factors referable to both external and constitutional factors as active producing agents. Gout, rheumatism, digestive disorders, disorders of assimilation, general wasting, hypotension of the nervous system, allergy, and hypothyroidism are important constitutional influences. Extremes of temperature, irritants of chemical or vegetable nature are external factors.

*Pathology*.—The pathology of eczema is situated chiefly in the rete and papillary layer, and consists of a simple inflammation of the skin with later involvement of the corium and subcutaneous tissue in long standing cases.

*Treatment.*—The successful treatment of eczema is based on attention to both local and systemic measures: Hygiene, regulation of diet, finding of reactions of allergic nature to certain articles of diet, general tonic and eliminative treatment. Local treatment consists in restricted but judicious use of soap and water, and local applications of a soothing nature in those cases associated with acute inflammation. In the chronic or indolent types the use of moist dressings of aluminum acetate (1 ounce to 1 pint water), calamine lotion, use of oily preparations to remove crusted lesions, control of itching, and in infants binding of arms to prevent scratching, and dusting powders in erythematous acute cases are valuable. In the type associated with oozing and fissuring behind the ears the following ointments are serviceable:

No. 1: Calamine.....	4.....	̄j
Zinc oxide ointment.....	28.....	̄j
or		
No. 2: Ichthyol.....	0.90.....	Gr.xij
Zinc oxide.....	3.60.....	̄j
Calamine.....	30.00.....	̄j
Ung. aqua rosæ ad.....		
or		
Crude coal-tar, using formula No. 2.....	2.....	̄ss
Crude coal-tar.....	2.....	̄ss
Zinc oxide.....	16.....	̄ss
Cornstarch.....	16.....	̄ss
Petrolatum.....		

In eczema of the auditory canal a smooth ointment consisting of 2 to 3 per cent. salicylic acid to the ounce is advised.

*Eczema secondary to chronic discharging middle ears* is frequently observed in otological practice.

The symptoms of this disease of the external ear are no different than those described above. The most important fact to remember is the etiology, namely the chronic middle ear disease, because the cure cannot be hoped for unless this latter is first cured. Conversely, the middle ear disease is frequently aggravated by the eczema which is sometimes associated with the narrowing of the external auditory meatus, preventing proper ventilation and drainage. It stands to reason that here we have one of the most discouraging types of ear diseases to treat, and it has been the experience of the writer to be compelled to do a radical mastoid for the cure of the chronic suppuration of the middle ear before a cure could be established.

The treatment of the eczema of the canal is the same as that described above.

In connection with eczema of the canal we must consider other types of skin inflammation which perhaps behave differently in this locality than anywhere else, as for example that form of dermatitis in which the epithelium desquamates very rapidly and accumulates and mixes with the wax, sebum, and other foreign material. There is created a mass that again by its foreign position irritates the underlying skin, often becoming infected, and may even ulcerate. This condition is known as *cholesteatoma externa*. This process has been known to spread to such a size as to perforate the tympanic membrane or cause necrosis with sequestration of the external canal, especially is this possible in older and decrepid persons. Following

such condition after healing, narrowing or stricture of the canal may result.

The treatment of this desquamative form is no different than that described for eczema. However, the prevention of accumulation of the masses, ulcerations, and stricture formations is most important.

In the differential diagnosis one must recognize the disease of *seborrheic dermatitis* which has the grease-like scales associated with the condition due to the fat contained in them. Seborrheic dermatitis involving the auricle usually occurs in association with a scalp involvement and the auricle contiguous to that, which is usually the site of a low-grade dermatitis with the presence of greasy crusts and scales. The condition is most frequent between the ages of fifteen and thirty. The ear canal as well as the pinna may be the site of the disease.

*Treatment* consists in general tonics and the external use of sulphur, salicylic acid, and resorcin.

Recently, encouraging advances have been reported in the treatment of eczema by the use of the quartz light, Roentgen ray, and radium.

**Otomycosis** (otitis externa parasitica), or an involvement of the external auditory canal with a fungus infection, although comparatively rare, is an exceedingly troublesome condition. The entire canal including the drum may be involved with a moist looking coating like blotting-paper, of dirty gray or brownish-gray color, with occasional scattered yellow, green, or black spots. When the scabs are removed the underlying surface is noted to be abraded and raw looking, bleeding easily. Itching, stinging, and pain of varying degrees are complained of. Hearing may be somewhat impaired, and a watery discharge may be noted.

The exact etiological fungus is not definitely known, but it is believed to be due to *Aspergillus niger* and *A. glaucus*.

*Treatment.*—The condition is fairly resistant to treatment, which consists in washing the canal with a weak alkaline solution and the application of a mild parasiticide. Salicylic acid, 10 per cent. in alcohol or sodium hypsulphate solution 1 per cent. is also valuable. Recently quartz applications energized by a water-cooled mercury vapor apparatus has given satisfactory results.

**Erysipelas.**—Most cases of erysipelas of the external ear are observed for a few days following a mastoid operation, during the dressing of the wound. The initial chill with rapid rise of temperature usually causes the clinician to be looking for some complication of mastoid disease as sinus thrombosis or brain abscess. However, within a few hours, the auricle will show the characteristic swelling and the red line of demarcation.

Erysipelas may occur about the ear as well as anywhere else, and the writer has seen a case of such marked infiltration into the entire pinna with subsequent abscess formation that finally terminated into a great deformity from shriveling up.

*Treatment.*—Painting the entire ear and beyond it as far as the hair line with ichthyol-collodion is indicated. Should blisters and abscess form, it is important here to evacuate early to prevent the chondroperichondritis with the inevitable deformities. The support of the patients general condition and prevention of complications, especially a nephritis, are advised. A number of cases of erysipelas in other regions than the ear have recently

been given intravenous injections of mercurochrome with very good results. The writer used it in one case successfully. Cold compresses with Epsom salt or aluminum acetate wet dressings (1 ounce to a pint of water) is efficacious. Recently, success has attended the use of the new anti-erysipelas serum.

**Furunculosis** develops usually at the junction of the pinna with the external auditory meatus. It is essentially a perifolliculitis. There is a marked reaction about the follicles with closure of the canal at that point; especially is this true when more than one abscess is forming. There is found an edema in the close proximity of the furuncle as for instance the tragus and parotid region or retro-auricular (mastoid) region simulating a mastoiditis. The process may be only of a mild character or it may be so severe as to cause a marked breaking down, even a sequestration of some of the bone of the canal. Cases have been known to break into the mastoid cells or rupture in the anterior and downward direction reaching the retromaxillary fossa.<sup>3</sup>

The *symptoms* are usually out of proportion to the findings on examining the canal. The pain is often severe, and to lie on the affected side is at times impossible. Any manipulation of the auricle is very painful, and this is the most valuable diagnostic symptom of the condition. Frequently the opening of the jaw is quite restricted on account of the pain in the ear. The progress of liquefaction is quite slow, and since furunculosis is usually multiple, it is also progressive so that while one furuncle has broken down, eventually discharging, or been opened, another has formed. In the event of opening or spontaneous rupture, the pus obtained on smear or culture will show it to be *Staphylococcus albus* or *aureus*.

The *differential diagnosis* is (1) between an otitis media and mastoid, and (2) parotiditis. The cardinal differential point in the first is the hearing, which is very little changed, if any, in furunculosis, whereas in mastoiditis or otitis media it may be much reduced. The tenderness over the mastoid in furunculosis is elicited on superficial pressure, while in mastoid disease it is on deep pressure. The roentgenogram clinches the diagnosis. The reader is, however, cautioned against the possibility of there having been an acute mastoiditis previous to the development of a furunculosis, or there may be present a non-pneumatization of the mastoid, in which event the roentgenogram may show a cloudy mastoid. In parotiditis there will be diffuse swelling in the region of the gland with an encroachment on the canal without any point of redness or tenderness in the same. The hearing is unaffected and there is usually dryness on that side of the mouth.

In the *treatment* the control of the pain as well as the reaction from the infection is best accomplished by warm moist dressings in the canal as well as about the auricle, employing the dilute acetate of aluminum solution; topically, metacryselyn or cotton in the canal. The use of the hot-water bag and internal administration of pyramidon, aspirin, or salicylates is indicated. Furuncles should not be incised before liquefaction has occurred, and the use of ethyl chloride spray will minimize the pain of this procedure. After opening, squeezing, mopping, and swabbing should be avoided so as to prevent the spread of the infectious material. The material may be well taken up by capillary suction. Ointments, particularly ichthyol in vaseline, 10 per cent., employed after the furuncles

have been emptied will prove beneficial. From the pus a rich culture should be grown, eventually recultured two or three times for an autogenous vaccine which should always be employed to hasten the healing as well as prevent recurrence. One may also use the staphylococcic stock vaccines as well as various non-specific protein substances, such as yatrene-casein to which the writer is partial. This is obtained in ampule form. Heat from a 500-watt lamp is a valuable adjunct especially when the pain is pronounced. This should be used for at least twenty minutes and the lamp placed about 6 inches from the affected ear.

**Tank or Beach Ear.**—In recent years during the hot summer months when people, particularly in large cities, crowd our beaches and pools for swimming, there have arisen large groups of inflamed ears following these exercises. This is due to the fact that the water is allowed to remain in the canal too long with the result that maceration takes place. This, in turn, causes itching and in response to the urge to relieve this the patient employs the finger-nail, toothpicks, matches, hairpins, pencils, etc. for scratching, thus infecting the canal.

The symptoms are oftentimes very stormy. Great pain and swelling with considerable deafness are present. Since the act of swimming is brought into the history one must distinguish between inflation otitis due to swimming and diving which may coexist. Since inspection of the tympanic membrane is often difficult or impossible the differential diagnosis is not easy.

The treatment employed should consist of hot light, ichthyol packs, or packing with acetate of aluminum solution. Surgery is seldom required and only when a real abscess has formed.

**Pruritus of the External Auditory Canal.**—This is one of the most distressing conditions for otologists to handle since it is most refractory to treatment and the findings are negative excepting a traumatized surface due to insults offered by the patient in scratching the canal with all sorts of usually infected implements. The etiology is unknown although the writer has seen quite a few cases of this severe itching disappear when an impacted molar was removed.

The most satisfactory treatment appears to be found in the use of the quartz rod energized by a water-cooled mercury vapor lamp and the instillation of 10 per cent. salicylic acid in alcohol.

Speaking of sensations in the canal, one cannot help but recall the striking reaction of infants to manipulation of the canal as in wiping or packing the same. In speculating as to why this occurs one wonders whether this cannot be explained on the Freudian basis.

**Perichondritis, Chondritis, and Abscess—Acute, Subacute, and Chronic.**—These will vary depending principally upon the organisms present as causative factors. They will also depend upon the resistance of the individual, whether a patient has an underlying tuberculous, syphilitic, diabetic, nephritic, or anemic condition.

The type of perichondritis that is of particular importance to the otologist is that following the performance of the plastic procedures of some radical mastoid operations. A common habitat of the *Bacillus pyocyaneus* is the external auditory canal, and in making the incision of the membranocartilaginous portion of the canal the cartilage becomes infected with said organism, producing at times (the writer has never seen this in his cases) a very severe inflammation even to the degree of causing a

deformity. The cardinal signs of this affection are a sense of tenseness, redness with breaking down of the cartilage, and as a result a crippling of the ear. In some cases calcareous and osseous deposits follow the inflammation. Perichondritis may and often does follow other causes aside from

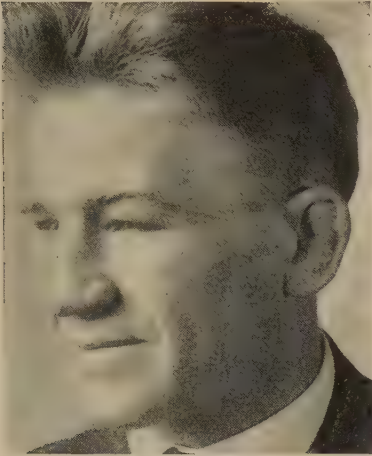


Fig. 224.—Chronic perichondritis. Prize fighter's "tin ear."



Fig. 225.—Chronic perichondritis. Roll or cauliflower ear following spontaneous hematoma opened and infected.

the mastoid operation. For example, a protracted eczema or dermatitis secondary to an otitis media suppurativa will involve the perichondrium which, however, has not the same tendency of breaking down. Associated

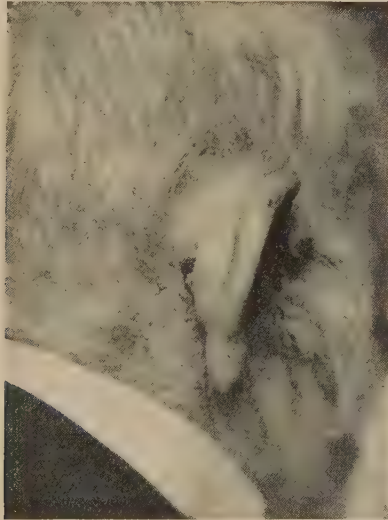


Fig. 226.—Chronic perichondritis with fistula following incision of a subperiosteal abscess.

with diabetes, particularly when boils are apt to form in the pinna or canal, we may also have perichondritis. In the common forms of furunculosis, such as frequently recur, the perichondrium is endangered. Practically in

every type of trauma of any severity the perichondrium is involved and inflamed. Syphilis of the auricle is likely to affect the perichondrium.

*Treatment.*—In the more acute forms a moist dressing of a diluted acetate of aluminum solution is most gratifying. The use of an electric bulb apparatus (500-watt lamp) for a period of ten minutes daily is of great value to relieve the pain. When the process undergoes coagulation necrosis or abscess formation, then, of course, opening and thorough removal of all the granulations is essential. As the acute process passes into a less active type, the use of ointments of ichthyol or albuminate of silver (Credé) is advised. A light dressing and bandage should be worn for some time to give support to the auricle and thus drooping or shriveling may be prevented. The chronically thickened auricle as a result of perichondritis is not much influenced by treatment by repeated use of above-mentioned lamp or medical diathermy, but gentle massage may assist somewhat. One example of this immobile type of auricle is the tin ear of a prize fighter.



Fig. 227.—Stricture of external auditory meatus.

**Strictures and closures of the external auditory canal** are usually due to a long-existing discharging middle ear, although their presence following the use of caustics and from traumatic causes must be taken into consideration. Their association in conditions of perichondritis and dermatitis due to chronic eczema has already been mentioned. The formation of strictures in connection with a chronic suppurative process

is very important on account of possible retention resulting. Strictures may be due to the thickening of the skin, but more frequently they are caused by the accumulation of connective tissue below the skin. Not infrequently the bone and cartilage are involved in the process. The canal may be strictured externally or deeper in, and at times such stricture may be almost complete. In the event of a normal middle-ear condition, the hearing may be not at all affected (Fig. 227).

The *treatment* of strictures depends on the etiology. All inflammatory conditions should be first controlled. Gradual dilatation by packing of gauze saturated with ichthyol in glycerine, gives the best results. In the event of stricture being of firm or bony consistency and not responding to this sort of treatment, surgical intervention may become necessary. In such cases, retro-auricular incision with the removal of a part of the posterior wall of the bony canal and a splitting of the strictured membranous canal, including the cartilage, is considered good surgical treatment. The after-treatment of long persistent packing until complete epidermization has resulted, is necessary.

**Impetigo contagiosa** is an acute contagious inflammatory affection characterized by the formation of discrete, superficial vesicles in blebs that become seropurulent and dry to form yellow crusts. It may occur on the ear *per se* or be associated with lesions in the scalp, nits in the hair, or asso-

ciated involvement of the face. Pustulation may occur at the onset of the condition. The disease is inoculable and auto-inoculable, and is largely seen in infancy and early childhood. It may occur in men by extension from an involvement of the bearded region of the face. It is due to the pus cocci—*Staphylococcus aureus* and *albus* and *Streptococcus*. It responds quickly to treatment as a rule, and treatment consists of destruction of the contagious elements in the crusts and contents of the lesions. The crusts should be removed by washing. If much inflammation is present, moist dressings of aluminum acetate should be applied and then the application of ammoniated mercury ointment in 2 to 5 per cent. strength.

**Syphilis Auriculæ.**—All forms of the disease have been observed and the clinical diagnosis as to history, blood, and other laboratory findings and lesions on other parts of the body are most important in the diagnosis. Sendzias<sup>27</sup> described 4 cases following a bite of one individual by the other, wherein primary chancre was present. Passow<sup>24</sup> reports one following kiss-



Fig. 228.—Lupus vulgaris.



Fig. 229.—Tuberculosis of external ear.

ing. The location of this chancre was the concha. Secondary and tertiary lesions of syphilitic character of the external ear differ in no way from such syphilitic skin lesions elsewhere and, therefore, will require the same type of local, as well as general, treatment. In some of the more destructive types wherein the perichondrium and cartilage suffer from loss, there will occur the deformities depending on the severity of the lesion. The opening to the external canal may become cicatrized and require surgical intervention or the replacement of the lost parts, either by plastic operation or artificial ear may also be required.

**Tuberculosis of the skin** is in reality lupus vulgaris. In most instances it attacks the ear from neighboring structures. When the ear is once attacked it becomes a very active and destructive process, especially if all the parts of the auricle become involved. The writer has seen a case wherein the skin alone (Fig. 228) remained involved and another in which all the tissues were destroyed in spite of all heroic efforts to stem the tide (Fig. 229). Roentgen ray and radium were not known at that period. A primary tuberculous nodule has been described by Henrich as a bluish-red hyper-

trophy about the lobule which never did ulcerate in contradistinction to lupus which has the characteristic apple-jelly tubercle breaking down, healing up by marked scar formation, while in advance another tubercle has formed and begun to break down. The diagnosis of lupus can further be made by microscopical section which will demonstrate the characteristic picture of tuberculosis.

The *treatment* has received quite an impetus through the aid of the various rays such as the Roentgen, radium, quartz, and carbon arc lamp, many cases of successful cures without marked scarring and deformity being reported. The writer has seen several such cases and cures. The case above illustrated had no such opportunity having been seen before Roentgen or Curie discovered their rays. While this patient lost the external ear, the process was brought to a standstill by the use of the acid nitrate of mercury. Operation for reconstruction of the ear was not considered wise. The external canal and middle ear remained unaffected. Recently the use of organic compounds of the heavy metals intravenously has been attended by good results. This is especially true of the gold salts.

**Chondrodermatitis nodularis helicus**<sup>32</sup> or **painful nodular growth of the ear**<sup>10</sup> is a small nodular growth which occurs on the rim of the ear. The condition although rare, at times bears a clinical resemblance to epithelioma, and must be differentiated from this. The condition occurs between the ages of thirty and sixty-five and is uniformly situated on the upper pole of the ear in the region of crown angle on or near the free border of the helix. The condition may be bilateral, but is usually on one side. They are immovable and firmly fixed to the cartilage. Pain is a characteristic feature in all cases and usually first attracts attention to the presence of the lesion. The pain is aggravated by pressure.

Histologically, there is found a chronic inflammatory process in the corium involving the cartilage and a circumscribed hypertrophy of the epidermis.

The *treatment* consists of the excision of the nodule, inclusive of the cartilage, and excision must include the entire lesion to prevent recurrence. Electrolysis, radiotherapy, fulguration, and galvanocautery may also be tried.

Recently in the writer's practice, such a condition was accidentally discovered. The patient was suffering from a carcinoma of the laryngoesophagus and a four plus Wassermann. He did not complain of this ear condition, and it was not treated or proved microscopically.

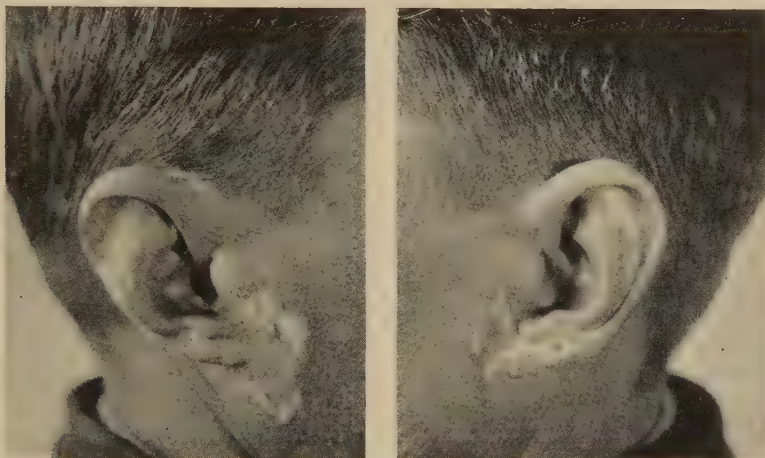
**Exostosis Auriculæ.**—These bone excrescences are most often seen in the vicinity of the annulus tympanicus and reaching into the middle ear. However, they do project externally. They appear as roundish or oblong bodies, and have been designated by Kaufman (Prague) as osteophytes. Exostoses have also been frequently recorded in connection with fistulous formation within the canal secondary to a chronic mastoiditis, especially in tuberculous processes.

**Hyperostosis of the External Auditory Canal.**—These may be described as multiple bone formations with broad base, located in the canal wall. These may be congenital or acquired. They may be so extensive as to block the entire canal. There are usually found other hyperostoses about other parts of the head. In the event that the canal is blocked the treat-

ment will necessarily be surgical, and the best way to attack them is with the burr.

The writer has had experience in 2 cases of this kind, one patient being a physician who has these hyperostoses in both ears. In one the patient developed an acute otitis media suppurativa which threatened complication. It became necessary to do a retro-auricular operative procedure to widen the canal.

**Lupus erythematosus** is essentially seen in early and middle adult life. Its relationship to tuberculosis is a much discussed question. Its presence about the ear is in association with the characteristic distribution over the bridge of the nose and cheeks—butterfly distribution—and it begins early as a patch of redness around the opening of a sebaceous gland which spreads, scale forming, the margin being well-defined and slightly raised. Centrally, the lesions show atrophic changes. The disease may be of the



Right ear.

Left ear.

Fig. 230.—Lupus erythematosus.

disseminated, nodular, or telangiectatic types. The course of the condition is chronic.

The *treatment* consists of constitutional measures of a supportive tonic type, elimination, avoidance of acid and sun and the restriction of alcohol and tobacco. Locally, the use of mild applications, *e. g.*, calamine, zinc oxide lotion, or a mild ointment of 2 to 4 per cent. salicylic acid and sulphur in cold cream for the scaly type, is desirable. Application of carbon-dioxide snow with moderate pressure for fifteen to fifty seconds is good treatment. Arsenic pastes, acid nitrate of mercury, or other caustics may be used in selected and more violent cases. Roentgen ray, radium, and carbon arc lamp are also valuable. Intravenous gold therapy as advocated in lupus vulgaris has been very successful.

The writer has observed one such case (Fig. 230). This patient had noted that practically simultaneously, the skin lesion appeared on the lobules of his ears and continued to advance or spread toward the face and neck. Roentgen therapy brought the condition to a standstill, but not without leaving a noticeable scarring about the lobules and the neighboring skin of the face.

**Noma of the External Ear.**—R. Hoffman<sup>15</sup> describes this condition and a case most minutely. The progress of the disease was that usually observed in noma.

**Malignant pustule about the external ear** has been described by Haug.<sup>13</sup> The anthrax bacillus was recovered in pure culture and the destruction of the external ear was almost complete.

**Variola of the external ear** has been observed as a transplant from a fresh vaccination of the arm. The history that such a fresh vaccination exists may be the only way to make a diagnosis because the lesion may appear as an ordinary pustule due to other causes.

**Herpes Zoster Oticus.**—Koerner<sup>19</sup> in 1896 described a disease of the auricle in which blisters formed along the distribution of a twig of the sensory nerve and in which there occurred facial paralysis and difficulty with the eighth nerve. In this country it was J. Ramsey Hunt<sup>18</sup> who first called attention to a symptom-complex about the ear and soon after wrote quite extensively with report of cases illustrating various phases of the disease. He brought out the analogy between herpes zoster of the intercostal and that associated with the geniculate ganglion inflammation with the disease. Soon after, case reports began to appear by other authors and the writer, himself, reported a very interesting case of this type. He brought out several new points in regard to the disease: first as to the causative factor being a tonsillar or peritonsillar infection; second, the vestibular symptoms were marked in the case with secondary involvement of the auditory portion. The facial was also involved in the upper two branches, a paralysis from which the patient never recovered. Also the deafness remained permanent. Recently Dennis<sup>8</sup> went into the subject of vestibular as well as cochlear tests in two of his cases, and in reviewing the literature brought out some interesting conclusions on the etiology and treatment. The probable cause is a bacterial infection as from the teeth, tonsils, or some other focus of infection involving the geniculate ganglion which corresponds to the posterior root ganglion of the spinal nerves.

*Treatment.*—As soon as possible remove the probable focus of infection. One will do well to err on the side of removing some suspected focus as for example, dead teeth, tonsils, eradication of infected sinuses, appendix, gall-bladder, in order to prevent the perpetual infection of the ganglion and other nerves. The herpes themselves are variable. Some of the blisters are very superficial but most of them are deeper and when they become confluent as they sometimes do actual necrotic areas with scar formation will result. Antiseptics and large quantities of ointments are applied, such as ammoniated mercury ointment 3 to 5 per cent. Internal administrations of salicylates are indicated, as is also any and all forms of elimination by skin, kidneys, bowels, etc. These patients require a very carefully selected, generous diet, and tonic treatment. For the paralysis the accepted treatment of electricity, massage, and strychnin should be carried out, but are usually of little benefit in themselves. The most important measure is to find the focus of infection.

**Bullæ of External Auditory Canal and Tympanic Membrane.**—While the tympanic membrane is described under another chapter and is considered in connection with the middle ear, there occur pathological changes about the external ear that extend on to the drum. In particular are bleeding and serous exudate with subsequent lime deposits. Hemorrhagic

blisters with apparently inflammatory origin are most frequently observed during influenza epidemics. These may be located anywhere within the canal or on the tympanic membrane, and diagnosis is very important when located on the drum, for if one mistakes them for a bulging of the ear drum and opens into this cavity, an infection will be carried into it. It has been claimed by several writers, particularly James Ramsey Hunt, that these blisters are a result of toxic involvement of the ganglia, both gasserian and geniculate. These blisters are due to an accumulation of serosanguineous fluid beneath the epithelium and they vary in size. Some of the larger ones may break spontaneously and the smaller ones may become absorbed. Ullman<sup>34</sup> has recently shown by his experimental work that the direct etiological factor was a filtrable virus. The *treatment* should be that of the primary influenza. Local heat and the topical application of metacresol acetate to the vesicated areas tend to allay the pain. Above all, incision is definitely contraindicated.

**Horny formation** on the external ear was observed by Bouvier<sup>3</sup> starting from a wart and becoming very large. In older people there are actual calcareous degenerations formed in the auricle as in the ribs and larynx, although the cartilage is not of the same type. The skin becomes loose and the cartilage takes on a bit of a slump, often rolling or sagging. Those changes produce certain definite malformations in the shape of the meatus and external auditory canal. Again the gouty deposits about the auricle should be mentioned, which, when inflamed, may become painful.

**Pearly Formations on the Tympanic Membrane.**—These occur at the site of a small perforation following either an incision or a spontaneous rupture. They are located in the superficial layers of the tympanic membrane and progress peripherally to lose themselves in the canal wall. If they, however, accumulate and form somewhat deeper, they remain and grow to a larger size, finally breaking, discharging and a scar remaining. Some authors consider them epithelial inclusions with small cyst formations occurring during the healing process of the perforation. This pearly formation is not at all common causes no symptoms and is discovered during the casual examination.

#### TRAUMATIC CONDITIONS

**Othematoma.**—Psychic and mental disturbances were formerly very prominently mentioned in othematoma, but most of these opinions have been relegated to the scrap-heap. The only thing that is believed is that these individuals, being difficult to manage, were often beaten by keepers of the asylums wherein these patients were confined, with the result that othematomas were produced. Especially was this true in Europe. In a report from one of the clinics there is an interesting statement to the effect that whenever a patient from the psychiatric department was referred to the otologic department with othematoma, the clinician was able to foretell from which ward the patient came and who the attendant was.

Blood alone gives a much better prognosis of recovery than if blood and serum are contained within the tumor mass, for more rapid absorption and less destruction has been noted when blood alone is present. Infection is also more likely in the presence of serous effusion. In the opening of such masses one may at times find pieces of cartilage floating in the sero-

sanguineous accumulations. As an end-result we may have either shriveling or stiffening (Fig. 231).

The most frequent etiological factor of othematoma is found in boxers (Fig. 231), and the writer believes a special reference will be of practical value. In that connection treatment of all types of othematoma is described.

**Pugilist's Ear.**—These may be othematomas or rhexis (blood-clot or tears). Following a blow, the ear begins to swell and the contour of the helix and anthelix are lost by the formation of the blood-clot. Men describe it as feeling "as big as a house." Unfortunately, since the fighter



Fig. 231.—Othematoma (stiffened ear).

must go on with his usual activities, the condition cannot have proper and immediate attention. The writer has in one case advised the second to apply a rubber bandage between the rounds and believes that this prevented the formation of a large hematoma.

**Treatment.**—Under strictest antiseptic precautions the skin is incised widely and the clot delivered. If there be an actual bleeding vessel, that should be either ligated or compressed by forceps. The skin is replaced very carefully, stitched, and the ear held in place by a modeling compound dressing, held by a not too firm bandage. The auricle is carefully surrounded by pads of cotton to avoid decubitus or pressure. This dressing should not be removed for forty-eight hours unless great pain or

elevation of temperature results. Unfortunately in most cases the hematoma is not interfered with or if interfered with, not enough care is taken in the asepsis and the perichondritis supervenes. This is invariably followed by a stiffness of the whole pinna (tin ear, Fig. 231) or it shrivels up and forms the characteristic pug or cauliflower ear. For the aforementioned conditions operations have been devised with negative results. Recently in one case of cauliflower ear the writer was fairly successful by implanting a thin sheath of ivory. What will follow a combat in case the ear is struck, remains to be seen. In case of rhexis or tears, which usually take place at the junction of the upper part of the auricle with the side of the head, the treatment is to put in an immediate stitch or two. A good second has been known to do that temporarily between the rounds, and after the combat the surgeon should do a clean job.

**Burns.**—All types and degrees of burns observed elsewhere have been met about the external ear. The lightest form (first degree) and often found about the auricle is that following the use of hot-water bags and other forms of hot applications. Almost everyone has seen these and at times one will even observe small blisters, especially about the helix, anthelix, and tragus, the most prominent parts of the ear. The more active processes even to complete destruction occur usually from being caught in flames (Fig. 232). Another form of burn is the electric one (Fig. 235).

Burns from chemicals, from carbolic and other acids, as well as alkalis (Fig. 233), are frequently reported in the literature. An interesting case of the like is that of a Russian who, during the conscription period of the



Fig. 232.—Burns by flames.



Fig. 233.—Partial loss and contracture of external ear and side of face following accidental application of 95 per cent. carbolic acid.

war, wished to evade service and poured pure carbolic acid into his ear causing a marked sloughing of the canal and destruction of the tympanic membrane (Fig. 234). He was treated with normal saline irrigations and



Fig. 234.—Intentional phenol burns of ear.



Fig. 235.—Electric burn.

bland ointments, recovering with fair epidermization and completely dry ear. The hearing remained very good considering the injury. Although cases of perforated ear drums were usually rejected, the writer, being on the advisory board, used his influence with the medical superior officer at the

camp and the man was compelled for moral reasons to serve as a stable-man for the duration of the war.

The *treatment* in the severe forms is first the relief of pain which is excruciating. Keep the patient under morphine hypodermically. In mild burns apply locally in abundance ichthyol in vaseline 10 per cent. and no bandage. In the severe form the old reliable linseed oil and lime-water mixture is still very good treatment, although the chances for secondary infection are greater. A favorite application of the writer has been a mixture of phenol 5 per cent., iodine 5 per cent. in vaseline 90 per cent. (the phenol acting as an anodyne and antiseptic, and the iodine as a mild antiseptic), the burned parts being kept well covered at all times. Recently tannic acid in 3-5 per cent. solution, topically applied has proved of great value.

Complications of the burns are, of course, perichondritis and abscess with more or less loss of tissue and a great deal of firm scar formation. These conditions will require their appropriate methods of treatment.



Fig. 236.—Roentgen-ray burn.

There is a form of burn which, perhaps, should not be called that but as corrected by an eminent radiologist, "a reaction" which is the expression for Roentgen-ray, radium-ray, and other ray burns. Since these various rays have come into the realm of therapeutics, especially for malignant diseases, wherein larger or heavier doses are required and for longer periods, than are needed for surface lesions or for diagnosis, there have been observed many so-called burns, though not often about the external ear. These vary the same as burns from any cause except that one scarcely ever finds the charring form as from flames. The first stage of the burn is often considered by radiologists as a good reaction, especially if they have

observed their rules of screening, type of rays, etc. The more advanced type of this so-called burn will show blisters and superficial sloughing of the skin, and if the effect is more severe, deeper destructions will be seen. These lesions are painful and of slow healing because the histological pathology has shown the blockage of the blood-supply in these regions thus affected. The writer had a case in which a part of the ear was affected and which followed the use of Roentgen-ray treatment for tuberculous glands of the neck, which shows to what degree the skin may be affected (Fig. 236). It must be stated that this patient was treated by a radiologist very early in the game and no such accidents are nowadays observed as a result of treatment of tuberculous glands of the neck.

The treatment for the ray burns is the same as that employed in burns from any other cause. The prognosis must be guarded as to time of healing which is much slower than in other burns. When the burns are deep they invariably require secondary corrections such as plastics.

The writer desires merely to call attention to sunburns and burns from

the carbon and mercury lamps that are now being used very extensively and are possible producers of burns. Treatment is the same as outlined above for burns.

**Frost-bite.**—More frequent than fingers, toes, or the tip of the nose is the frost-bite of the pinna. Three degrees of the condition may exist at the same time, although an advanced first stage is the rule, that is, an ischemia with subsequent edema.

*Symptoms.*—A numb feeling in sector fashion, usually the upper half, followed by a tingling sensation and pain characterizes this condition. The degree of the pain depends on the degree of the pathological process. The appearance of the pinna is at first pale, in fact, whitish in comparison with the neighboring skin. This is soon followed by a swelling which is in the nature of an edema. Should the process be one of greater severity in that there be blister formation, one will observe a great deal of reactionary inflammation of the pinna.

*Treatment.*—Preventive. If one's ears have been exposed previously to severe cold and become slightly bitten, extreme care should be taken to avoid subjecting them to a quicker and more severe second attack. It therefore behooves such an individual to wear ear-muffs or cap which comes down over the ears whenever the weather is cold. In fact it would be best if everyone in very severe cold weather would protect his ears, at least rub them at frequent intervals to stimulate the circulation in this peripheral blood-supplied organ. When the ear is once frost-bitten and this is usually called attention to by someone else who sees a distinctly blanched auricle, one must avoid warm or hot applications. On the contrary, the use of friction with snow, ice, or cold water is indicated. As soon as one feels that the blood is returning to the tissues, free application of an ichthyol ointment (10 per cent.) is indicated. Should there be an abrasion present, no matter how minute, this must have serious consideration because infection is likely to occur and result disastrously. The writer has seen a case that resulted in a marked perichondritis with loss of the shape of the auricle, simply because asepsis was ignored. A frost-bitten ear should be bandaged during the entire time of its activity and for some time after in order to avoid trauma which is so easily sustained in these cases.

**Injuries.**—A great variety of conditions may be described but since each case is a law unto itself, a clear-cut text is impossible in the consideration of generalities. The position of the ear exposes it very much to trauma. The illustrations of some of the cases coming under the writer's observation will hint at the multitude of possibilities. The types of lesion are classified as erosions, cuts, punctured wounds, tears, and partial or total losses. The major traumatic conditions as encountered during the war and subsequently for repair are still fresh in the minds of many surgeons who were in service. The term "débridement" coined by Le Maitre for the immediate correction by removing what was considered not recoverable, proved very successful in the rapid healing and prevention of greater deformity. The replacement of parts, even though they were hanging on only by a thin portion, was many times successful. Brown<sup>5</sup> tells of having replaced an ear that was entirely bitten off by a dog and says it healed on, while Holt<sup>17</sup> had a similar case except that the ear was bitten off by a horse. Courtad<sup>7</sup> had a case of an ear torn off during delivery by forceps. It has been claimed possible by some workers in this line to replace an external ear

entirely lost by trauma or surgery if the donor's ear is immediately removed after death following accident and at once transplanted on to the living. It is stated that such a transplant will adhere. However, not sufficient authenticity appears to accompany such reports (Boyer<sup>4</sup> and Detrochet<sup>9</sup>). In this connection it might be well stated that prosthesis, partial or complete, of the external ear should be the logical means of correction. It has, however, been the writer's as well as other men's experience that the patient does not take as kindly to this as to plastic reconstruction. The main



Fig. 237.—Traumatic ears.

From left to right:

Top row: 1, War injury from shrapnel, with loss of greater part of external ear as well as bony structure including facial paralysis. 2, Partial deficiency of external ear from sabre blow. 3, Pugilistic ear. 4, Partial destruction of right auricle and side of head, with resulting scars and deformity following shrapnel injury in late World War.

Middle row: 1, Part of external ear bitten off by dog. 2, Artificial ear prosthesis used in correction of deficiency, aluminum which is painted to correspond with skin. 3, Artificial ear held in place by spectacles. 4, External ear completely torn off in elevator accident.

Bottom row: 1, Severe automobile injury resulting in loss of greater portion of face and ear torn off. 2, Immediate replacement of torn parts. 3, Patient prior to injury.

reason for objection is the inconvenience the patient is put to in putting on and taking off the prosthesis and the marked difference in its appearance from the neighboring tissue, especially in change of temperature. Grove<sup>11</sup> employed vulcanite and Zundle<sup>33</sup> used celluloid, claiming great adaptability of these substances. Henning of Vienna has perfected material to make prostheses adhere, the ingredients of which are not stated in his report. The writer, in the few cases of prostheses of the ear which came under his

care, employed the services of his technician in this work (Mr. F. Maschmann) who used aluminum coated with a color-fast compound which is non-inflammable and indestructible. This has given fair success. Recently there has been developed by Klocke of Berlin,<sup>35</sup> a former technician in Passow's clinic, a prothesis material that is light, elastic, and possesses a "feel" that can hardly be distinguished from real tissue turgor. In addition it can be tinted to blend with the natural skin color and applied in such a manner that the artificial appearance that is seen in our former prothesis is entirely absent. This has solved our prothesis problem, and the patient can no longer complain of a crude makeshift that not infrequently was more disfiguring than the deformity which it was supposed to hide.

Gunshot wounds, wounds sustained by high explosives and shrapnel, as well as stones, etc., formed a large group of external ear traumas during the war. Their immediate restoration to as near normal as possible with not too accurate suturing but very thorough disinfection caused many of them to heal most kindly. Of course in all traumatic conditions the use of tetanus antitoxin (one or two injections) is absolutely indicated. Trautman<sup>9</sup> reported the loss of an auricle from gangrene by too tight a bandage about the head. While making repairs of the pinna one must always observe the contour and size of the meatus and canal.

In civil life and on a marked increase are the injuries of the external ear due to automobile accidents. Partial tears are the most common. The othematoma also occurs quite frequently and since there is usually an external opening present these cases stand a good chance of being infected and therefore suppurate.

The treatment is the same as employed in war injuries. The hematoma is quickly removed, parts thoroughly disinfected (mercuochrome is now very much favored as an application), the skin readapted and parts held in properly supported position by bandage (refer to treatment of hematoma of pug ear).

It may have to be redressed much earlier on account of the exposure to infection. The tetanus antitoxin must not be omitted. Complication of perichondritis and abscess are quite common and must be treated as such (refer to respective subjects). Strictures of the external auditory canal are likely to occur and must be guarded against.

**Retro-auricular Fistula.**—Following the earliest mastoid operations by the radical method for chronic suppuration as advised by Zaufal, retro-auricular fistulas or rather cavities were the rule for it was the principle of the operation to allow the epidermization of the cavity behind the ear. Following the improved or modified method in which the retro-auricular incision was immediately closed after the radical mastoid operation and the epidermization allowed to be complete through the splitting of the membranous canal, these large cavities or fistulas were comparatively rare. It has, however, occurred and still does in isolated cases that a retro-auricular opening remains which requires subsequent closure for cosmetic reasons if for no other.

In a recent case that the writer had under his care there was an exposure of the lateral sinus in the fistula, the sinus being thoroughly covered by an epidermal scar (Fig. 238). In this case the indication for closure of the fistula was for protection of the sinus as well as cosmetic reasons.

Of the various operations that we have employed we have found the following universally successful: An elliptical incision is made about the fistula to allow such flap formation as to permit them to meet in the median line when turned upon themselves, that is, the dermal layer is turned toward the exenterated mastoid cavity. Three separate catgut stitches hold them

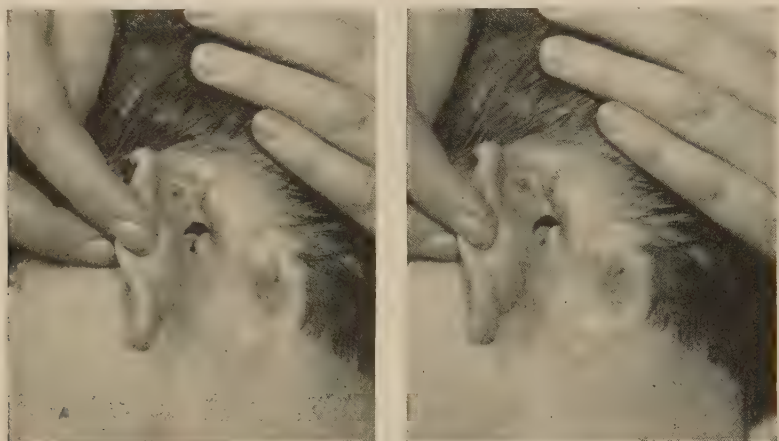


Fig. 238.—Stereoscopic photograph. Retro-auricular fistula with lateral sinus in view.

in place. A perpendicular incision  $1\frac{1}{2}$  inches in length is then made beyond the hair line down to the bone. By means of a sharp dissector this double pedicle flap is lifted off from the bone and so loosened as to enable one to move it forward to meet the dissected skin at the auricle where it is



Fig. 239.—Retro-auricular fistula, before and after operation, also showing hairline wound for closure.

stitched devoid of tension with horsehair. The created defect is packed with gauze to prevent this flap from slipping back while healing of the united retro-auricular suture line is taking place (Fig. 239). Thus a double-decker covering of the fistula is made, closing the fistula as well as overcoming the depression.

## NEOPLASMS

The most frequently met with neoplasm of the external ear is **epithelioma**. The favorite location for its starting point is the meatus and concha (Fig. 240). The destruction in its progress is quite rapid and marked. It has a tendency to invade the parotid and other glands (Fig. 241), and when it does so the condition becomes very serious, in fact, hopeless. At least surgically, it has never been the writer's fortune to see a case recover when these regions once become involved. He has seen several cases get well following the employment of radium and Roentgen rays. One very interesting case which came under his observation and was treated successfully by Dr. William A. Pusey with Roentgen rays, showed the entire auricle destroyed and bone exposed without any evidence of necrosis (perfectly dry) (Fig. 242). The writer has seen at least 2 cases of epithelioma of the squamous-cell type which were treated by the so-called arsenic paste specialists (quack institutions) that remained for years without recurrence. Another type of treatment recently employed by the writer in 1 case was the removal of the greater portion of the auricle by means of the electrocoagu-



Fig. 240.—Epithelioma of pinna and parotid gland.



Fig. 241.—Epithelioma of pinna and external auditory canal having a parotid salivary fistula, facial paralysis, adenopathy, and partial ankylosis of mandibular joint.

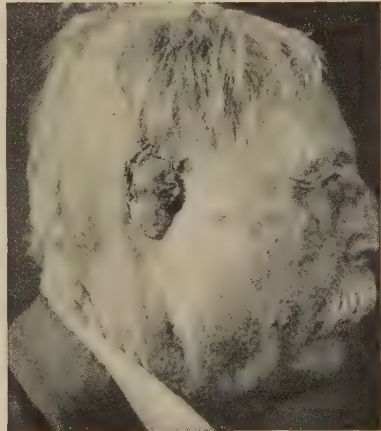


Fig. 242.—Total loss of pinna following use of radium for epithelioma. The squama, root of zygoma, and mastoid process exposed and dry (no evidence of bone necrosis or odor).

lation knife (surgical diathermy), but it is still too early to judge of its permanent cure. All of the cases that recover may have secondary plastic operations or prosthesis correction.

Epithelioma of the auricle is frequently of basal-cell type, and not very malignant unless invasion of contiguous structures has taken place. The exposure of the ear to the elements, the close application of the skin to the cartilage with little subcutaneous tissue and the thin dry skin being a frequent site of seborrheic patches, predispose to epithelioma.

Epitheliomas of the ear are usually painful. Roentgen ray and radium are therapeutic measures of choice, radium being probably the better. The use of caustics in selected cases is a serviceable measure, and the application may be preceded by use of the curette. Acid nitrate of mercury and trichloroacetic acid are valuable agents. Last, but most important, is the use of surgical diathermy in the eradication of the lesion.

**Endothelioma.**—This is usually of the perivascular type and a much slower growth, and one less likely to break down or invade the glands of the neck. However, several cases are recorded which started in the parotid gland and secondarily involved the auricle.



Fig. 243.—Neuroma.



Fig. 244.—Chondroma of pinna and external auditory canal.

*Treatment* consists in the use of surgery, radiotherapy, and especially surgical diathermy.

**Sarcoma.**—Ruttin<sup>26</sup> and Herzfeld<sup>16</sup> report on this condition, having found the round-cell variety, whereas Bezold,<sup>2</sup> Szenes,<sup>29</sup> and Sugar<sup>28</sup> found sarcomas of melanotic and other varieties of the external ear. They are comparatively rare and the microscope only can make the positive diagnosis. Roentgen rays, radium, and surgery are the methods of treatment to be employed with special emphasis on surgical diathermy.

**Lipoma** and **neuroma** are two other rare manifestations about the ear and have been observed by Lannois,<sup>21</sup> Gruber,<sup>12</sup> and Ole Bule.<sup>6</sup> The writer has had the fortune of observing a case of each (Fig. 243). In the case of lipoma, patient refused interference. The neuroma was of considerable interest particularly from the standpoint of recurrence and

excruciating pain. The patient was operated on eleven times, the neuroma always recurring in spite of most radical excisions and Roentgen-ray and radium treatment. The final relief from pain was only obtained after posterior root resection of the gasserian ganglion following which the tumor stopped recurring.

**Chondromas** of the auricle are not supposed to exist, but those of the canal have been described by Politzer<sup>25</sup> and Konietzko<sup>20</sup> which were, however, ecchondroses. There is frequently found after a severe attack of perichondritis or abscess of the auricle, deposits of calcareous material even resembling bone (pseudo-osteoma) and some of these have been mistaken for chondroma. Also in some gouty patients, deposits have formed which, when not inflamed, may be mistaken for chondromatous formations.

In view of the rarity of and the writer's observation of a genuine case of chondroma of the auricle, it is herewith recorded: This was (Fig. 244) a very slow growing tumor (nineteen years' duration) giving no other symptom than slight tightness, but objected to from the standpoint of its appearance. It felt harder to touch than the rest of the cartilage of the auricle, and appeared to be intimately connected with it. In the removal it was found to be part and parcel of the postero-lower portion of the pinna (helix). The skin was more adherent to the cartilage than elsewhere. The growth was sliced off from the cartilage of the pinna and the result was cosmetically good, there being no recurrence now after seven and one-half years. Microscopically, it proved to be an elastic fibrocartilage with very little activity of growth.

**Fibromas** are connective-tissue new growths that may be sessile or pedunculated, soft or firm, of different shapes, and non-painful, situated beneath and in the skin.

Unless the tumor blocks the external canal it will produce no symptoms, and is only of cosmetic interest.

The *treatment* consists in the surgical removal of the tumor which offers very little difficulty since it may be performed under local infiltration anesthesia. Only in the multiple form of fibromas or the painful forms of neurofibroma is the case complicated in the treatment. In the former there may result a deformity of the auricle, and in the latter there is a great liability to recurrence.

**Osteomas.**—These are usually bony changes in a fibrous or cartilaginous tumor. Kretzschmann reports a true osteoma of the external auditory canal which grew to such an extent as to distend the canal considerably. He considered this tumor to have developed from the periosteum of the mastoid and to have grown inward into the canal. In a discussion it was considered a calcified fibroma.

**Papillomas** are not so frequent about the external ear as are warts. In older individuals they should be considered as possible precancerous or pre-epitheliomatous conditions. They should be removed by operative methods, Roentgen ray, radium application, or surgical diathermy. The use of carbon-dioxide snow as first recommended by William Allen Pusey has been very successful in permanently removing a retro-auricular wart or congenital verruca vulgaris (Fig. 245) in 1 case in the writer's experience. Caustics or fulguration may also be employed.

**Paraffinoma** belongs to the neoplasms of foreign body irritation and usually results from the injection of paraffine for cosmetic purposes. This

is much more frequent in the region of the nose than anywhere else, but its occurrence is much more rare than formerly since paraffine is now rarely employed by the regular medical profession.

It is well known that in certain tissues or individuals this growth is more liable to develop as in the case of keloid. The first sign of its development is enlargement with increased vascularization of the overlying skin. Pain and ache are constant accompaniments. In regard to cases where the paraffine has been used for the purpose of stiffening a collapsed ear following perichondritis or some congenital malformation, this condition may result. In the writer's experience one such foreign-body growth developed which may have been caused by either the paraffine contained in the mixture or the bismuth subnitrate. Following a simple mastoid operation the cavity, in the after treatment, was filled with bismuth paste No. 2 (bismuth subnitrate 10 per cent., paraffine 10 per cent., vaseline 80 per cent.).



Fig. 245.—Congenital verruca vulgaris.



Fig. 246.—Paraffinoma of pinna.

Several weeks after the mastoid cavity was healed there was seen a roundish sessile growth behind the auricle which appeared to be growing quite noticeably. It was deemed best to remove it, and on examination the tissues showed microscopically the characteristics of both a bismuthoma and paraffinoma (Figs. 246, 247).

The *treatment* of paraffinoma is excision including the overlying skin unless the skin be absolutely free and movable. Since the tumor is rarely encapsulated the removal is incomplete, but regrowth is not very frequently observed.

**Keloid.**—In former times when piercing of the lobule and wearing of earrings was the custom, keloid was observed at the site of the puncture, especially in the colored girl. More rare now, but in individuals subject to keloid formation we find keloid scars in and about the auricle (retroauricularly) following mastoid operations.

The typical elevated scar with increased vascularization, always recurring after surgical removal no matter how carefully the edges are adapted, is diagnostic of this condition.

The cause is not known nor has there been any research work done along this line to throw the least light on the various theories advanced.

*Treatment.*—The Roentgen ray and radium applications have given the best results. In one very extensive case following mastoid operation with jugular vein ligation, the writer operated twice for the scar formation without result, then employed radium (50 mg. with 2 mm. of aluminum filter for three hours at different points of the scar, using same period and dose for each) for six weeks. The usual reaction followed which was again treated by local applications of ung. zinci. After all reaction from the radium application disappeared and the scar flattened, it was excised and the wound edges very carefully adapted and sutured subcuticularly with the happy result of non-recurrence of the keloid.

*Cysts.*—The most frequent location is in the lobule, although the writer has had 2 cases located on the posterior surface of the auricle, one of which took up the greater portion of the pinna.

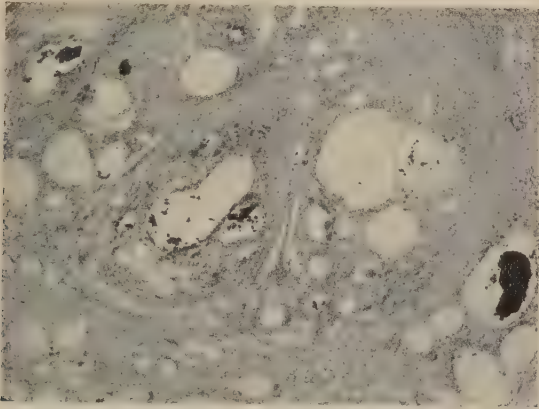


Fig. 247.—Low-power photomicrograph of bismuthoma and paraffinoma.

Cysts of the lobule are small, tense, accumulations of sebaceous material producing very few, if any symptoms. They are irritating to the patient, and become more aggravated by the habit of constant squeezing. In so doing the patient does at times succeed in expressing this sebaceous material which, however, very quickly reaccumulates and even increases in amount. This, of course, is due to the presence of the lining membrane of the cyst, which contrary to the type found in the larger cyst of the posterior surface of the pinna is very difficult to destroy owing to the fact that one cannot dissect it out entirely as may be done in the form found on the pinna. There is, therefore, greater liability to recurrence.

The *treatment* advocated is to open such a cyst by means of a crucial incision, curetting thoroughly the lining membrane. The cavity is packed with gauze for twenty-four hours. Then, by means of an electro-coagulation point, the eventual remains of the membrane are destroyed. The healing is uneventful as a rule and only a small dimple may remain to be seen.

**Dermoid cysts of the auricle** like other teratomas have been reported in

literature very rarely and never to the extent as found in other parts of the body. Hoover reports a case of a cyst of the auricle extending into the neck that was diagnosed as an upper branchial cyst, but proved to be a true dermoid of this region. Hair and epithelial masses were found within the cyst fluid.

#### CONGENITAL DEFORMITIES AND DEFICIENCIES OR MALFORMATIONS

(These will be best described in common with the external auditory canal.)

**Etiology.**—There is nothing definitely known but consanguinity of marriage appears to play a rôle. Mischel<sup>23</sup> states his belief that the position in utero has something to do with the cause of these malformations. This conclusion is reached because most of the malformations occur on the right side.

The variety of defect or deformity will (Fig. 248) range from a closure by a diaphragm of the canal or a small tubercle (Darwin's) on the pinna



Fig. 248.—Number of congenital deformities of external ear.

to complete absence of the auricle and a complete obliteration of the canal. One will also observe cases especially wherein on one side there is a deficiency and on the opposite side there are small projections known as supernumerary ears—polyotia. The ear as well as the canal may be extremely small (microtia—Fig. 249) or extremely large (macrotia—Fig. 250). They may roll or stand out beyond the usual confines from the side of the head. There may be a split in the lobule, the lobule in some cases may not be present and is seen to fuse with the side of the face. In regard to the small congenital external auditory meatus and canal, one must con-

sider the formation of the glenoid fossæ with the condyloid process influencing the same, which has nothing to do with any malformation of the ear. In all such cases it is very difficult at times to inspect the tympanic membrane, particularly since retention of wax and secretions are likely to be associated with these conditions. It must also be remembered that



Fig. 249.—Microtia.

in these congenital deformities of the external ear, the remainder of the ear is found affected, particularly the internal ear. The writer has demonstrated by carefully carried out labyrinthian tests that that is true, as is also shown by roentgenological studies of the absence of the internal ears and mastoid cells.



Fig. 250.—Congenital macrotia and macrocephalia. Front and back views

**Treatment.**—Surgical correction is the only possibility. The great difficulty is to obtain a good cosmetic result when the external ear is more than two-thirds absent. Physiologically, there is very little to be obtained in most of these cases, that is, for hearing. However, if determined beforehand by hearing tests as well as the presence of a middle ear

by roentgenograms, it may be practical to attempt to find the external auditory canal and tympanic membrane.

The writer has made another important observation of these individuals and their parents; namely, the improved mental state of all when correction is attempted.



Fig. 251.—Case of congenital deformity, showing steps of plastic reconstruction.

Surgery however has not proved equal to the task. The long, tedious, multiple plastic procedures do not produce results that are esthetically acceptable, and only serve to substitute monstrosity for deformity (Fig. 251). With the advent of the newer prosthetic aids as elaborated by Klocke, the writer believes that the solution of the problem, for the present at least,

lies in the direction of education and encouragement toward accepting the prothesis.

#### VASCULAR PATHOLOGICAL CONDITIONS

Telangiectases, varices, ecchymoses, nevi, and pseudo-angiomata are of rare occurrence about the external ear, and there is usually involvement of the neighboring structures. Warnecke<sup>30</sup> described **varices** of the external ear in connection with pregnancy. These occur both on the pinna and in the canal. They always disappear after childbirth.

**Nevi** about the ear are usually found either in the tragus or lobule and have the same characteristics as nevi in any other part of the body. The writer had experience with a very extensive nevus extending over the half side of the face (Fig. 252), including the pinna and the anterior wall of the canal. The result from treatment was so interesting that it may not be amiss to describe it briefly. Wishing to determine the value of the various methods of treatment in nevus the entire lesion was divided into definite areas for specific treatment and results observed. The forehead



Fig. 252.—Rapidly developing nevus.

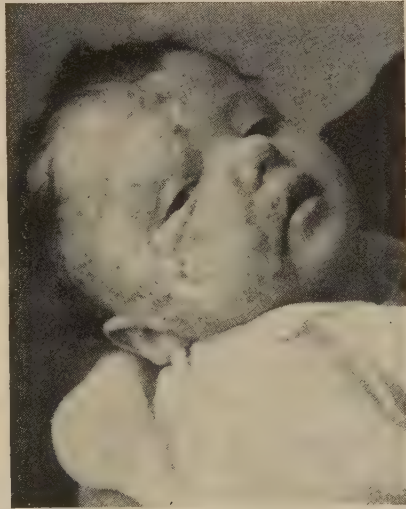


Fig. 253.—Results following treatment of nevus by various methods.

and temple were treated with carbon-dioxide snow (William A. Pusey), cheek with boiling-water injection (Wyeth), the eyelids with radium, the lips and chin with Roentgen ray, and the region of the ear and parotid with subcuticular ligation (Carl Beck). Altogether the result as a whole was most gratifying (Fig. 253), although the reactions from treatments are at times very stormy, particularly as to bleeding. The best results with least amount of scarring were obtained from the radium and next, the subcuticular ligation.

**Ecchymosis** of the external ear has been observed in connection with ecchymoses occurring in other parts of the body, in such constitutional diseases as leukemias and anemias. Also slight trauma produced, for example, by puncturing with the hypodermic needle, a slap on the side of the head, or in obtaining blood for a count, may cause ecchymoses.

There is a slight thickening and elevation of the epidermis. The course is usually uneventful, absorption taking place and leaving a slight discoloration.

**Telangiectases** are described as rare occurrences and have been observed following Roentgen-ray treatment. The recommended treatment for telangiectases is electrolysis which, however, gives very unsatisfactory results.

**Pseudo-angioma** in reality is a misnomer for this condition should be classified among the neoplasms. It is, however, a definite nodular condition of bluish character, and the histological examination shows a true angioma.

**Elephantiasis of the Auricle.**—This condition is due to a blockage of the lymph-vessels leaving the auricle. It has been most frequently observed in the wake of some very acute process such as recurrent attacks of erysipelas. Ruttin reported before the Austrian Otologic Society a case of a severe type following the vigorous pulling of the auricle, no doubt tearing some of the larger lymphatic vessels without any external wound having been sustained and without chance for escape of the lymph. The feeling to touch of such condition is somewhat soft and therefore must not be mistaken for a new growth as lymphangioma, which is also a rare condition about the pinna.

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## THE MEMBRANA TYMPANI

The tympanic membrane or drum-head forms a complete partition between the external auditory canal and the tympanic cavity, and is fully developed at birth. It is irregularly ovoid in shape, and measures from 9.5 to 10 mm. in its longest diameter which extends from the postero-superior to the antero-inferior part of the periphery and from 8.5 to 9 mm. in its shortest diameter. Its thickness is about 0.10 mm. except at its periphery where it increases as it merges into the annulus fibrosis which occupies the sulcus of the tympanic ring—annulus tympanicus. The ring is absent anteriorly and superiorly, and at this place the membrana flaccida of the drum-head is attached directly to the margotympanicus of the temporal bone and to the lateral ligament of the malleus.

The drum-head is made up of three layers. The outer or cutaneous layer is continuous with the skin of the external auditory canal and consists of two or three layers of cells and a delicate layer of connective tissue without definite corium or papillæ. The middle or fibrous layer, lamina propria, is composed of an outer stratum of radiating and an inner layer of circular fibers. The lamina propria is practically absent in the membrana flaccida. The inner or mucous membrane layer is continuous with the mucous membrane of the tympanic cavity and consists of large, low, non-ciliated epithelial cells with a delicate layer of connective tissue. Embedded in the drum-head is the handle of the malleus.

The **functions** of the drum-head are the collection and transmission of sound-waves to the ossicular chain and the protection of the tympanic cavity.

On **inspection** its obliquity to the external canal is conspicuous, the antero-inferior canal wall being longer than the postero-superior. It forms an angle of about 27 degrees with the anterior wall and a very obtuse angle with postero-superior. In general, it is concave, with the greatest depression at its middle, the umbo.

Its **color** may be described as pearly gray, but it is modified by the color of the light used and by the reflection from the inner wall and contents of the tympanic cavity.

A broad grayish-white line extending from above and in front downward and backward to the umbo marks the malleus handle or manubrium. At its upper end is a nipple-like projection produced by the short process of the malleus while the lower extremity is broadened. Extending downward and forward from the umbo is seen a conspicuous shiny, triangular area called the light reflex, triangle of light, or cone of light. Its apex is at the umbo while its base is at or near the periphery. The brilliancy of this area is due to the concavity of the drum-head and to the direct reflection of the light from this portion of the membrane. Extending anteriorly and pos-

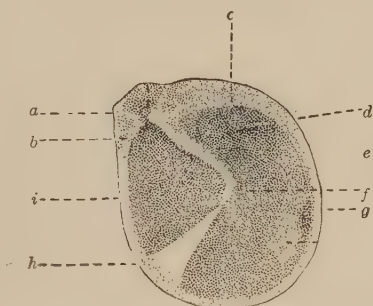


Fig. 254.—Normal drum-head showing light triangle, malleus handle, and folds about the short process: *a*, Short process of malleus; *b*, anterior fold; *c*, posterior fold; *d*, incus shank; *e*, malleus handle; *f*, umbo; *g*, round window; *h*, light triangle; *i*, annulus. (American Text-book of Diseases of the Eye, Ear, Nose and Throat.)

teriorly from the short process may be seen the anterior and posterior tympanic folds produced by slight bulging of the membrane. The posterior is longer and usually the more conspicuous of the two.

Approximately parallel with and posterior to the malleus handle can be seen a broad grayish line which marks the long process of the incus showing through the drum-head, while farther back the stapes, the tendon of the stapedius muscle, and the niche of the round window are often visible. Occasionally the chorda tympani nerve can be seen crossing the cavity just below the posterior tympanic fold.

For convenience of description, it is customary to divide the drum-head into four parts by two imaginary lines. One of these is an extension from the malleus handle to the periphery, while the other crosses this at right angles at the umbo.

The portion of the drum-head above the short process of the malleus is called the *membrana flaccida* or Shrapnell's membrane. It is thinner, contains very little connective tissue and is more flaccid than the remaining and larger part of the drum-head named the *membrana tensa*. It forms the outer wall of Prussak's space, and is bounded laterally by two straight striæ which extend from the short process of the malleus to the anterior and posterior ends of the tympanic ring.

The inspection of the drum-head is incomplete without the use of the pneumatic speculum, the magnification of which is very valuable. By alternately compressing and releasing the bulb of this instrument the mobility of the drum-head can be studied. Normally, the whole drum-head moves quite freely. The mobility is increased by atrophy or the formation of large cicatrices and decreased or lost through thickening of the drum-head, by adhesion of the membrane or malleus to the tympanic wall, or by ankylosis of the malleo-incudal joint.

**Pathological Changes.**—The drum-head may assume the pink color of congestion or the redness of acute inflammation. As the largest vessels lie in the manubrial plexus which follows the malleus handle, this area is most frequently reddened. It is well to remember that the redness as well as the lustre of an acutely inflamed drum-head may be obscured by exfoliating epithelium and that marked thickening may obscure all landmarks. Circumscribed redness may be due to granulation tissue or to inflamed mucous membrane of the tympanum seen through a perforation. Congestion of the promontory may show as a pink area in the corresponding portion of the drum-head.

The drum-head may be abnormally light in color due to fibrous thickening or even chalky white in certain areas due to calcareous deposits. The latter are more sharply circumscribed than the former, and both increase the opacity of the membrane. Large scars and atrophic areas usually are dark in color, thin, transparent and, unless adherent, more mobile than the normal drum-head. Large cicatrices are usually sharply circumscribed, and sometimes show light reflexes. Occasionally a dark hair-like line may be seen passing more or less horizontally across the drum-head caused by fluid within the tympanum. This line marks the level of the fluid and changes with its quantity and position.

**Change in Position.**—The normal concavity of the drum-head may be increased by absorption of the air within a closed tympanum producing a partial vacuum. This retraction or depression is recognized by a decrease

in the size and brilliancy of the light reflex, an increase in the prominence of the short process of the malleus and of the anterior and especially the posterior tympanic folds, while the handle of the malleus is foreshortened and more nearly horizontal than normal as it is drawn inward, backward, and upward. If the drum-head has not lost its normal transparency this retraction increases the visibility of the tympanic contents. Bulging of the drum-head may be general or localized, and is commonly due to pressure of fluid or air from within. If the bulging includes the antero-inferior quadrant, the normal light reflex will be decreased or lost. Large scars or atrophic areas frequently cause circumscribed depressions or bulgings.

**Loss of substance** may vary from a **perforation**, which is invisible to the naked eye, to complete destruction of the drum-head. Perforations may be circular, oval, elliptical, kidney- or heart-shaped. Usually they occur singly, but occasionally two or more are present. Multiple perforations suggest tuberculosis or diphtheria. Perforations may occur in either the flaccid or vibrating portion of the drum-head or both. The claim that perforations in Shrapnell's membrane are sometimes congenital is not borne out by embryology. The margins of recent perforations are red, while those of old dry ones are white and scarred. Those due to recent traumatism are usually linear and marked by fresh or dried blood; later, if persistent, they may become oval.

The *diagnosis* of a moderately large perforation can usually be made by inspection alone as its margins and the interior of the tympanic cavity can be seen. If, however, it is tiny or includes all but the rim of the drum-head with loss of the malleus, it is more difficult. In the former, one may see a drop of fluid coming through it, while in the latter the introduction of a small hook, which impinges on the margin, will establish the diagnosis. The diagnosis may be confirmed by inflating the middle ear thereby producing a perforation whistle, an increase in the discharge in the external auditory canal, or the appearance of air bubbles in this discharge or in a fluid which has been instilled into the canal for the purpose. Conversely the appearance of fluid in the pharynx after syringing the ear proves that a perforation is present. As there are no mucous glands in the external auditory canal, the presence of mucus in the discharge shows that it has come from the tympanum. A pulsating light reflex in the inner end of the canal indicates a perforation of the drum-head.

*Etiology.*—Perforations are usually due to incision or rupture in purulent otitis media. Occasionally they are caused by direct or indirect violence such as a blow on the ear, surf-bathing, fracture of the skull, the condensation of the air in the canal from a heavy explosion, caustics introduced into the canal, or the penetration of a foreign body. Inflation of the tympanum, when the drum-head is atrophic or the seat of a large scar, rarely causes a rupture.

*Symptoms of traumatic rupture* include a sharp pain of short duration, deafness, tinnitus, and occasionally vertigo. Its diagnosis is established by the history and by the presence of blood covering the margins of the perforation. Usually the bleeding is slight except in fracture of the base of the skull. Unless the ear is infected or the labyrinth is involved, the prognosis is good and the perforation generally heals within a few days, with normal hearing.

*Treatment* in early uncomplicated cases should be limited to protection

by plugging the meatus with cotton as attempts at sterilization may introduce infection. If infection occurs it should be treated as an acute myringitis or otitis media. If an uninfected rupture does not heal it should be covered with a sterile disk of wet tissue-paper as this acts as a support and favors closure. The closure of a dry perforation, resulting from purulent otitis media, may be facilitated in the same way, although it is usually necessary to destroy the epithelium lining the margins of an old perforation by scarification or by the application of a caustic such as trichloroacetic acid before applying the paper. Before attempting to close a chronic perforation it is best to apply the paper and retest the hearing which may be better with the perforation open. If the perforation refuses to heal a disk of paper or cotton may be useful in improving the hearing by acting as an artificial drum-head.

### DISEASES OF THE DRUM-HEAD

**Hyperemia** frequently occurs as a part of congestion of the tympanum or canal after inflation of the middle ear, after manipulation of the canal or massage. The large vessels along the hammer handle are usually most conspicuous.

**Inflammation** of the drum-head, myringitis, is usually secondary to otitis media or otitis externa. Only the comparatively rare primary type will be considered in this chapter. It may be acute or chronic.

**Acute primary myringitis** occurs from traumatism such as the removal of cerumen or foreign bodies or the introduction of irritants. The symptoms vary with the degree of involvement and include slight or severe pain, throbbing, and slight deafness. Ecchymoses, vesicles, or even abscesses may develop in the dermal layer. At first the drum-head is red, but the redness may disappear as the epithelium becomes macerated and exfoliated. Marked swelling obliterates the normal landmarks. A slight serous or bloody discharge may appear in the canal. The prognosis is good and the condition usually lasts but a few days. The *treatment* consists of hot applications, protection, cleansing with unirritating antiseptics, and insufflation of powdered boric acid. Abscesses should be incised.

**Chronic primary myringitis** is very rare, but may occur from neglect of the acute form. The outer layer of the drum-head is macerated, reddened, and sometimes partly covered with granulations. Usually there is a sense of fulness, little or no pain, slight deafness, and a small amount of fetid purulent discharge. The *treatment* consists of cleansing and the application of antiseptics such as boric acid or aristol powder, 5 per cent. solution of boric acid in alcohol, or 10 per cent. solution of nitrate of silver.

Occasionally the eruptions of the exanthemata and syphilis occur on the drum-head. Condylomata have been reported. New-growths such as warts, vascular tumors, moles, tuberculous ulcerations, miliary tubercles, and hematoma occur.

**Myringotomy** or incision of the drum-head is usually performed to secure drainage in purulent otitis media and occasionally for ossiculectomy. The field of operation should be sterilized and either a local or general anesthetic may be used, the latter being more reliable. A mixture of equal parts of the crystals of cocaine, menthol, and carbolic acid is one of the most efficient local anesthetics. If the drum-head is bulging the incision should be made through the most prominent part. If not, then the incision should

be made midway between the handle of the malleus and the posterior periphery of the drum-head, beginning near the floor and cutting upward so as to include one quarter to one half of its diameter. It should be free and follow the curve of the posterior periphery. If the mastoid is involved it should be extended into the posterosuperior wall of the canal. The point of the knife must not be introduced deep enough to injure the tympanic contents. In those rare cases in which a blue color at the lower part of the drum-head suggests that the bulb of the jugular encroaches on the tympanum, it is safer to make a horizontal incision above the danger line.

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## DISEASES OF THE MIDDLE EAR

### ACUTE MIDDLE EAR-CATARRH

(*Otitis Media Acuta Catarrhalis*)

**Etiology.**—This condition in most instances is caused by the spread of a catarrhal condition in the nose or nasopharynx up the auditory or eustachian tube into the ear. Any inflammatory condition in the nasopharynx predisposes to middle ear catarrh. The presence of a hypertrophied pharyngeal tonsil or adenoid vegetations is by far the most important predisposing cause. These growths by their presence press laterally on the auditory tube, and interfere with the ventilation of the middle ear. At the same time they are generally in a state of chronic catarrh, and catarrhal processes spread with great readiness along a mucous membrane. Youth may even be said to be a predisposing factor, as in children adenoids are generally present and the auditory tube is much wider than in the adult. Children also are more prone to catarrhal affections in general than adults. Anything which interferes with free movements of the palate and the opening and shutting of the tubes during swallowing is likely to set up a catarrh of the middle ear. Nasal obstruction from various causes, such as hypertrophied turbinates, deviated septum, as well as infective conditions such as suppuration in one or more of the accessory nasal sinuses are fertile sources of ear catarrh. In a number of cases catarrh of the middle ear is set up by a cold wind blowing directly into the ear, and it sometimes follows a long motor drive in cold weather.

**Pathology.**—The pathological changes which take place in catarrh of the middle ear are essentially similar to those in catarrh of the nose. There is first a hyperemia and swelling of the mucous membrane, and this is followed by the secretion of an exudate which is generally thin and watery at first. The tympanic cavity becomes filled partially or completely with this fluid, and as the eustachian tube is also swollen and congested, leakage of the fluid into the throat is not possible. After a day or two the inflammation may subside somewhat, the eustachian tube becomes patent, and the fluid leaks out or is absorbed. In other cases, however, the inflammation continues and leukocytes and epithelial cells pass into the exudate, which at the same time becomes thicker from secretion of mucus from the tube, which is plentifully supplied with racemose mucous glands. The condition may persist and pass imperceptibly into the subacute and chronic forms.

On the other hand the inflammation may become more severe and develop into an acute middle-ear suppuration.

**Symptoms.**—During an acute coryza the patient usually experiences a full feeling in the ear which may go on to actual pain. The fulness in the ear is sometimes relieved at first by swallowing or yawning, but soon this is no longer possible. There are usually at the same time crackling sensations in the ear and some buzzing or pounding noises. There is a variable degree of deafness, and the patient's own voice sounds abnormally loud in the affected ear.

**Otoscopic Appearances.**—The ear-drum may have the normal color and transparency, but the handle of the malleus, which is normally white, is bright red from dilated vessels running down it. In a rather more advanced case the whole membrane may be slightly pink and not quite as glossy as usual. A little later when some of the first inflammation has subsided the appearances may be almost normal. This is the case when the tympanic cavity is full of clear serous fluid. There may be a slight bulging of the posterior half of the drum-head. When the tympanic cavity is only partly full of clear fluid its upper edge may be seen through the drum-head as a thin line (Fig. 255). More commonly air bells are seen shining through (Fig. 256). The use of alternate suction and pressure through a Siegle spec-

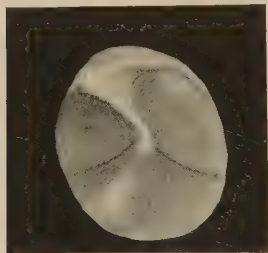


Fig. 255.—Appearance of the drum-head in acute middle-ear catarrh. The curved dark line represents the upper edge of the fluid in the tympanum.

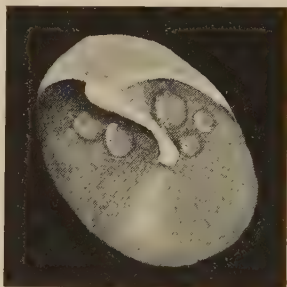


Fig. 256.—Air bells shining through the drum-head indicating fluid in the tympanum.

ulum (Fig. 257) makes them move and confirms their presence. When it is suspected that the tympanic cavity is full of clear fluid, inflation through the eustachian tube either by Valsalva's or Politzer's method or with a catheter will cause air bells to appear through the membrane and make it certain that there is fluid present. In those cases in which the exudate has a tendency to be mucopurulent the membrane is, as a rule, somewhat opaque and has lost its light reflex. Frequently radiating vessels are seen running across the drum (Fig. 258). This condition merges into subacute middle-ear catarrh (*q. v.*). When the catarrh is more intense it becomes indistinguishable from acute middle-ear suppuration.

As regards functional disability, when there is merely congestion in the ear, the hearing is little affected. Similarly if the ear is only partly filled with exudate the hearing may be almost normal. However, when the exudate fills the tympanic cavity there is considerable deafness. The deafness is of the sound-conduction variety, *i. e.*, the lower tones are lost but the medium and higher tones are heard. Rinné's test is negative and

Weber's test refers the sound to the affected side. Bone conduction is intensified.

**Treatment.**—Rest in the house or in bed is indicated in the early stages. A good purge is advisable as well as a large dose of aspirin or Dover's powder with hot lemonade or toddy to induce perspiration. Heat applied to the side of the head by a hot-water bag alleviates the discomfort. After the redness has gone from the drum inflation will open the tube and allow some of the fluid to escape. Drops of 10 per cent. solution of argyrol dropped into the nose while the patient is in the recumbent position with the head thrown back will help by reducing the congestion round the pharyngeal end of the tube. If in a few days the exudate has not escaped



Fig. 257.—Siegle's speculum.

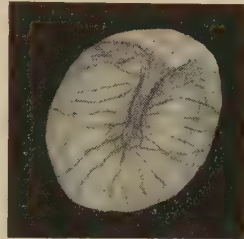


Fig. 258.—Acute middle-ear catarrh. Dilated vessels are seen running across the drum-head, and the membrane has lost its gloss.

or been absorbed it is advisable to do a myringotomy under careful aseptic precautions. Then by a combination of inflation *per tubam* and suction with the Siegle speculum the exudate, which is generally clear yellow, may be completely removed. Sterile gauze is lightly packed into the meatus and changed daily, and the wound heals in a few days or a week with complete restoration of function.

To prevent recurrences any adenoid vegetations which may be present should be removed.

### SUBACUTE MIDDLE-EAR CATARRH

(*Otitis Media Subacuta*)

**Etiology and Pathology.**—This condition generally follows acute middle-ear catarrh and is brought about by the same causes, namely, an inflammatory condition in the nasopharynx. The exudate which may at first be serous or watery becomes inspissated or mucoid with a large number of cellular elements, mainly leukocytes and desquamated epithelial cells. The inflammation in these cases is mainly tubal and the exudate becomes extremely thick and tenacious. There is very little tendency to spontaneous resolution and the condition is apt to persist and develop eventually into chronic middle-ear catarrh. The exudate tends to become thicker and thicker. Vessels grow into it and organization takes place with subsequent formation of fibrous strands and adhesions across the cavity. This process interferes with the movements of the ossicles, and there is also a special tendency to fill up the niches of the two labyrinthine windows, which causes permanent impairment of hearing.

**Symptoms.**—Acute symptoms are generally absent, the main complaint being deafness and feeling of fulness or heaviness in the ear and side of the

head. Tinnitus is usually present. There is no mastoid tenderness and no elevation of temperature.

**Otoscopic Appearances.**—The drum membrane is usually more opaque than normal. There is sometimes loss of the light reflex and the handle of the malleus may be reddened. Radiating vessels may also be seen (Fig. 258). Inflation is generally difficult and produces little improvement in hearing. The deafness is of the sound-conducting type.

In children where the nasopharynx is badly choked up by adenoids the membrane is markedly indrawn with a dull reddish tint due to the congested mucous membrane shining through it. The appearance is characteristic of adenoids, and is not necessarily associated with the pathological changes described above.

**Diagnosis.**—The history of recent deafness following a cold, the opacity of the drum membrane, the degree of deafness of middle-ear type, and the slight improvement on inflation all point to subacute middle-ear catarrh. There is one condition, or rather a variety of the same condition, which may be confused with it, namely, acute otitis media due to the *Streptococcus mucosus*, *q. v.*

**Treatment.**—In the first place any unhealthy condition in the nose or nasopharynx must receive appropriate treatment. The middle ear must next be freed of its sticky secretion as early as possible. Palliative measures such as are effective in acute middle-ear catarrh are of little avail here, and if inflation does not give decided benefit it is necessary to perform a myringotomy or paracentesis followed by suction and inflation in order to remove the stringy and tenacious exudate. Sometimes syringing is necessary as well. There is a certain amount of risk of setting up an acute middle-ear suppuration by this treatment, but even if this occurs, healing with complete restoration of function usually takes place. If the condition is left untreated it persists and becomes chronic adhesive middle-ear catarrh.

When the exudate contains capsulated organisms it is advisable to keep the patient under observation for some time after the ear has returned to normal and have the mastoid process Roentgen-rayed once or twice in order to detect early any latent breaking down of the mastoid cells.

### CHRONIC MIDDLE-EAR CATARRH

(*Otitis Media Catarrhalis Chronica*)

**Etiology.**—This condition may arise from an acute or subacute middle-ear catarrh which has not cleared up. Repeated attacks of acute catarrh may gradually produce the same effect. Any unhealthy conditions in the nose or nasopharynx are predisposing causes, such as adenoid vegetations around the eustachian tube, a deviated nasal septum, chronic nasal catarrh, or sinus suppuration.

**Pathology.**—The pathological changes characteristic of this condition are a chronically inflamed and thickened mucous membrane. The eustachian tube takes part in this and becomes more or less stenosed. In some cases these changes arise from the filling up of the tympanic cavity with thick mucoid exudate, which instead of disappearing becomes organized into masses of loose granulation tissue covered by epithelium. These contract and become strands of fibrous tissue, which stretch across the tympanic cavity in various directions. The niches and hollows in which thick mucus would naturally collect become filled up with new tissue over

which epithelium grows. The result of this is that the normal mucosa is changed into a very much thickened fibrous covering which has a tendency to be thickest in the narrowest parts of the cavity. The niche of the round window is particularly liable to be filled up in this way, as is also the niche of the oval window. The stapes becomes surrounded by loose bands and folds which have a tendency to contract and toughen in time and so interfere considerably with its mobility (Fig. 259). The folds and pouches of mucous membrane around the head of the malleus and body of the incus similarly become much thickened and sclerosed. Owing to the narrowing of the tube free ventilation of the middle ear is interfered with and the air in it becomes partly absorbed with the result that the tympanic membrane is pulled strongly inward.

**Symptoms.**—In some patients the condition can be traced to a severe cold, but in the majority the symptoms begin gradually with tinnitus or subjective head noises followed by a feeling of fullness in the ear, and slowly increasing deafness. The tinnitus is generally continuous and frequently is like the sound of escaping steam, waterfalls, or bells. The noises are

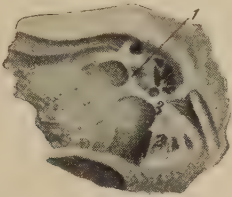


Fig. 259.—Inner wall of tympanum from a case of chronic middle-ear catarrh showing adhesive bands (1, 2) around the stapes (3). (Brühl-Politzer.)

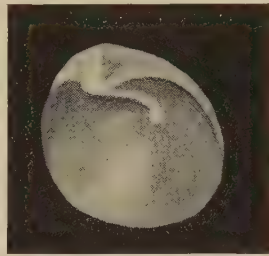


Fig. 260.—Indrawn tympanic membrane.

aggravated when the general health is poor or the patient is fatigued. They cause much more discomfort in most cases than the deafness. The loudness of the subjective noises has no exact relationship to the degree of deafness. Colds in the head increase the symptoms markedly, and in many cases changes in the weather influence the condition to a considerable extent.

**Otoscopic Appearances.**—In the majority of cases there is some alteration of the drum-head. The commonest appearance is that of diffuse thickening. The drum is opaque and whitish, and there may or may not be absence of gloss. During a cold there is sometimes a diffuse pinkish tinge. Others again have the whitish thickening only around the periphery. Along with this the drum as a whole is in many instances drawn inward. In other cases where the disease is confined to the region of the tube the drum may have its normal translucency and gloss, but is markedly indrawn. This condition is recognized by the fact that the short process of the malleus appears very prominent and the handle has a much more horizontal position than usual and appears foreshortened (Fig. 260). Dead white patches are sometimes seen on the drum and are an evidence of previous inflammation. They are commonly called chalk patches, but are really thin plates of new bone.

**Functional Disturbances.**—The deafness is generally of the middle ear or sound conduction type. That is, the lower tones of the scale are lost,

but the medium and upper tones are heard. Rinné's test is negative, *i. e.*, a tuning-fork is heard better when the stem is applied to the mastoid than when the vibrating ends are held to the ear. If the condition is unilateral a sounding tuning-fork placed on the vertex is referred to the bad ear (Weber's test). Bone conduction is lengthened above the normal (Schwabach's test). Paracusis willisiana, or ability to hear better in a noisy place, such as a train or car, is sometimes present, though this is more characteristic of another condition, namely, otosclerosis.

Inflation of the eustachian tube may be extremely difficult, but in cases where the drum is not much thickened and distinctly indrawn it may improve the hearing considerably. In some of the other cases where there is considerable thickening of the drum (which is presumptive evidence that the all-important inner wall is also much thickened) inflation has very little effect on the hearing. Patients with this condition hear well over the telephone as a rule.

**Diagnosis.**—The main points in diagnosis are: (1) Long history; (2) worse with colds; (3) thickened or indrawn drum-head; (4) middle-ear type of deafness; (5) narrowed eustachian tube.

Otosclerosis gives a similar type of deafness, but usually though not always has some family history of deafness. The drum-head may be normal and the eustachian tube be perfectly patent. It preponderates in the female sex and generally comes on in the "teens."

Gellé's test is said to differentiate the ankylosis of the stapes in otosclerosis from this condition. However, the stapes may be almost completely ankylosed in advanced middle-ear catarrh from tough fibrous bands and adhesions, so that the results are somewhat similar.

**Course of Disease.**—It is generally progressive. Each succeeding cold or inflammatory attack is apt to leave the condition a little worse. After a time a certain amount of nerve deafness supervenes on the middle-ear catarrh and increases the deafness, mainly by lowering the upper tone limit.

**Treatment.**—When a case is seen in the earliest stages, that is, when there is still mucoid exudate in the middle ear, preventive treatment as described in treatment of subacute catarrh may be entirely successful. Abnormal nasal or nasopharyngeal conditions must be remedied, such as adenoid vegetations in the nasopharynx, hypertrophied turbinates, or deviations of the septum or sinus suppuration. Any exudate in the ear must be removed by paracentesis and suction.

The majority of cases, however, are only seen in a much more advanced condition where the ear no longer contains any exudate and the mucous membrane is already sclerosed. In these cases remedying any nasal abnormality is generally disappointing as far as the effect on the ear is concerned. Considering the pathology of the condition this is only to be expected, as the scar tissue once formed cannot be removed by any known method.

Vibratory massage occasionally gives some temporary relief, probably by improving the local circulation and by stretching the adhesive bands. Direct massage of the ossicles by pressure of specially designed probes is difficult, painful, and of questionable value. When eustachian tube obstruction is the leading feature of the case inflation through a catheter at intervals with medicated vapors through Dench's attachment (Fig. 261) will give some benefit.

A suitable formula is the following:

R. Iodi.....	gr. iij.....	.194
Ether.....	drams ij.....	7.103
Acid carbol.....	drams ij.....	7.103
Creosote.....	dram j.....	3.552
Alcohol.....	drams iij.....	10.655

As an alternative:

R. Mentholis.....	gr. x.....	.64
Ol. eucalypt.....	℥v.....	.296
Ol. cinnamomi.....	℥v.....	.296
Alcohol.....	drams ij.....	7.103

When the stenosis is very obstinate the use of a bougie will re-establish the lumen and may alleviate the symptoms. In the last few years treatment by the Roentgen ray has been used with beneficial results both as regards hearing and subjective head noises.

Harris<sup>1</sup> has reported good results in the treatment of chronic progressive deafness with the galvanic current. The negative pole causes hyperemia, local elevation of temperature, and softening of scar tissue.

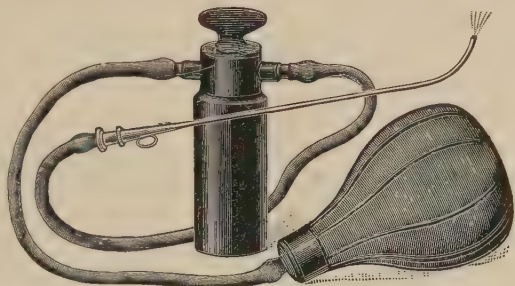


Fig. 261.—Dench's apparatus for insufflating vapors into the ear.

In severe cases where there

is a high degree of deafness lip reading should be advised. If this is learned when the patients are still young and receptive it is of the greatest value in allowing them to associate freely with other people and lead a normal life.

When it is not possible to learn lip reading, some artificial aid to hearing may be used, such as ear-trumpets, speaking-tubes, or one of the various types of electrical amplifying devices.

### ACUTE MIDDLE-EAR SUPPURATION

(*Otitis Media Acuta Purulenta*)

**Etiology.**—Any abnormal condition in the nose or nasopharynx associated with catarrh predisposes to infection of the middle-ear tract. Chief among these is the presence of an enlarged pharyngeal tonsil or "adenoids." The importance of adenoids as a predisposing factor in the setting up of inflammation of the middle ear can hardly be overestimated. The presence of pus in the nose from infected nasal sinuses increases the probability of an accidental infection of the ear from violent blowing of the nose or even from swallowing while lying on the side with pus lying in the pharyngeal end of the eustachian tube. To a lesser extent simple nasal obstruction from hypertrophied turbinates or a bad deviation of the nasal septum are predisposing causes.

Of the direct causes of acute middle-ear suppuration the common cold or coryza is by far the most frequent. It also occurs in the exanthemata such as scarlet fever, measles, diphtheria, and whooping-cough, and, in fact, in any upper respiratory infection. In scarlet fever or measles a secondary rise of temperature after the initial fever has subsided generally indicates the onset of acute otitis media. In children it frequently follows the irritation of the mucous membranes from teething.

Otitis media may occur from accidental forcing of water into the ear while douching the nose or while swimming. Although in the majority of cases the infection reaches the middle ear by way of the eustachian tube, it may come from the external meatus as in traumatic rupture of the drum, which is followed in a number of cases by an acute otitis. The latter may be of a very severe type. In cases of fracture of the skull infection sometimes spreads through the ruptured drum into the middle ear with very grave results.

In young infants otitis media is not uncommon from forcing of liquor amnii or meconium into the middle ear during parturition. Many of these cases of otitis are sterile and are merely the reaction against foreign body irritation. According to Preysing<sup>2</sup> those that are infected are generally pneumococcal.

Age has an influence on the incidence of acute middle-ear suppuration, and children are more subject to catarrhal infections than adults on account of the presence of adenoids and the much wider eustachian tube. The majority of acute cases of middle-ear suppuration occur in children.

Debilitating diseases such as diabetes predispose to middle-ear infections. The weakening effect of certain epidemic diseases, such as mumps, measles, or influenza, facilitates the passage of streptococcal infections into the middle ear.

**Pathology.**—The pathological changes which occur in acute middle-ear suppuration and acute middle-ear catarrh are essentially similar and differ only in degree. They resemble the changes which take place in acute inflammation of the nasal or any other mucous membrane, modified by the shape and relationships of the middle-ear cleft. In all but the rarest instances the infection reaches the tympanic cavity by way of the tube. The first change is an intense hyperemia and swelling of the mucous membrane. This is rapidly followed by the outpouring of an exudate which is at first serous, but after a short time becomes sero- or muco-purulent from diapedesis of leukocytes and shedding of epithelial cells. The eustachian tube takes part in the process and on account of the narrowness of the lumen at its tympanic end speedily becomes blocked. Leakage of the fluid from the tympanum into the throat becomes impossible. The mucous membrane meantime has become swollen to ten or twenty times its normal thickness so that with the continually increasing exudate and the narrowed cavity there is considerable tension, which causes severe pain. The tympanic membrane becomes bulged outward and sooner or later gives way and allows a copious discharge of pus. If, however, the drum membrane does not rupture early the purulent exudate becomes forced back into the mastoid cells and may fill them completely. Perforation of the drum is not a purely mechanical process, but is preceded by small celled infiltration of its layers with destruction of part of the fibrous network. In a large proportion of cases perforation occurs before the mastoid cells are involved to any serious extent and the discharge of pus is followed by a gradual recession of the inflammation, lessening of the discharge, and finally healing of the perforation, and the return to normal.

In many instances, however, the perforation is too small or the pus is too thick for free drainage to take place and only partial relief of the pain is obtained, and the healing process is much delayed. A few days after perforation has occurred healing of the edges begins to some extent, and the

hole tends to become smaller and smaller. If there is at the same time a copious secretion of pus there is a recurrence of pain and danger of the pus being forced into the mastoid cells. When there is a delay of some days before perforation occurs and the inflammation continues the mastoid cells nearly always become involved. When the subsequent drainage through the perforation is free the inflammation in the mastoid cells may clear up, although on account of the complexity of their arrangement drainage from the more distant cells must be imperfect at the best. Healing is protracted in these cases and may last several weeks.

When the drainage is insufficient or absent and the infection severe the mastoid cells become intensely inflamed. Necrosis of the mucous membrane occurs and the bony framework is broken down and absorbed and an abscess is formed (acute mastoiditis). In time the pus works its way to the surface or through the inner table of the skull, causing a subperiosteal or intracranial abscess respectively.



Fig. 262.—Section of a normal mastoid cell. The mucous membrane is thin and covered by a single layer of epithelial cells. (Compare with Fig. 263.)

The borderline between a mild acute suppuration and an acute or sub-acute catarrh is very indefinite. The deciding factors depend on the type and virulence of the invading organism, the resistance of the patient, and the anatomical peculiarities of the region.

**Histological Changes.**—In acute inflammation of the ear as already mentioned, the mucous membrane becomes hyperemic and edematous. A fact which has been frequently noted is that the epithelium in the tympanic cavity, which is normally cubical, becomes columnar and ciliated during the course of an acute inflammation. In the normal condition ciliated epithelium is found only in the tube and around its tympanic orifice. The changes which occur in the mastoid cells may be conveniently studied microscopically in bone chips removed at operation. In a mild inflammation the mucous lining, which is very thin and lined by squamous epithelium, becomes swollen and the cavity fills with clear exudate. Later the exudate becomes increasingly rich in leukocytes, cast-off epithelial cells, and micro-

organisms (Figs. 262, 263). The mucous membrane shows intense small celled infiltration which later spreads down to the bone. When the cavity has been full of pus for a day or two and the virulence is only moderate small strands are seen extending from the mucous membrane into the exu-

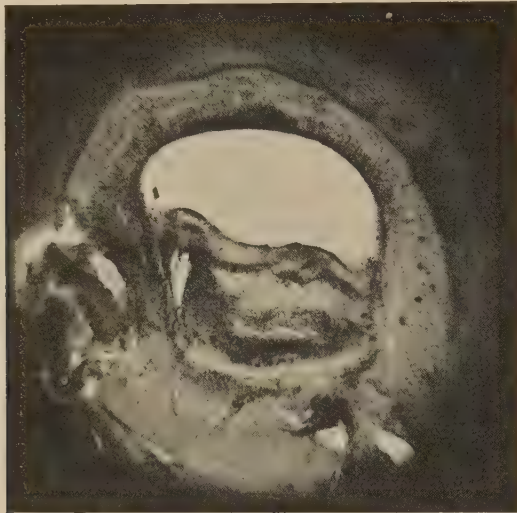


Fig. 263.—Section of a mastoid cell from a case of acute middle-ear suppuration. The mucous membrane is thickened and infiltrated and the cavity is partly filled with pus.

date (Fig. 264). On closer inspection these are seen to be fibroblasts which are migrating into the exudate. They are followed by capillaries and threads of fibrin, and soon there is some definite organization around the periphery which gradually spreads toward the center. Epithelium spreads along the capillaries on to the partly organized mass which is suspended in the center of the cell (Fig. 265).

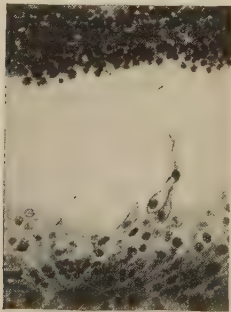


Fig. 264.—Commencing organization of exudate. Fibroblasts are migrating from the mucous membrane into the exudate (Döderlein).

In many cases it gradually shrinks and is finally absorbed altogether, after which the capillaries, etc., disappear and leave the cell much as before or with a few thin fibrous strands crossing the cavity. In others the mass becomes replaced by a network of fibrous tissue in which small centers of

new bone appear. These grow and coalesce, and finally the whole cell becomes obliterated by new bone (Figs. 266, 267).



Fig. 265.—Section showing a mastoid cell in a case of acute otitis media of five weeks' duration. The mucous membrane is thickened and infiltrated. The blood-vessels are engorged. The center of the cell is occupied by masses of semi-organized exudate.

When the infection is more virulent none of this organization takes place. The whole cell becomes filled with pus and there is intense infiltra-

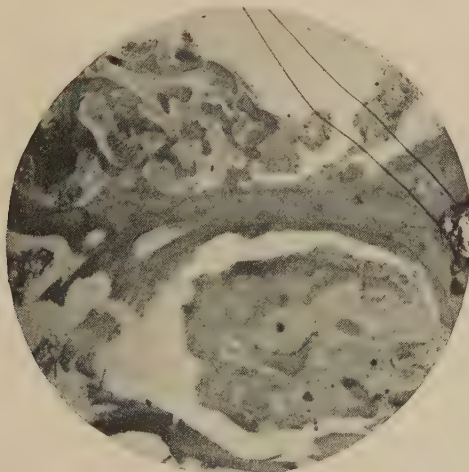


Fig. 266.—Acute mastoiditis showing organization of the exudate with areas of new bone formation. Healing is taking place by obliteration of the cells.

tion of the mucous membrane. Its outline becomes lost from necrosis of the epithelium. The bony walls are now filled with pus and broken-down tissue (Fig. 268). Osteoclasts are seen on the edge of the bone which begins

to be pitted and shows signs of absorption. Necrosis occurs from interference with its blood-supply added to the direct toxic effects of the infection. Absorption advances rapidly. The cells coalesce and the whole

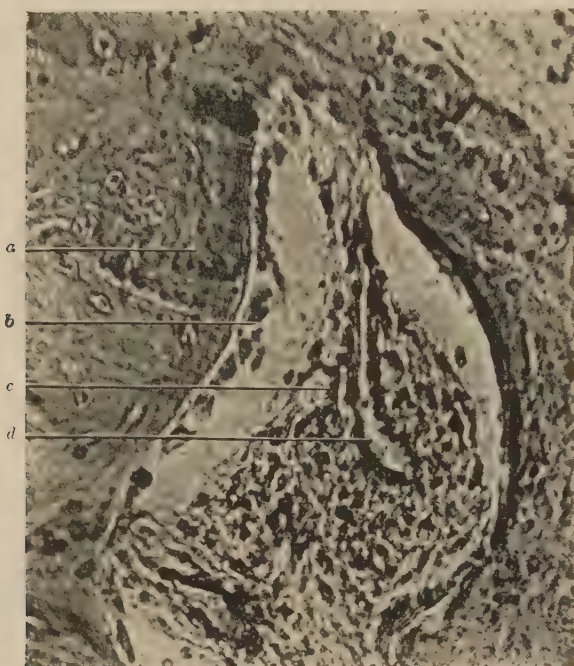


Fig. 267.—High-power photograph from a case of mastoiditis of nine weeks' duration. Extensive proliferation of new bone had taken place. *a*, New bone; *b*, osteoblasts; *c*, organized exudate; *d*, blood-vessel.

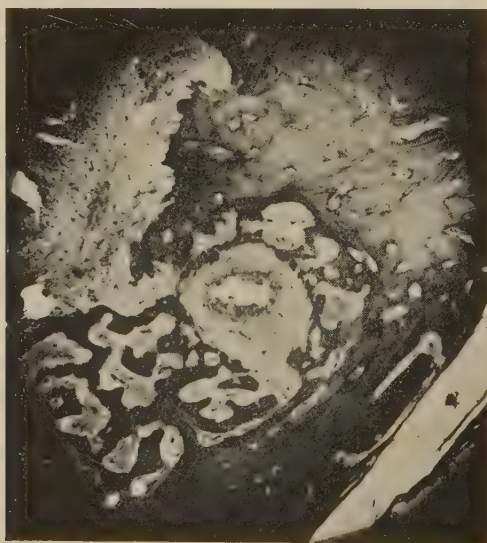


Fig. 268.—Section showing acute mastoiditis with caries and necrosis of the bone. The outline of the cell in the center has become blurred owing to the intense infiltration and absorption of the bony walls. The infiltration is spreading along the marrow spaces.

mastoid structure becomes broken down. It is not infrequent to find each of these processes occurring in the same mastoid at once. One area may be healing while another is breaking down. Similar changes to those described take place in the tympanic cavity itself, although on account of better drainage the more intense forms are rare. In mild cases, in which there has been either no perforation or a very inadequate one, organization of the exudate takes place much as in the case of the mastoid cells. These changes are most marked in the small pouches and niches of the cavity where the exudate tends to stagnate. The niches of the two labyrinthine windows from their situation and relationship are specially liable to become filled up with new tissue. Such tissue becomes more fibrous as time goes on and contracts, leading to interference with the movements of the ossicular chain.

**Bacteriology.**—The most frequently recovered micro-organism from acute inflammatory conditions of the ear is the streptococcus in one or other of its varieties. Next in frequency are the pneumococcus and staphylococcus in that order. Other less frequent organisms are the pneumobacillus, *Bacillus pyocyaneus*, *B. diphtheriæ*, *B. tuberculosis*, *B. coli*, etc.

While streptococci are capable of causing severe otitis and mastoiditis in healthy individuals, their virulence is greatly increased when there has been some antecedent infectious disease, such as influenza, measles or mumps. This was seen in the armies during the World War when in certain areas there were epidemics of otitis media and mastoiditis following shortly after measles or influenza. These have been fully described by Lathrope,<sup>3</sup> Scott,<sup>4</sup> Hill,<sup>5</sup> and others. In the majority of the areas *Streptococcus hemolyticus* was recovered from the mastoid in pure culture though in other areas *S. viridans* predominated. These cases of mastoiditis were extremely rapid or fulminating, and in some the involvement of the mastoid appeared to be almost simultaneous with the invasion of the middle ear.

The *Streptococcus mucosus capsulatus* of Schottmüller causes a type of otitis and mastoiditis which differs considerably from the ordinary suppurative form. This organism is now regarded by most bacteriologists as belonging to the pneumococci rather than to the streptococci. It has a tendency to grow in chains and is not lanceolate in shape, but it has a thick capsule and is soluble in bile and gives fermentation reactions like the pneumococci. It is now classed as a Type III *Pneumococcus* (*vide infra*).

**Symptoms and Course.**—Acute middle-ear suppuration usually supervenes on a common cold or on one of the exanthemata. There may be a slight chill or in children a slight convulsion. A feeling of fulness in the ear is experienced along with deafness and some subjective noises. The feeling of fulness becomes more marked till it becomes a severe pain which is sharp in character and is felt in the ear itself. It is generally continuous, but with more acute exacerbations at times, and like toothache it is usually worse at night. Later it may be felt all over the side of the head. Pain in the ear is caused by air or liquid under pressure and is relieved when perforation of the drum occurs and a free escape of discharge takes place. In the early stages of a severe infection the ear may ache even before the filling up of the cavity with fluid. If the drum is opened at this stage the imprisoned air hisses out and the pain is relieved. When the inflammation is mild the discharge is at first thin and yellowish or pink, but after a day or two it lessens in quantity and becomes thicker and more purulent.

After flowing for a period varying from a few days to a few weeks the discharge dries up, the perforation heals, the drum gradually regains its normal appearance, and the hearing returns.

In other cases in which the perforation is too small or the discharge too copious or too thick the pain is not relieved to any great extent and the condition does not subside. When there has been pent-up fluid in the ear for several days there is generally some tenderness over the mastoid antrum or over the tip of the mastoid. The longer the inflammation has lasted without perforation and free discharge the more liable is the mastoid to become involved.

Late paracentesis may not be enough to avert an acute mastoiditis. It not uncommonly happens that after perforation the discharge ceases and the pain returns. This means that the perforation has been too small and has closed over. Unless this is treated promptly an acute mastoiditis is likely to result.

In adults there may be no rise of temperature, and if any it is usually only slightly above normal. In children on the other hand, it is not unusual to find a temperature of 102° to 104° F. In very young children there may be head retraction leading to a suspicion of meningitis.

An acute otitis media may if untreated become chronic, may cause acute mastoiditis, or even infect the labyrinth. These conditions will be discussed under their appropriate sections.

**Otoscopic Appearances.**—The appearances vary according to whether perforation has already occurred or not. If it has not occurred the drum membrane in the early stages is seen to have a bright red stripe in the middle caused by the handle of the malleus. A little later the whole membrane becomes bright red as does also the skin of the innermost part of the external meatus. Soon afterward it loses its gloss and becomes bulged outward (Figs. 269, 270). On account of its oblique position in the bottom

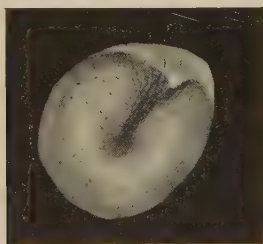


Fig. 269.—Acute otitis media with bulging of the drum-head.

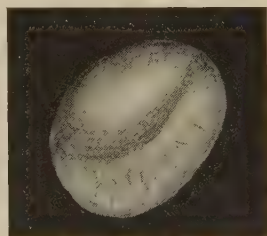


Fig. 270.—Acute otitis media. Marked bulging of posterior part of drum-head.

of the meatus the bulging is most evident in the posterosuperior quadrant. The handle of the malleus is frequently indistinguishable except as a dimple in the center of the membrane. A certain amount of desquamation of the epithelium begins to take place after a few days, which hides the real appearance of the drum unless it is wiped or syringed clean. A small yellowish spot indicating impending rupture is sometimes seen.

When perforation has occurred the meatus is generally full of pus. After syringing or wiping it clean a pulsating spot of light is often seen and this is specially noticeable in the reflection of light from pus exuding through the perforation. The perforation is generally very minute and somewhat

difficult to see. It is most commonly situated about the center of the drum posterior to the malleus. If in doubt as to whether there is a perforation or not Siegle's pneumatic speculum should be used. By applying suction a drop of pus will appear and reveal the perforation.

In children there is often considerable tenderness on pressing over the tragus, but this is uncommon in adults.

**Diagnosis.**—Several other conditions may give rise to pain in the ear and may cause confusion. A boil or furuncle will cause not only pain but also discharge, and sometimes deafness. This is distinguished by the extreme tenderness of the meatus which is absent in acute otitis media. The swelling is frequently so great as to close the meatus completely, causing deafness, and if there is discharge in addition it becomes important to know whether there is an otitis media as well or not. If a small speculum is introduced carefully beyond the swelling and the deep meatus cleansed the hearing may be tested, and if normal, otitis media can be ruled out.

It is not uncommon to meet with cases of otalgia which are really cases of toothache with the pain referred to the ear rather than to the tooth. This is associated most frequently with a lower molar on the same side and particularly with the wisdom tooth. Tinnitus is also found at the same time. The normal drum-head with good hearing and a defective tooth will clear up the diagnosis.

An inflamed tonsil will cause a referred pain in the ear, but is not likely to give rise to difficulty in diagnosis. Among the rarer causes of otalgia is herpes oticus. In this condition pain is severe and may be accompanied by tinnitus and some deafness. Till the characteristic herpetic eruption appears diagnosis is difficult, if not impossible.

**Treatment.**—In the earliest stages it may be possible to abort the condition by administering a brisk purge, putting the patient to bed and endeavoring to produce free perspiration by means of a hot bath, 15 grains of aspirin or Dover's powder, and a large hot lemonade or hot toddy. Heat to the ear in the form of a hot-water bag relieves the pain slightly. The pain is due to mechanical tension and cannot be relieved to any appreciable extent by the use of anesthetic drops in the ear. They may, however, be used if for any reason paracentesis or myringotomy cannot be carried out at once. A suitable formula is the following: Cocaine hydrochlor., acid. carbolic.  $\bar{a}\bar{a}$  gr. vj; glycerini,  $\bar{3}$ ij. Menthol may be substituted for the cocaine if desired.

The ear-drum must be opened as soon as any bulging of the drum is seen. The earlier the paracentesis is done, the less will be the likelihood of mastoid complications and the quicker will be the healing.

The old adage "ubi pus evacua" holds good here as everywhere else and relief of the tension in the middle ear should be given as soon as possible. Myringotomy may be carried out in infants without anesthesia, but in all older children and adults an anesthetic is necessary as incision of the ear-drum is excruciatingly painful and even the most stout hearted patient cannot keep still without jerking, and an insufficient incision is the result. In adults this may be done under local anesthesia by Bonain's or Blegvad's drops. Bonain's solution is: Phenol pur., menthol., cocaine hydrochlor.,  $\bar{a}\bar{a}$  gm. j; adrenaline hydrochlor., mg. j. This forms a thick syrupy liquid. Blegvad's solution is cocaine hydrochlor., acid. salicylici,  $\bar{a}\bar{a}$  gm. 4; alcohol, 8 c.c.

These drops are best applied on a small piece of absorbent cotton laid carefully and firmly against the drum and left in position for fifteen to twenty minutes. At the end of that period the drum-head is blanched white and sufficiently anesthetized to make the incision. If there is no blanching the anesthetic has not acted.

In the majority of cases general anesthesia is preferable, when the incision may be made carefully and efficiently. A mere stab of the drum-head is of little use. A free incision from top to bottom is necessary to get good drainage (Fig. 271). Some surgeons prefer to make a curved incision to avoid too early closure of the opening (Fig. 272). There is no fear of damaging permanently the drum-head and injuring the hearing as the edges grow together with great rapidity; in fact the difficulty often is to prevent healing till the discharge has ceased.

As an anesthetic ethyl chloride sprayed on an ordinary mask has been used by the writer for many years with complete satisfaction. About one minute of complete anesthesia is obtained, which is as much as is necessary.

The meatus may be packed with sterile gauze but, as a rule, the discharge is so free that it becomes soaked immediately. It is enough to keep

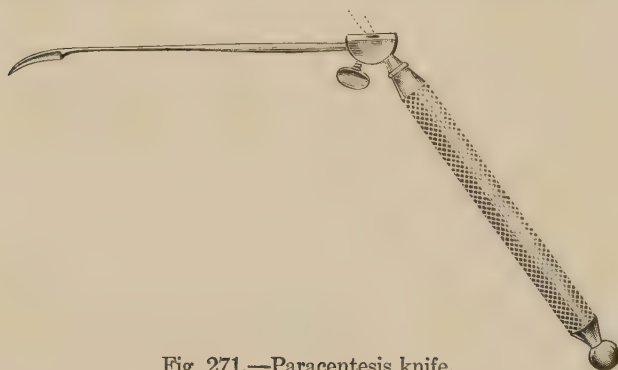


Fig. 271.—Paracentesis knife.



Fig. 272.—Outline of drum-head showing line of incision as an interrupted line.

absorbent cotton in the ear and change it whenever it is soaked. Next day the ear is syringed with warm boracic solution at frequent intervals and this is followed by instillation of drops of hydrogen peroxide or 10 per cent. solution of argyrol. The ear is plugged with clean cotton between treatments. This treatment is continued till the discharge ceases altogether.

Immediate relief of pain is obtained after paracentesis unless the mastoid is already involved, and in children even when there is already mastoid tenderness the condition will generally clear up. The duration of the discharge varies from a few days to several weeks. If it continues to be profuse even after three weeks any adenoids present must be removed as they constitute by far the most important factor in setting up and keeping up ear infections. If that is not enough to clear up the condition the question of a mastoid operation must be seriously considered to get rid of the infection and restore the hearing, as each week that the inflammatory process continues makes it more likely that there will be some permanent damage to the delicate sound-conducting apparatus.

## SPECIAL VARIETIES OF MIDDLE-EAR SUPPURATION

**Otitis Media Necrotica.**—This fortunately rare variety occurs commonly in severe scarlet fever, especially when complicated with diphtheria. The infection is extremely virulent and causes a rapid destruction of the drum-head which may slough out entirely. The mucous membrane of the tympanic cavity becomes necrotic and leaves areas of bone exposed. These in turn necrose and may cause extension into the labyrinth. This form of otitis is always accompanied by severe throat lesions and has a very high mortality.

Otitis media in the milder cases of scarlet fever is similar to the normal acute middle-ear suppuration from other causes.

**Otitis Media Hemorrhagica.**—This condition is found most characteristically in influenza. The drum membrane shows hemorrhagic spots or bullæ. These may apparently cause acute pain in themselves, since occasionally when they are pricked the pain subsides even though the drum itself has not been punctured. The exudate in the middle ear is a thin pinkish pus which only becomes yellow after several days.

Bone from cases of this type shows intense congestion with hemorrhagic exudate in the cells. The hemorrhages are sometimes an indication of extreme virulence of the infection, but in other cases the condition runs a fairly normal course and is not associated with a high mortality.

**Otitis Media from Capsulated Organisms.**—Some years ago Neuman<sup>6</sup> and Ruttin<sup>7</sup> drew attention to a special type of otitis media and mastoiditis. It was caused by the *Streptococcus mucosus capsulatus* and gave rise to a number of serious complications. Clinically the onset of mucosus otitis is characterized by painlessness and absence of fever. There is some impairment of hearing, tinnitus, and a sensation of fulness in the ear. The drum-head shows only trifling changes, such as slight opacity with reddening of the malleus. Frequently no perforation occurs and the condition apparently subsides, though the deafness and sensation of fulness persist. After a variable period which may last as long as three months symptoms of mastoiditis or intracranial complications develop. During the quiescent stage it is unusual to get mastoid tenderness even where there is extensive caries of the underlying bone. The discharge is noticeably sticky and mucoid.

Repeated roentgenograms are of very great value in cases of suspected infection with the *Streptococcus mucosus*, or where that organism has been identified, since the clinical signs are so vague and indefinite. This may be the only means of detecting a latent destruction of the interior of the mastoid.

The condition in the early stages is almost indistinguishable from mucoid subacute middle-ear catarrh and hence the latter condition must always be viewed with suspicion and kept under observation for some time.

## CHRONIC MIDDLE-EAR SUPPURATION

(*Otitis Media Purulenta Chronica*)

Chronic middle-ear suppuration is the result of an unhealed or recurring acute otitis media. One of the most frequent causes of the transition into the chronic form is the lack of treatment in the acute stage. On this account it is uncommon in people of good social environment, but very common among the slum population.

Another factor which delays cure of the acute otitis media and leads to its becoming chronic is the presence of adenoid vegetations in the nasopharynx which cause continual reinfection of the tympanic cavity through the tube.

The chronicity may be due to the severity of the original infection, as in scarlet fever or scarlet-diphtheria, where the greater part of the drum-head may slough out and parts of the mucous membrane become necrotic, leaving areas of denuded bone. This results in irreparable damage to the ear with persisting discharge.

According to Wittmaack<sup>8</sup> the suppuration becomes chronic in many cases owing to a weakened state of the mucous membrane of the middle ear from faulty development. Cheate many years ago advanced the view that the reason why chronic middle ear suppuration and sclerotic mastoids are associated is that the sclerosis of the mastoid is the cause, not the effect, of the chronicity. He held that the diploëtic or sclerotic mastoid has remained infantile and undeveloped and, therefore, offers less resistance to infections.

Wittmaack has shown that the normal process of cellular development of the mastoid goes on from birth to the third or fifth year. In the early years the mucous membrane of the antrum and cells is thick and of embryonic character. If any pathological process such as an infantile otitis should occur before the embryonic tissue has disappeared further development is arrested and the patient remains with a diploëtic or sclerotic mastoid, but, most important of all, with a mucous lining to the antrum of embryonic and unresistant tissue.

**Pathological Changes.**—In the milder cases the mucous membrane is thickened and fibrous. The contours of the tympanic cavity are smoothed out, and the niches are filled by hyperplastic tissue which shows a variable amount of small-celled infiltration. Small cystic spaces are constantly found in it and the vessels are generally dilated. In others the bone shows signs of caries in certain areas. The small-celled infiltration reaches down to the bone and into the marrow spaces and vascular channels. Osteoclasts are found in the dimples and hollows of the bone, indicating active absorption. This condition is fraught with considerable danger if the caries is on the inner wall of the tympanum. The mucous membrane over such an area of caries is generally markedly hyperplastic or granulomatous (Fig. 273). A granulation may be so large as to protrude through the perforation in the drum-head, when it usually becomes stalked and enlarges in the meatus. It is then known as an aural polyp. It is generally covered by epithelium of some sort, though in spots the epithelium may be absent. A polyp is recognized by the presence of a reddish mass filling partly or wholly the external auditory canal and not attached to its walls. This may be verified by passing a probe round it.

In most cases of long duration a certain amount of caries of the ossicles has taken place. When there is a large perforation, the tip of the handle of the malleus has generally been absorbed. The long process of the incus and the body frequently become eroded so that the bone falls out of its position and is lost. This has the advantage that it leaves the aditus and part of the attic empty, and so gives improved drainage.

In another type the suppuration may be mainly in the attic or recessus epitympanicus. The attic is subdivided into many small cavities by the

ligaments of the ossicles and numerous folds of mucous membrane. The space between the head of the malleus and the outer wall in particular is filled by a network of small mucous membrane folds, and in such a situation infection is apt to lurk indefinitely on account of the poor drainage. The main part of the tympanic cavity may be completely shut off by swelling of the folds of mucous membrane at the level of the neck of the malleus where the cavity is always narrow in any case, and it is not uncommon to find cases where the suppuration is confined to the attic and the rest of the tympanum is normal. These are associated with a perforation in Shrapnell's membrane.

In certain circumstances the epithelium of the meatus or outer surface of the drum-head may grow into the middle ear and spread itself over the inside, replacing the mucous membrane. When the perforation is very large

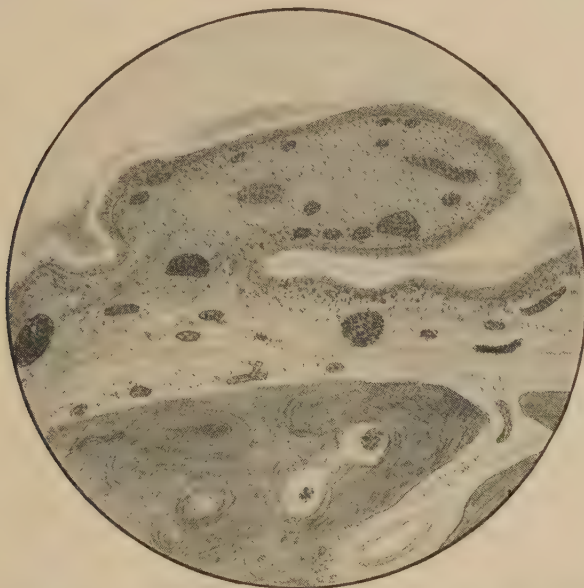


Fig. 273.—Section of the inner wall of the tympanum in a case of chronic middle-ear suppuration. Note the hyperplastic and fibrous mucous membrane with dilated blood-vessels. A commencing polypus is seen.

and caries is absent the whole of the tympanum may be lined by dry skin with cessation of the discharge. In other cases, especially when the attic or antrum is the seat of chronic suppuration, the course is not so favorable. The skin growing in through a marginal or Shrapnell's perforation spreads into a cavity where granulations and pus are present with many obstacles to the complete epidermization and drying up of the cavity. The skin becomes soaked with pus and desquamates freely and continuously. The result of this is the formation of a lamellated or onion-like mass of pultaceous material full of micro-organisms of many varieties (cholesteatoma). This gradually increases in size from continual deposition of fresh layers till it fills the cavity completely. Slow erosion of the bone occurs, especially in any projecting spots. The projection of the external semicircular canal is particularly liable to become eroded, resulting in invasion of the labyrinth by the septic process. The erosion of the bone

may be extensive in all directions, laying bare the dura mater over the roof of the tympanum or the lateral sinus posterior to it, the consequences of which may be of the utmost gravity.

**Symptoms.**—A purulent discharge from the ear which may be continuous or intermittent is the main symptom. Deafness is usually present to some extent but varies considerably. Cases with the disease confined to the attic may have almost normal hearing, while in others a shout is heard with difficulty. When there is little change round the region of the stapes and the round window the hearing may be fairly good even though the drum-head is almost completely absent.

Pain is uncommon but when present is a sign of insufficient drainage and is of grave import, as it may be the first sign of some impending intracranial complication. In attic suppuration a sensation of heaviness on the affected side of the head or actual headache is not infrequent but should always give rise to some anxiety, and the patient should be kept under close observation while it lasts. It is sometimes due to a dried crust blocking up the perforation, and when this is removed the discharge escapes and the symptoms subside.

The pus in most cases of chronic middle ear suppuration is very foul smelling and contains a wide variety of microbes, both pathogenic and saprophytic. *Bacillus proteus* is commonly found and is generally a harmless saprophyte, but under certain circumstances may in combination with other microbes cause serious complications. White flaky material in the pus indicates the presence of cholesteatoma.

Dizziness is sometimes complained of. It usually indicates some erosion or thinning of the labyrinthine wall or even invasion of the labyrinth itself.

**Otoscopic Appearances.**—The ear-drum may be completely obscured by pus, which must be removed by syringing or mopping. The perforation in the drum-head is generally single and may vary in size from a pinhead to almost complete absence of the drum-head. The membrane is generally much thickened and is frequently adherent to the promontory. When the perforation is very large the whole of the promontory may be visible as well as the head of the stapes and niche of the round window. In the case of attic suppuration the perforation is generally in the *membrana flaccida* (Shrapnell). It is generally small and often obscured by a small crust of dried secretion, but in others it is large enough to make the head of the malleus visible.

The situation of the perforation is of some clinical significance. Thus an anterior perforation is generally associated with chronic tubal catarrh, a central one with suppuration mainly in the floor of the tympanum and a posterosuperior one with suppuration mainly in the antrum. A marginal perforation in this situation is generally associated with cholesteatoma and this may be brought into view by syringing or suction.

In some cases the perforation heals between the periods of recrudescence and a transparent cicatrix is seen which may at first sight be mistaken for a perforation. The use of a pneumatic speculum or inflation of the tube will distinguish it from a perforation by making it bulge.

**Hearing Tests.**—The deafness is of the middle ear or sound-conducting type except in very advanced cases where some nerve-deafness is super-added. When the deafness is very marked the good ear must be excluded from the testing by using a Bárány noise-box or buzzer.

**Prognosis.**—Any patient with chronic middle-ear suppuration is potentially in danger of his life. It may be likened to a person carrying a live bomb around for years without accident, but some day the safety catch may be released and the bomb goes off. A patient may live for years with ear discharge with no untoward results, but there is always the possibility of a sudden flare up of the infection, accompanied by symptoms of one or more intracranial complications. A cold, an attack of influenza, a blow or jar on the head may be the immediate exciting cause. Most of the life insurance companies regard persons with chronic middle-ear suppuration as bad risks and either will not accept them at all or only at considerably higher rates.

When there is a large perforation with very little discharge the risks are not so great as in the case of attic suppuration with cholesteatoma and a small perforation. Many cases dry up for long periods under treatment and are not so serious as those which continue to discharge in spite of treatment.

It is very inadvisable for anyone with an unhealed chronic otitis media to go into the wilds or out of reach of medical attention for any long periods. In the case of those whose occupation takes them far from civilization a radical mastoid operation should be advised in order to free them from the danger of some complication.

**Treatment.**—Where there is a large perforation with little or no discharge careful cleansing followed by the instillation of antiseptic or astringent drops may dry up the ear in a few days. Failure to clear up the discharge in chronic middle ear suppuration is generally due to the difficulty in reaching the most diseased areas which are usually not in the main tympanic cavity itself but in the tube, attic, antrum, or mastoid cells.

**Routine Treatment.**—The large numbers of cases of chronic middle ear suppuration met with in a public clinic necessitate some routine method of treatment. Routine treatment will not of course suit all cases and may have to be modified to suit the individual and be supplemented by special methods to be described later. A great deal has been written about the respective merits of the wet and dry methods, *i. e.*, with or without syringing.

The dry method consists in mopping out the meatus with cotton-tipped applicators and applying some medicament, such as boracic powder or liquids such as solution of argyrol. The objection to this method is that the meatus only is cleansed and the middle ear is not touched unless the treatment is carried out personally by an aurist.

In hospital practice syringing is best carried out with one of the large all-metal syringes (Fig. 274). For home use, however, the ordinary all-rubber pear-shaped syringe is the most satisfactory when properly used (Fig. 275). The patient or his relatives must be instructed how to use it. The points to be attended to are: (1) The lotion must be about blood heat or a little warmer. Boracic lotion or weak lysol 3ss to the pint may be used. (2) The syringe must be *filled* with the fluid and contain no air. (3) The nozzle of the syringe must be inserted carefully into the meatus as far as it will go. (The tendency is to hold it right outside the meatus, and it should be explained that the point cannot reach far enough to touch the drum.) (4) The syringe is emptied by a single steady pressure of the bulb. One or two syringefuls may be necessary before the return flow is clear. (5) Hold the head to the side till the water runs out. (6) Lie on the sound side and

fill the meatus with one or other of the following solutions: Hydrogen peroxide solution full strength; saturated solution of boracic acid in alcohol 90 per cent.; solution of argyrol 10 per cent. Allow to soak five minutes or more before letting it run out. (7) Keep the meatus plugged between treatments with absorbent cotton. In some cases douching the ear with a fountain douche is preferred to syringing. The advantage of this method is the large quantity of fluid used. The disadvantage when the patient uses it himself is that it is sometimes painful to introduce the nozzle far enough into the meatus, and as a consequence it is not done.

Fowler's method of douching the ear has several advantages over ordinary douching. A glass bell or cup fits over the ear tightly, enclosing the whole auricle (Fig. 276). Through the center of it projects a narrow nozzle tipped with rubber, which is inserted into the ear. The other end of the nozzle is attached to the douche can or fountain. The return flow is carried off by a tube attached to the dependent side of the bell. Owing to the fact that the bell fits tightly over the ear there is a certain amount of suction from the rush downward of the re-



Fig. 274.—Metal ear syringe.



Fig. 275.—All rubber ear syringe.

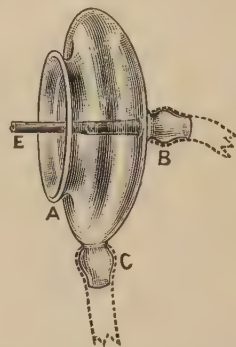


Fig. 276.—Fowler suction bell ear douche. Consists of a glass bell, A, which fits tightly over the ear, a central glass, rubber-tipped nozzle, B, E, delivers the irrigating stream to the aural canal. The return flow passes through the outlet at the bottom, C, and produces a partial vacuum.

turn fluid. This treatment is helped in intelligent patients by previous auto-inflation in order to blow the discharge into the meatus. The advantage of syringing is that not only the meatus but the tympanic cavity and tube are in most instances cleansed by it, as is shown by the fluid reaching the nasopharynx. The number of times daily that this treatment is necessary depends on the amount of discharge, the aim of the treatment being to keep the ear free from pus. In a series of school children treated once daily by syringing in a hospital clinic (Leith General Hospital) some years ago the discharge dried up in 50 per cent. of the cases.

In a number of cases the discharge comes from one or two points only, and when they are dealt with the discharge may cease. The orifice of the eustachian tube and the small group of cells in its neighborhood are one of the commonest sources of the discharge in the middle ear. If this corner can be satisfactorily reached it is cleaned out by syringing followed by application of silver nitrate either as a bead fused on a probe or as a 20 per cent. solution on a dressed applicator. Caustics must be used with

care in the middle ear as a severe local reaction may be set up, followed by labyrinthine symptoms. The part to be touched must be thoroughly cleaned and scrupulously dried so that the application will remain strictly on the spot intended. Curetting of this end of the tube is sometimes carried out but is not entirely free from risk of wounding the carotid artery below it or dura mater above it.

In other cases the mastoid cells or antrum are the chief sources of the discharge. On account of their complexity the chief difficulty in the case of the mastoid cells is lack of free drainage. At the same time artificial removal of the discharge by syringing or suction is unsatisfactory and incomplete, and antiseptic fluids rarely reach the affected areas. For this reason when an acute otitis media does not improve after several weeks and when the mastoid is a cellular one an operation is frequently advised in order to clear out the area of infection which is otherwise liable to persist indefinitely.

Intratympanic syringing by means of a slender cannula (Fig. 277) with a bent tip which is inserted through the perforation is efficacious in certain cases where ordinary syringing fails. This is true especially where the main seat of disease is in the attic or antrum. Even cholesteatoma may be washed out piecemeal and the cavity thoroughly cleaned out. Insufflation of air followed by instillation of alcohol will in many cases dry up the discharge and give a long period of freedom from the suppuration. This method can only be used by one accustomed to delicate manipulations in the ear. Granulations or a polypus when present should be removed.



Fig. 277.—Attic cannula.

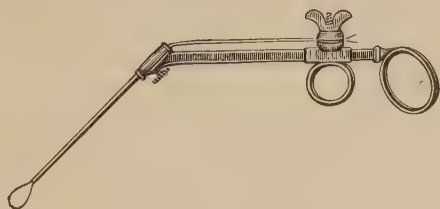


Fig. 278.—Aural snare.

They frequently shrink up during routine treatment by systematic syringing followed by drying with alcohol. If that is not enough they may be treated with caustic or curetting. An aural polypus is most conveniently removed with the aural snare (Fig. 278). Before doing so it is advisable to test the condition of the labyrinth as these growths are sometimes attached to a fistula in the labyrinth wall or even to the dura mater, and the tearing out of the stalk has been known to set up a fatal meningitis. The hearing should be tested with a buzzer in the other ear, and if absolute deafness is discovered the vestibular apparatus should be investigated by the caloric or rotation test, or both. If the tests reveal a dead labyrinth extreme caution must be taken not to tug on the stalk of the polypus in removing it. In other cases a radical mastoid must be considered.

**Removal of a Polypus.**—Local anesthesia by Bonain's or Blegvad's drops after thoroughly cleansing is generally used, although complete deadening of sensation is rarely possible. Nitrous oxide or ethyl chloride may be used in a timid patient. The slender wire of the snare is passed over the polypus as far as possible toward the stalk and drawn tight. The

growth is generally severed and may be picked out. The stalk may require to be touched with caustic to shrink it up. In many cases the discharge dries up after its removal. If after a period of thorough treatment by conservative methods the discharge persists operative treatment may be considered. When the disease is mainly in the upper part of the cavity removal of the remains of the malleus and incus may clear up the condition.

The indications for a radical or modified radical operation are: (1) Persistence of discharge in spite of treatment. (2) Attacks of pain and headache. (3) Dizziness or vomiting.

It must be recognized that the mere cessation of the discharge is not the most important aim of the operation. The danger of an intracranial complication, such as lateral sinus thrombosis, meningitis, or brain abscess, is very real in a case that is resistant to treatment, and by a radical mastoid operation all the diseased areas are cleared out and the patient is freed from danger even if the discharge should return.

### RUPTURE OF THE EAR-DRUM

The ear-drum may be ruptured by direct or by indirect violence. In the former it may happen through scratching the meatus with a hairpin or knitting-needle or through unskilled attempts to remove wax or a foreign body. During the war numerous cases were seen where the drum-head had been perforated by fragments of shell or gravel thrown up by exploding shells. In most cases the rupture of the ear-drum was accompanied by penetration of the labyrinth or brain.

Rupture by indirect violence occurs commonly in fracture of the base of the skull. The fracture may be a transverse one and run along the line of the external auditory canal across the tympanum and through the labyrinth. In such a case there is a discharge of blood and cerebrospinal fluid from the meatus. When the fracture runs sagittally it follows the roof of the middle ear cavity and may not open the labyrinth. In such a case there is blood but no cerebrospinal fluid in the meatus.

Rupture by sudden alterations in atmospheric pressure such as follow explosions was very frequently seen during the late war. It was probably more common in the artillery than in any other branch of the armies, and especially in the gunners attached to batteries of high-velocity guns or heavy howitzers. In many instances the rupture had been through a previously weakened spot from an old otitis. Many cases were also seen where the rupture had been due to shell explosions. This was often accompanied by injury to the labyrinth or commotio labyrinthi. Not infrequently the labyrinthine disturbances were more severe when the drum-head was not ruptured.

The drum-head may be ruptured through a box on the ear, and this is all the more likely if there is already a cicatrix in the membrane.

The hearing is not much impaired as a rule unless there is an accompanying labyrinthine injury.

**Otoscopic Appearances.**—There is generally some blood in the meatus. In some cases there is so much that the drum-head cannot be seen. When the rupture is due to direct violence the perforation may be in any part of the membrane, but is most common in the posterior half. In cases of rupture by indirect violence the tears are most frequently found in the antero-inferior quadrant. They are generally in the direction of either the

radial or circular fibers of the membrane and are in the form of a more or less straight slit gaping in the center. The edges are generally dark from blood adhering to them. The membrane itself may be purplish in color below the level of the rupture from the presence of blood-clot in the tympanum.

**Diagnosis.**—The history of injury, the presence of blood in the meatus, and lastly the tear in the drum-head make the diagnosis simple. Sometimes the drum-head cannot be seen on account of blood-clot, and it is inadvisable to remove it for fear of setting up infection.

**Treatment.**—If not already infected the tear in the membrane should be left alone. If the clot can be removed with forceps or a blunt ring curette without disturbing the drum-head this may perhaps be done. The ear must on no account be syringed as infection is then almost inevitable. The outer end of the meatus should be carefully swabbed with alcohol or tincture of iodine and plugged with sterile cotton wool. If no infection occurs the wound heals within a week or two. If infection has already occurred it is treated on the same general lines as an acute middle-ear suppuration.

#### TUBERCULOSIS OF THE EAR

Two types of tuberculosis of the middle ear are met with; the first in children and the second in adults suffering from advanced pulmonary tuberculosis. The second type is rare. The infantile type is common in certain geographical areas where regulations regarding the testing of cattle and supervision of the milk supply are lax. It is fortunately very rare on the North American continent. Logan Turner<sup>9</sup> some years ago showed that in Scotland 2 per cent. of all cases of middle ear suppuration attending hospital under fifteen years of age were tuberculous. Under five years of age 9 per cent. were tuberculous; under two years 27 per cent.; and under one year 50 per cent. In the majority of the last group the disease appeared within the first six months. Practically all the patients were bottle-fed babies who had been fed on unsterilized cow's milk.

The disease shows itself by discharge from the ear and enlargement of the periotic glands and swelling over the mastoid, all unaccompanied by pain. Facial paralysis is common and shows itself early. Turner found absence of pain in 92 per cent., and the first sign was ear discharge and enlargement of the glands in 95 per cent. Facial paralysis appeared in 45 per cent. Extensive necrosis and sequestrum formation occur in most cases with involvement of the labyrinth. Thus, a sequestrum was found in 60 per cent. and the labyrinth was necrosed wholly or partially in 22 per cent. Fraser<sup>10</sup> states that the disease spreads into the labyrinth in most cases through the oval or round window. The whole petrous bone may be necrosed and loose in the base of the skull.

**Diagnosis.**—The age of the patient, the history of feeding on unsterilized milk, the painless swelling around the ear with ear discharge, the occurrence of facial paralysis, and lastly the recovery of the tubercle bacillus from the discharge make the diagnosis simple.

**Treatment.**—When the disease is extensive treatment is useless. In rare cases where it is not very extensive and where the hygienic conditions are good operation may perhaps be of some benefit, but in nearly all cases the condition proceeds to a fatal termination within several months.

### SYPHILITIC DISEASE OF THE EAR

Fraser states that congenital syphilis is one of the commonest causes of deaf-mutism. In some cases this is due to basal meningitis and neuritis of the eighth nerve. In others there is a middle-ear infection which spreads into the marrow spaces, causing osteomyelitis. Apart from the congenital type syphilis has no special predilection for the middle ear, and manifestations there are of great rarity.

### TUMORS OF THE MIDDLE EAR

Primary tumors of the middle ear are extremely rare. Carcinoma is occasionally met with following an old middle-ear suppuration. Sarcoma may be of the round or spindle-celled variety and is sometimes seen in children. Both conditions are difficult to diagnose in the early stages and appear to be a chronic purulent otitis media with granulations or polypus formation. Microscopic examination is the only certain means of diagnosis.

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### THE MORBID ANATOMY OF THE TYMPANUM AND EUSTACHIAN TUBE

The tympanic cavity and eustachian tube being lined with a mucous membrane, the extension of that of the nasopharynx, it naturally shares the inflammatory processes to which the nose and throat are subject. The ear is one of the accessory cavities of the nose; and conversely the nose and nasopharynx are a vestibule to the ear and must be studied and treated if we are to do our duty in relieving ear-affections. Furthermore, the middle ear being a pneumatic cavity, dependent for its due supply of air upon the proper ventilation of these passages, there are mechanical factors of obstruction which count importantly in the causation of its disturbances. That these factors are habitually weighty was shown by Löwenberg, who noted that while two-thirds of the cases of chronic tympanic disease are on the left in men, who have the left naris the more usually obstructed; women have the same preponderance of trouble on the right, on which side in them the nose is apt to be less patulous. This is the more notable since acute affections pass more readily to the right ear in both sexes—most right-handed people having better action of the tubal muscles on the right, both in opening the tube to morbid invasion and in giving easier exit and ventilation of the right tympanum. Koerner's claim that the sigmoid sinus is larger, more anterior and superficial, and the cerebral fossa lower,

making more vulnerable relations on the right and in brachycephalic skulls, has met little confirmation by Schultske, Randall, and others; and the intracranial extensions of otitis are probably more frequently encountered on the left, where aphasia is oftener an aid to the localization of brain abscess, and a causative *chronic* suppuration, at least in males, is more usual. This "Löwenberg lateralization," as it may be termed, has its practical value when "cases against the rule" are met with. They demand extra scrutiny as to their causation—probably out of the ordinary. Another observation of Löwenberg's may be here cited. If chloroform vapor be blown into the tympanic cavity when otosclerosis is suspected, a feeling of cold rather than heat will be reported, even when the inner promontory wall displays the reddish color stressed by Schwartze as pathognomonic. It has been very positively stated by Politzer and others that changes of the tympanic mucosa are no part of the otosclerotic process. Yet it is hard to believe that the mucous lining of the tympanum is wholly normal when its redness over the promontory shines through the drum-head and its response to stimulation is the reverse of the normal.

Ignoring the primary meaning of "catarrh"="a flowing down"—if we apply the term to any mucous-membrane inflammation, even "dry catarrhs"—most tympanic affections can be called and classified as acute, subacute or chronic catarrh; distinguishing the more infectious as perforative or suppurative when the purulent exudate bursts through the drum-head or shows its yellow tint and slight fluidity as it distends this membrane. To those who claim a microbic causation of all these affections, the precise determination of the germ-form present seems more important than to the old-fashioned clinician, who judges the nature and prognosis of the attack by the pulse, temperature; and the otoscope rather than the microscope; although he cannot dispense with the blood-count or culture and other modern aids to diagnosis. Only in the unopened drum-cavity can we usually find pure cultures—most frequently *Pneumococcus*; but those who dread especially the finding of *Streptococci* as threatening carious involvement may learn that unrelievable meningitis is oftener sequent upon pneumococcic infections—perhaps non-purulent. Early incision of the drum-head may release only blood, clotting into a cast of the drum-membrane; yet the meningitic symptoms may advance despite operative attempts to secure relief by draining the middle cerebral fossa or cisterna; and subcutaneous or intravenous injections of normal saline may wash out the cranial cavities as well as any direct irrigations within the skull, which have rarely proved curative. Mastoid empyema or caries, like septic phlebothrombosis of the lateral sinus, extradural abscess or even brain abscess, has its improving lists of operated patients; but few are the cases where septic meningitis, even if circumscribed, has recovered. Prevention, then, must be our main dependence.

Passing over mere hyperemia, often cited but of slight significance except when marking otosclerotic processes at work at the promontory, acute inflammation of the drum-cavity, as of mucous membranes elsewhere, causes sharp injection, swelling, pain, and some exudate—serous or mucoïd at first—which tends to drown out the hearing by submerging the round, if not also the oval window. This fluid ought to be promptly swept down into the pharynx if not too tenacious, with relief of the painful tenseness, deafness; and tinnitus that mark its presence; except that the cilia

of the cells of the tympanum and tube are apt to be paralyzed, the patency of the tube reduced by swelling and even the tubal musculature impaired in activity. The pressure is apt to grow painful, perhaps suddenly and violently, so that the patient, especially if a child, awakes from sleep with a cry and may be for hours unconsolable. Such symptoms are the more readily understood if we remember that inflammatory processes increase toward night. The feverish, uncomfortable child is apt to be sleepy and little able to locate the pain, and after snuggling the vaguely painful ear to the pillow until relieved by its warm protection, will turn over and expose the ear unless protected by cap or bandage, more than 100° F. in temperature and perhaps moist with perspiration, to rapid chilling at the 60° F. or less of the room. A sharp spasm of vasomotor contraction is the result; and fright, as in a croup case, adds to the acute suffering. Rapid disintegration of the mucosa-cells adds mucin and leukocytes to thicken the fluid, and it may refuse to flow through a fairly ample incision; although in the earlier stages its surface may be visible through the drum-head and seen to vary as movements of the head displace it or to churn to foam as air enters it from the pharynx. Adults may tell of how instantly the pain and deafness began at a nose-blowing; but the exudate was probably already present and the added pressure has brought the painful consciousness of it. Indeed, in the many cases where the trouble has been at first an eustachian stuffing, that has prevented renewal of the air absorbed by the moist mucosa, subnormal pressure in the tympanic cavity has given rise to the exudation, preponderant outside pressure has forced in the drum-head with the painful tension felt on diving into deep water or entering a compressed-air chamber, and the sudden outward flap of the distended drum-head in response to increased intratympanic pressure may be positively agonizing. The labyrinthine pressure may share in the changes, and vertigo and other distress be thus added. Tinnitus, which is one of the usual symptoms, may be aggravated rather than relieved; so inflation, whether by Politzer's or Valsalva's method, should be gentle. In fact, the Valsalva auto-inflation with its congestive influence, especially in too vigorous or unsuccessful straining, is generally to be avoided except by ardent believers in Bier's "passive hyperemia," even if it were not putting a procedure potent for harm and overuse in the hands of the laity. Gently dealt with by detergent spray and astringent mopping of the nasopharynx, but especially by hot gargling, eustachian patency can be often more safely and lastingly restored with natural drainage. So too it is clear why atropine, with its drying effect added to its analgesic, is so much more pain-relieving than morphine or the utterly obsolete "laudanum and sweet oil," which is locally valuable only as a vehicle of heat. Physics tells us that the "specific heat" of water is double that of most oils—that the hot water (110° to 115° F.), which we can so readily instill by the pint into the meatus, carries at the same temperature twice the quantity of heat—even if, not content with olive oil, we employ that of almond, mullen, or crocodile, each supposedly more potent in proportion to its unavailability.

Heat applied to the thin drum-membrane is more potent than that upon the surface of the head; yet the dry air in the canal can convey it, free from the disadvantage of maceration, most valuably from an external electric pad, a hot-water bottle or the traditional but efficient salt-bag. Even moist heat can be borne up to 112° or 115° F.—dry heat much higher; so it, like the atropine, is the preferred analgesic because rationally and

effectively combating the inflammation which gives rise to the pain. When the inflammatory invasion cannot be thus aborted, it can at least be guided with all possible safety and gentleness through its stages to resolution, and needs as a rule but a few careful inflations to restore the catarrhal tympanic structures to full physiological function. Any infiltrations of the drum-head, the ossicular joints, or adventitious bands that may impede the sound-conduction can generally be absorbed or modified by pneumatic measures, including the use of Siegle's pneumatic otoscope: and when this instrument has shown that blind massage will be safe, the pneumatic play of the finger-tip in the meatus or the palm placed against the auricle furnishes ready and efficient means of relief and is devoid of the too rhythmic or vigorous action of more mechanical appliances. Employed *per tubam* alterative vapors such as iodine or solutions like dionine 2 per cent. can be potent in absorbing scar-tissue. Use of thiosinamine has usually fallen far short of the gain expected of it; although in capsule with bismuth tri-iodide (cirrhohysin) it has seemed not only harmless but helpful.

Turning to the suppurative inflammations, these almost always entail perforation of the drum-head and by ulcerating the tympanic lining, which is the mucoperiosteum of the walls and ossicles, they lead to carious destruction of the auditory apparatus and dangerous penetration to the inner ear, the facial nerve, the sigmoid sinus, or the brain cavity. The meningitic extensions, except through the internal auditory meatus, are generally limited by the dura, which may be covered with pachymeningitic granulations yet present a normal aspect on its arachnoid surface. Yet even through such an unopened brain membrane toxic, or possibly septic, penetration can occur. Within the tympanum and the communicating pneumatic spaces—which are by no means limited to the mastoid—the infected mucosa can fill all cavities with purulent fluid and the swelling, uniform or poly-poid, of the lining. This is *empyema* not abscess, unless the nutrient function of the mucoperiosteum is lost, or pressure breaks down the walls to form cavities not pre-existent. Safe drainage by natural channels has been rarely proved; yet it surely does occur far oftener than is generally believed. Drum-head perforation and exit, spontaneous or by incision, is so easy that we are properly distrustful of adequate drainage otherwise; and we are ready, so soon as this is not surely enough, to surgically open the mastoid. Yet large clinical experience recognizes a great proportion of spontaneous resolutions, even after most disquieting symptoms. Under these circumstances we must believe that the resolution, which we can watch through an extensively perforated drum-head or a widely chiselled mastoid, proceeds most smoothly under cover; and cure may be as complete and trustworthy in a week or two as it would be in double that time after operation. The suppuration becomes a mucoid or merely serous exudation; the red swelling diminishes; and soon normal relations are restored. Yet here, as elsewhere, subinvolution can occur—a mucous discharge may continue indefinitely—indeed, a stringy flow usually warns us of an obstinately chronic purulency. In some such cases when pressure in the meatus fails to force our medication, even  $H_2O_2$ , through the drum-head perforation to the persistently suppurating area, medication through the catheter and eustachian tube should be tried before turning to operation.

With the cell-infiltration of the tympanic lining which accompanies purulent, and even severe catarrhal invasion, secondary changes are prone

to occur. The round cells may become fusiform connective tissue, causing fibroid thickenings of the drum-head or other structures; and organizing surface-exudates may form or thicken adventitious bands or adhesions to hamper mobility or impair drainage. The in-pressed drum-membrane may attach itself to the opposite wall, from which its funnel-like center is normally but little distant. The tendon of the tensor tympani may be sclerotically shortened and the drum-head become actually "indrawn," as it is oftener erroneously called. The areas infiltrated with cells of poor vitality may be seats of calcareous deposit—very rarely of true bone; and such changes in the niche of the oval window may give true bony ankylosis of the stapes—a great, possibly a total, obstruction to transmission of sound-waves. Drum-head perforations of long standing are apt to show distinct loss of substance of the membrana propria. They may heal, but usually with flaccid scar-tissue which ill-replaces the more tense membrane and often so collapses into contact with the inner structures that even experts regard the opening as unclosed. Such depressed cicatrices may blanket the stapes and greatly impede its mobility until held away. Multiple incisions have been used to stiffen them into better maintaining their proper plane, or McKeown's painting with contractile collodion may fully accomplish this. The openings may refuse to heal, although the discharge has ceased; and temporary closure may show that they impair the function of the drum-membrane in conveying low tones. Blake's paper patch may then serve not only to show the benefit to the hearing that is to be expected from their closing, but to promote its prompt attainment. Perforations of half the area of the drum-head may heal spontaneously or with such aid—the patch serving to stimulate the margins to reparative activity and as a scaffolding to support the bridging tissues until capable of self-maintenance. Such closures may cut off the penetration of the highest tones, which are somewhat impeded by even the normal drum-head (Blake); but this rarely affects the highest sibilant sounds of speech; and the theoretical loss is more than offset by the protective value of the closed drum-head and the probable betterment for low tones. The normal moist mobility of the tympanic structures is threatened, if not lost, when drum-head perforation permits of dryness of the cavity; just as we must warn our otorrhea patients that the hearing may fall off disturbingly when first we arrest the discharge, lest they desert in the belief that we are mistreating their ears, which will soon gain again under the (better?) treatment or inaction of some one later consulted.

Marginal perforations, more than those located elsewhere, have a tendency to remain open. Instead of bridging the gap, the reparative cells, derived mainly from the external epithelium, skin-over the margin and maintain a permanent opening. Far worse than this, they invade the tympanic cavity, grafting squamous epiderm upon the succulent mucosa. Already one of the most rapidly proliferating tissues of the body—fitted by Nature in their growth from the center of the drum-head to move glacier-like across the periphery and out upon the walls of the canal, carrying with them any extraneous matter and even their own extravasations, keeping the drum-head clear of any clogging débris—they grow still more rapidly within the drum-cavity. Here they tend to form flakes of pearly epiderm compacting into laminated pellets, which Nature has no normal method of removing. Like a make-believe republic—"a despotism tempered by

insurrection"—the tympanum can rid itself of such an incubus only by washing it out with pathological secretion or pushing it out by granulation-growth. If successful at all, this leaves the matrix there to renew the process periodically. Therefore the writer stated in 1881, "The cause of chronic or recurrent otorrhea is cholesteatoma"—a rule having few exceptions. Most perforations in the flaccid Shrapnell membrane have behind them an attic or at least a Prussack pouch packed with these white leathery flakes. Opening up the region by excising the drum-head and ossicles or by more radical evisceration of the three tympanic cavities has been claimed as the only way to cure these obstinate cases. The former operation has been generally abandoned in favor of "the radical"! Yet while Schwartze claimed 74 per cent. success by evisceration in followed-up cases, many of these were radically operated upon immediately, without trial of really conservative treatment; and many of our newer men claiming even better results have not followed them adequately for convincing report. Actually hardly 50 per cent. seem to have been cured by operation: and this is better than the results gained by conservative measures only in so far as the cases were such as had already resisted milder treatment. The vicious circle can be broken only by periodic housecleaning with curet and probe—and this almost as much in the sufferers of "radical cure" as in those treated with old-fashioned patience. The walls of the attic and antrum having been removed, the flakes ought to utilize the free exit afforded and extrude themselves. In far too many cases they do not, but need periodic removal.

The intricate cavities of the three-fold tympanum and the adjacent cells—more often those at the tympanic tube-mouth than in the mastoid—should be at least more open to our cleansing and medicating after operation; but we may find that we have only enlarged the area of pathological exfoliation and secretion, and may have notably reduced the hearing—possibilities without any commensurate gain. Yet there will always be patients as well as surgeons who will prefer cutting of the Gordian knot to any slower (?) effort at its disentangling; and the resulting palsies of the facial nerve, which no skill can wholly avoid, may have been inevitable—only precipitated, not caused, by the surgical intervention.

B. ALEX. RANDALL.

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## CHRONIC GRANULOMATA OF THE EAR

**General Remarks, Histology, Bacteriology, and Pathology.**—The type most frequently seen in the tympanic cavity, external canal, and internal meatus is the non-specific granuloma (Fig. 279).

Chronic non-specific granulomata in the mastoid antrum and cells are also quite common. They spring from the diseased surfaces of the antrum, mastoid cells, or epitympanum. They may be single or multiple. The mass at times becomes so large that it invades the tympanic cavity, and may extend through a perforation in the membrana tympani into the external canal. Large-sized masses of this kind are sometimes pedunculated, and may take the form of polypi, myxomata, and fibromata. In the mastoid

the non-specific granuloma is the type most commonly seen during mastoid operations. Tuberculous and syphilitic granulomata are rarely found in the mastoid. Non-specific granulomata, as well as the tuberculous and syphilitic varieties, also occur on the tympanic membrane and in the eustachian tube.

One variety of non-specific granuloma has an irregular granular surface and bleeds readily when touched with a probe. It is of fairly common occurrence in the tympanic cavity and external meatus, and is the form usually associated with middle-ear suppuration of long duration. It is also frequently present in cases of caries in the tympanic cavity.



Fig. 279.—Chronic non-specific granuloma of the tympanic cavity projecting into the external canal. This patient developed a mastoiditis and the mass in the tympanic cavity was removed during the mastoid operation. The growth in the canal had been previously removed and on microscopical examination was found to consist largely of granulation tissue. Microscopical diagnosis of granuloma of the middle-ear mass also. No growth was found in the mastoid antrum or cells. Macroscopically the growth suggested sarcoma and that diagnosis had been made before sending the specimen to the laboratory. (Author's case and drawing.)



Fig. 280.—Microscopical section of a non-specific granuloma or granuloma simplex from the external auditory meatus. It extended through into the external canal from the tympanic cavity. Shows the typical large and small lymphocytes. This granulation tissue was evidently in a state of acute inflammation. As these granulomata are usually surrounded by an infected area a purulent secretion sometimes penetrates through to the surface. (Author's case and drawing.)

The pedunculated variety, which may properly be called a polypoid granuloma, has a smooth surface caused by one or more layers of epithelium forming a sheath. It differs, however, from the true polyp in being free from glandular and other elements associated with a mucous membrane. It also usually indicates diseased bone.

Histologically, the non-specific granuloma of the tympanic cavity and meatus consists of lymphocytes held together by a matrix of globulin. After hardening it appears as a delicate meshwork (Fig. 280). Thin loops of capillaries run through it, and giant-cells are occasionally found. This form of granuloma, just as in the pharynx, may be associated with any form of bacterial infection—pyogenic or non-pyogenic. The polypoid form of granuloma of the tympanic cavity sometimes takes on a rapid increase in

size, mainly caused by an increased development of the blood-vessels. There is usually associated with this a serous or mucous edema.

The granuloma pyogenicum also occurs in the tympanic cavity as a fairly large pedunculated tumor, although not always pedunculated, and is dark red in color.

Histologically staphylococci are found implanted in the intima of the blood-vessels and as a result granulation tissue forms. It is not a fungus but practically always the result of a *Staphylococcus aureus* infection.

The tuberculous granuloma, a much rarer form in the tympanic cavity and meatus, has some characteristic features histologically (Fig. 281). Lymphocytes and a well-marked activity of the mesothelial elements are essential features of this form of granuloma. The mesothelial elements replace the lymphocytes and form solid masses of tubercles surrounded by a zone of lymphocytes. At or near the center giant-cells are usually found. Tubercle bacilli are occasionally found. It contains fewer polymorphonuclear leukocytes than the non-specific granuloma.

The syphilitic granuloma in the writer's experience is next in frequency to the non-specific form of granuloma already described. Microscopically it differs from the other forms by its marked perivascular lymphocytes. Leukocytes are found in the loose connective tissue mixed with lymphocytes. *Spirochætæ pallidæ* are also found. There is a thickening of the intima of the smaller blood-vessels.

The non-specific form of chronic granuloma in the tympanic cavity almost invariably indicates underlying disease of the bone. When granulomata return promptly after removal and become very exuberant a usually extensive bone necrosis is indicated.

A chronic mastoid condition, which often flares up during acute middle-ear attacks in such cases, is sometimes associated with extensive formation of granulomata in the tympanic cavity. The diagnosis of chronic mastoiditis in such cases can usually be verified by a roentgenograph.

The lupus granuloma shows a slighter tendency to caseate and a greater tendency to sclerose. Giant-cells, irregular in shape, are found. Tubercle bacilli are absent.

**Etiology.**—As has been already stated, practically all non-specific granulomata of the tympanic cavity or meatus are the result of a bacterial infection or bone caries. In rare instances a small polypoid granuloma

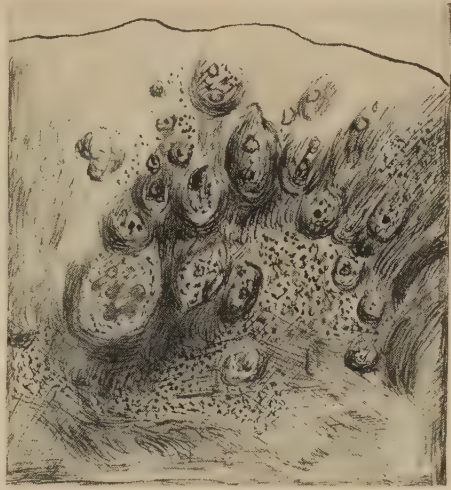


Fig. 281.—Microscopical section of a tuberculous granuloma from the external meatus. This patient had advanced pulmonary tuberculosis. The granuloma extended into the external auditory meatus from the tympanic cavity. Shows some epithelioid and giant-cells and tubercles. Also shows a tendency to beginning caseation differing in this respect from the lupus granuloma which, as a rule, has a tendency to become sclerotic. Allowing for imperfections in the author's drawing, this is the typical microscopical picture of tuberculous granulation tissue. (Author's case and drawing.)

in the tympanic cavity may occur as a primary growth without evidence of suppuration. It is a question however, even if no suppurative process is going on at the time the growth is seen, whether it was not the result of a previous infection.

When a non-specific granuloma does develop in the tympanic cavity, with a chronic inflammatory process without previous suppuration, or in rare instances as a primary growth, it may *cause* the caries and aural discharge. It has been a disputed point in the etiology for years whether such granulomata were the result of or caused the underlying pathological conditions.

The writer is of the opinion that in the majority of the cases the granuloma is the *result* and not the cause of the caries and discharge. A lupus granuloma on the tympanic membrane may be part of a lupus vulgaris of the face, the result of an extension through the external auditory canal.

Tuberculous and syphilitic granulomata in the eustachian tube are local lesions of those diseases and are never primary.

When large masses of granulomata persist in the tympanic cavity it practically always means bone caries associated with a chronic aural discharge. The caries can usually be detected with a probe.

Syphilitic granulomata of the meatus or in the tympanic cavity are manifestations of tertiary syphilis and often develop on the site of healed secondary lesions.

The tuberculous granuloma is rarely if ever primary in the middle ear or meatus. It is always observed during the course of a general tuberculosis with a tuberculous middle-ear condition.

The granuloma pyogenicum is caused by a *Staphylococcus aureus* infection.

In the mastoid antrum and cells the non-specific granuloma is the result of a suppurative process usually a chronic mastoiditis, or in rare instances may be primary and cause the mastoid condition. A gradual pressure erosion, if the mass is large, sometimes causes a destruction of the entire cellular structure of the mastoid process converting it into a large cavity filled with the growth.

The etiology of the true lupus granuloma of the tympanic cavity, which may be primary in extremely rare instances, is rather uncertain. It may occur with a lupus vulgaris of the face. It is rarely seen, and may be mistaken for the tuberculous granuloma, particularly if genuine tubercles are found microscopically.

**Diagnosis and Differential Diagnosis.**—The differential diagnosis of genuine tuberculous granulomata, and lupus granulomata containing tubercles, of the tympanic cavity and meatus is sometimes almost impossible clinically, and can only be decided by microscopical examination. There are certain macroscopical differences which distinguish the two. The color is much the same—a grayish red. The lupus granuloma is more nodular, firmer to the touch, showing a much slighter tendency to ulceration, and usually smaller in size. The tuberculous granuloma is softer, more granular in appearance, ulcerates quickly as a rule, and has a much more exuberant growth.

Microscopically the diagnosis is easily made, the main points having been already given in the part of the paper on histology and bacteriology. The important differences are the typical, sharply defined, centrally located,

Langhans type giant-cells, the presence of tubercle bacilli and tubercles caused by these organisms in the tuberculous granuloma, while in the lupus granuloma tubercle bacilli are absent and the giant-cells are more irregular in shape. Tubercle bacilli are sometimes found in the aural discharge in cases of tuberculous granulomata.

The syphilitic granuloma is more sharply defined than the tuberculous. It is soft and spongy to the touch, bleeds readily, and breaks down more rapidly.

Microscopically it is distinguished by its marked perivascular lymphocytes. A positive Wassermann and the evidence of constitutional syphilis will make the diagnosis positive. It is a tertiary lesion. *Spirochætæ pallidæ* can usually be found.



Fig. 282.—Showing tumor mass presenting after incision through skin and periosteum. Mass proved to be a granuloma on microscopical examination and filled the mastoid cavity. Pneumatic structure, cortex, and dural plates destroyed by pressure erosion. The mass was found to be attached to the dura of the temporosphenoidal lobe. When operation was extended the tympanic cavity was also found filled with the mass. When the usual mastoid incision was made the knife entered a large cavity as shown in the drawing (slightly large scale), which was completely filled by the tumor mass. The mastoid process had been converted into a large cavity, the entire cellular structure being destroyed, the tumor mass taking its place. (Author's case and drawing.)

The granuloma pyogenicum of the tympanic cavity is of bacterial origin occurring with *Staphylococcus aureus* infections. It may also be associated with caries. Clinically it is distinguished from other granulomata by its fairly large size, its usually dark red color, and its soft granular structure. It bleeds readily when touched with a probe. It resembles in its macroscopical features the granuloma simplex, and a positive differential diagnosis is difficult. Microscopically, the main findings are the presence of staphylococci implanted in the intima of the blood-vessels in the granuloma pyogenicum, while in the granuloma simplex large numbers of lymphocytes are found. Granulation tissue is of course a main microscopical finding in both.

The non-specific granuloma simplex of the tympanic cavity, mastoid cells, and internal meatus is also distinguished by its very exuberant growth,

its rapid recurrence after removal from the tympanic cavity unless radical operations are performed, its irregular granular surface, and soft spongy consistence. In cases in which the tympanic membrane has been destroyed it often projects into the external auditory canal.

The diagnosis of this form of granuloma can usually be made positively by its association with middle-ear caries and chronic aural discharge. It bleeds readily when touched with a probe, and is usually yellowish red in color. This variety of granuloma has a more massive growth than any other form. It will persist as long as there is any middle-ear caries or aural discharge.

In connection with the diagnosis of non-specific granuloma simplex of the mastoid the writer presents the following case seen by him several years ago:

The patient a five-year-old child had had a discharging left ear for more than a year. The mother stated that this followed a bad cold, and that she had never consulted a physician for the aural discharge. For five or six months before consulting the writer the mother noticed a gradually increasing swelling back of the ear. The child in the meantime had felt pretty well and had been going to school. When seen by the writer the auricle was pushed far forward, and there was a swelling behind the ear as large as an egg. It looked, and on deep palpation felt, like a typical subperiosteal abscess. There was a decided sagging of the posterosuperior wall of the external auditory canal, and a profuse discharge from the ear.

**Operation:** After the skin incision a large reddish mass was immediately seen which completely filled and occupied the site of the mastoid (Fig. 282). This was carefully removed and we then discovered that the mass had been attached by a broad base to the dura of the temporosphenoidal lobe. The bony dural plates had been destroyed by pressure erosion. The operation was then extended and the middle ear was also found filled with the tumor mass which was removed. It was considered best not to follow out the subsequent steps of the radical operation. The wound was two-thirds closed with interrupted silkworm sutures the cavity having been lightly packed with vioform gauze. The young patient had a perfectly normal convalescence and two months after the operation had a dry ear. The posterior incision healed perfectly. Laboratory report of the tumor examination showed that it was composed of granulation tissue probably a true granuloma.

A similar case has been reported by Dr. John D. Richards. This case also simulated a subperiosteal abscess. The patient was a one-year-old child. At the operation a grayish yellow tumor mass occupied the site of the mastoid, completely filling it. The tumor had as a base the dura of the temporosphenoidal and cerebellar lobes. The dural plates had been destroyed. It was found that the mass had also invaded the middle ear. Dr. Jonathan Wright reported that the tumor was composed of granulation tissue only.

**Symptoms.**—The two symptoms that are always present in all cases of granuloma of the tympanic cavity are the chronic aural discharge and deafness. The discharge occasionally stops for a considerable time, but any acute inflammatory process in the middle ear such as an infection via the eustachian tube during acute colds or sore throats will start it again. While the aural discharge is going on there is, as a rule, little or no pain in the ear. In all cases there is a large perforation through the tympanic membrane, and in some the membrane is entirely absent. In such cases the granuloma in the tympanic cavity can be seen and probed. Caries also can usually be detected by probing, and when this is present there is a bad odor to the discharge. As granulomata in the tympanic cavity indicate a chronic condition with diseased bone, a chronic mastoiditis is also present in many of these cases.

When there are no clinical evidences of chronic mastoiditis a positive

roentgenograph will indicate the mastoid involvement. A roentgenograph should be taken in every case of granuloma of the tympanic cavity. A chronic mastoiditis is a more frequent complication in children than in adults. There is as a rule, little if any temperature elevation except in the cases in which an acute flare-up of the chronic mastoid or middle-ear condition occurs, or if there is a secondary meningitis. A chronic mastoiditis must always be considered when a granuloma can be seen in the tympanic cavity.

The development of a granuloma in the mastoid antrum and cells may go along for a long time without any special symptoms pointing to a mastoid involvement. Unless a blood-count is made and a roentgenograph taken it may be easily overlooked.

In cases in which the granuloma has destroyed the cellular structure of the mastoid process and has pushed its way through to the periosteum, the postaural swelling will, of course, be a symptom pointing positively to the mastoid condition. When the tympanic cavity is full of a granulosomatous mass it will at times project through into the external canal.

In tuberculous granulomata there is usually considerable intermittent earache, and a foul-smelling aural discharge, bloody at times, in which tubercle bacilli are sometimes found microscopically. Evidences of general tuberculosis are practically always present in such cases.

In syphilitic granulomata of the tympanic cavity there is as a rule little earache. The aural discharge is a prominent symptom and there are symptoms of constitutional syphilis. Granulomata of the ear are usually confined to one ear although both may be involved. The loss of hearing, which varies depending upon the amount of middle-ear destruction and the size of the mass, is always a constant symptom. In cases in which the middle-ear involvement is considerable, as with extensive caries or ulcerative processes attending tuberculous and syphilitic granulomata, deafness of the affected ear may be complete.

In some cases of specific (mainly syphilitic) or non-specific granulomata of the tympanic cavity, there is an extension of an ulcerative or inflammatory process into the labyrinth, causing the typical Ménière symptom-complex. The disturbance in equilibrium may be particularly marked, so much so that the patient sometimes falls. This is at times accompanied by a brief period of unconsciousness.

Ulcerating granulomata in the eustachian tube result in strictures and at times occlusion of the tube, with the accompanying middle-ear involvement and deafness.

Tuberculous and syphilitic granulomata on the tympanic membrane finally cause a destruction of the membrane. There is always a good deal of pain and aural discharge while the ulcerative process is going on. An extension of the ulceration into the middle ear results in either partial or complete deafness.

The tuberculous granuloma on the tympanic membrane is associated with pulmonary tuberculosis, and the syphilitic granuloma is a tertiary lesion of constitutional syphilis. When a syphilitic granuloma on the drum-membrane breaks down, the ulceration may extend into the external canal, at times causing a good deal of tissue destruction resulting in cicatrices and more or less occlusion of the canal.

In cases with extensive middle-ear caries and mastoid involvement a

meningitis or brain abscess may result. The infection extends through the tegmen or by way of the middle fossa. A septic meningitis of this kind is practically always fatal. A complicating brain abscess is usually in the temporosphenoidal lobe, although in some cases there is a circumscribed abscess in the cerebellar lobe.

Granulomata of the ear occur at all ages. They are rather more frequently seen in young adults than in children. The youngest case of which the writer could find any record of a granuloma of the mastoid process is the case reported by John D. Richards—a one-year-old child.

**Prognosis.**—The prognosis of all forms of non-specific granulomata of the tympanic cavity and mastoid process may be said to be favorable, provided the condition is promptly recognized and appropriate surgical measures taken. In cases in which there is a good deal of middle-ear caries with a complicating chronic mastoid involvement the prognosis is not so good, that is so far as the subsequent hearing of the affected side is concerned. As the radical operation must be performed in all such cases with a thorough cleaning out of the middle ear, there is usually fairly complete loss of hearing. In many cases this existed before the operation.

In cases that are seen early with no mastoid complication, little middle-ear caries, and a granuloma limited to the tympanic cavity, more conservative surgical measures may be used and the patient promised some restoration of hearing.

Some cases with extensive middle-ear destruction and mastoid involvement have fatal terminations.

A meningitis, which is not an infrequent complication, is practically always fatal, although subarachnoid irrigations offer a slight chance. Temporosphenoidal abscesses which sometimes follow granulomata are much more hopeful. Modern brain surgery saves many cases of this kind formerly considered hopeless.

Labyrinth involvement unless suppurative also offers a favorable prognosis, particularly in regard to the cure of the disturbances in equilibrium.

Tuberculous granulomata are absolutely unfavorable from a viewpoint of prognosis as they are always lesions of constitutional tuberculosis.

Syphilitic granulomata are all curable if the constitutional disease can be cured. The same favorable prognosis may be given in regard to the ultimate restoration of a considerable amount of hearing in the syphilitic cases.

In all other cases of granuloma of the tympanic cavity with extensive destruction a very guarded prognosis so far as hearing is concerned should be given.

**Treatment.**—In all forms of granulomata of the tympanic cavity, meatus, external canal, or mastoid process, except the syphilitic form, treatment may be summed up in one word—surgical. The proper operation should be performed at once, followed by treatment for the aural discharge if it does not stop after the operation. If it does not it usually means that some diseased bone in the mastoid process or some slight area of caries in the tympanic cavity has been overlooked.

It goes without saying that in all cases of granulomata of the tympanic cavity in which definite evidence of a mastoid involvement has been determined clinically or by a roentgenograph, the radical mastoid operation should be performed.

Syphilitic granulomata can be cured by pushing iodide of potash to the limit of tolerance. In connection with the general treatment granulations in the middle ear may be destroyed with chromic acid or a 5 per cent. solution of acetic acid, care being taken to limit the action of the acid. Strong solutions of silver nitrate are also useful for this purpose. For the aural discharge prior and subsequent to operative measures, irrigations with lysol (a teaspoonful to the quart of warm water) or with a 1 : 5000 solution of bichloride of mercury are the most useful. The author has had the best results with bichloride solutions and uses practically nothing else. Dakin's solution has not been as successful in the hands of the writer in the treatment of the aural discharge. In cases in which there is no mastoid complication, granulomata of the tympanic cavity may be removed via the external canal. This is easily accomplished as the tympanic membrane has been destroyed in the majority of the cases. After the growth is removed, strong solutions of silver nitrate or a 4 per cent. chromic acid solution, are useful to prevent a return of granulation tissue.

A complicating septic meningitis as already stated, is a hopeless proposition so far as treatment is concerned. Surgical measures if the meningitis is localized, and frequent lumbar punctures in very rare cases may be successful. The number of cures recorded in the literature are very few however.

In chronic granulomata in the tympanic cavity and mastoid in which there is extensive caries, circumscribed abscesses may develop in the temporosphenoidal lobe, with a fair percentage of recoveries following modern operative methods.

Labyrinthine complications, the result of granulomata in the labyrinth or from an extension of an inflammatory process from middle-ear granulomata, are amenable to treatment so far as special symptoms are concerned. For example much can be done for the typical Ménière symptom-complex particularly for the disturbance in equilibrium. Iodide of potash and quinine in small doses are specific remedies in many of the cases.

In the writer's experience potassium iodide in small doses is particularly effective in the treatment of the vertigo and other labyrinthine symptoms occasionally associated with middle-ear granulomata. A saturated solution of iodide of potash in elixir of lactopeptin is used, beginning with 5 drops three times daily and increasing the dose 1 drop a day until 10 drops are taken. If a tolerance for the iodide has been established the patient is kept on 10 drops for a considerable time. In many cases this treatment is absolutely successful.

For the tinnitus, which is such a stubborn symptom in many cases of granuloma of the tympanic cavity, there are unfortunately very few effective methods of treatment. It will sometimes disappear after the granuloma has been removed from the middle ear and the aural discharge stopped.

Syphilitic granulomata in the eustachian tube will disappear if large doses of iodide of potash are given. The treatment should be stopped occasionally on account of the danger of laryngeal edema, which may suddenly develop if potassium iodide is pushed too steadily.

In very rare instances non-specific granulomata in the tympanic cavity show a tendency to become malignant. This is apparent clinically by the firmer consistency of the growth and the severe pain. When this occurs radium therapy will sometimes result in a disappearance of the growth which

may not recur for a long time. Radium therapy is sometimes successful for granulomata in the external auditory canal.

Granulomata in the eustachian tube are difficult to treat surgically. If they are situated in the pharyngeal ostium of the tube they can be reached with the aid of the illumination from the Holmes' nasopharyngoscope.

The treatment of tuberculous granulomata is unsuccessful as they are practically never primary, but develop as a rule in conjunction with advanced pulmonary tuberculosis. The general condition is so bad that no local measures are very successful unless the patient is sent to a proper climate.

The removal of tuberculous granulomata from the middle ear by curettage and applications of strong solutions of lactic acid is occasionally followed by temporary improvement. Very little can be done for the loss of hearing which is present in nearly all cases of middle-ear granulomata. In some cases there is a gradual improvement after the granuloma has been removed and the discharge from the ear stopped. Treatment is particularly unfavorable if there has been a long aural discharge and in cases in which there has been a good deal of destruction in the middle ear.

The illustrations in this article are from drawings made by the author while patients were under observation.

CLEMENT F. THEISEN.

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## THE FUNCTIONAL TESTING OF HEARING

Functional testing of hearing is done with a number of purposes in mind, among which are: (1) Determination of the presence of impaired hearing and the degree thereof; (2) definite localization of the hearing defect, that is to say, whether it is in the inner ear, in the middle ear, or in both; (3) to learn whether there is total absence of hearing in one or both ears; (4) to uncover simulated unilateral or bilateral deafness. Testing is done by means of the whispered or spoken voice, or the employment of a number of instruments and appliances such as the watch, acoumeter, tuning-forks, resonators, whistles, monochord, and audiometers.

Before proceeding with the examination of the ear and its functions, a general inspection of the patient is of value. Much may be learned regarding the state of the individual's hearing by observing him and his actions. The loudness of the voice is of aid in differentiating between impairment of the conduction and the perception apparatus. When there is marked middle ear deafness, as for instance in an otosclerosis or a chronic tubal catarrh, the bone conduction is materially increased, and the patient has an auto-phonia—that is to say, his own voice seems very loud to him. Believing that it also sounds very loud to others, he usually speaks very low. On the other hand, where there is a marked involvement of the perception apparatus the bone conduction is very materially diminished and the patient's voice sounds very weak to him. To make others understand him, he is likely to speak very loud. When the hearing is markedly impaired, one often notices a peculiar attitude of the head in that the patient inclines the better hearing ear to one side, or the hand is placed at the back of the auricle

to improve the hearing. Paul E. Sabine proved through experiments that the hand serves practically as well as a collector of sound and as a resonator as does any except the largest ear trumpets or horns. Furthermore, one often notices that those who have impaired hearing watch very closely the lips of the speaker, either consciously or unconsciously doing a great deal of lip reading, in an effort to understand the spoken word.

**Otoscopic Examination.**—It is necessary that the auricle, the mastoid region, the external auditory canal (*meatus auditorius externus*) and the drum-membrane (*membrana tympani*) be carefully inspected, to note if there are inflammatory swellings, tumors, etc., externally present. The meatus itself should then be examined to see if there is any obstruction or actual occlusion due to acute inflammatory swellings (either circumscribed or diffuse *otitis externa*); if pus, cerumen, foreign bodies, or an exostosis are present in the canal. The mastoid region should be inspected to see if there is redness, infiltration of the overlying structures or tenderness at certain points. The drum-membrane itself should be carefully examined particularly as to luster, color, mobility, or the presence of hyperemia, retraction, thickening, scars, or perforations. If the latter changes are noted, then the condition of the mucosa of the middle ear should be studied with reference to hyperemia, pallor, presence of granulations (commonly known as “polyps”), obstruction or loss of part of the ossicles, and the presence of secretion—purulent, mucoid, or serous.

Examine the nose, and note (1) the condition of the mucosa, (2) the presence of hypertrophy of the turbinates, especially that of the posterior end of the inferior turbinates; (3) see if there is a marked deviation of the nasal septum, and (4) whether tumors or polypi are present. In the nasopharynx, observe if adenoids and secretion are present, and if there is any change in the orifice of the eustachian tube or anything interfering with the tube's aëration. In the pharynx note the size of the tonsils, the presence or absence of caseous plugs or purulent material in the crypts, and the presence of any adhesions between the tonsils and pillars. Note if there are large lymphoid masses in the posterior wall of the pharynx and if there is inflammatory reaction of the mucosa.

To determine the presence of any rests of hearing, a number of reflexes are employed. Some of them are of greatest use in young infants in whom it is difficult of course to make any functional hearing tests. Three reflexes are in most common use, namely, the acoustical pupillary reflex, the general acoustical muscular reflex, and the acoustical palpebral reflex. The pupillary reflex consists of a dilatation of the pupil when a tuning-fork is struck quite hard and held near the ear. This reaction is found in a great many adults, but must be observed in good daylight and the patient instructed to look directly forward beyond the ear of the examiner. The general acoustical muscular reflex consists of a sudden muscular movement of the body musculature, and can usually be observed within a few hours after birth. The palpebral reflex is also a very valuable procedure especially in infants. It has been shown that this reflex is present within twenty-four hours after birth. It is essential that the child be awake, but quiet at the time, and that a shrill high-pitched sound be produced directly behind the patient's head, preferably by striking a  $c^4$  fork (2048 double vibrations) with a metal hammer. When the test is positive there is a sudden movement of the eyelids. As a rule this reflex can only be tried once in a day as

repeated performances fail to elicit it. Using a high-pitched fork, however, does not allow the exclusion of the opposite ear since the tones are carried over, and with an infant of course it is impossible to occlude the opposite ear without attracting his attention. Therefore, the test is only of importance in determining if there is any hearing whatsoever present, irrespective of one or the other ear. With older individuals it is possible to occlude the opposite ear, and while it is not as a rule possible to really prevent all the sound from entering the closed ear, the test is nevertheless of considerable value.

The **Politzer acoumeter** is a small instrument so arranged that it is possible to allow a hammer to strike a horizontal metal bar. This apparatus may be used both for bone and for air conduction. If the latter, the patient's eyes are averted and the opposite ear closed, the examiner approaches the ear that is being tested and clicks the apparatus until the sound is heard and the distance noted. This was assumed to be a very accurate instrument in that a sound with practically the same intensity was produced each time, thus creating a standard for comparison. We know, however, that the pitch of the acoumeter is higher than some of the sounds used in speech, and, therefore, the patient may be able to hear the high pitched tones, and still be quite deaf for the voice, or for other lower pitched sounds. Furthermore, a pure tone alone is not produced by this apparatus, but a tone and some noises. It might be stated here that a tone is a sound produced by a certain definite number of vibrations, giving it a musical quality, whereas a noise is often a mixture of many sounds of irregular vibrations.

The ordinary **pocket watch** may also be used for hearing tests. The method is the same as with the acoumeter, namely, closing the opposite ear, and approaching the ear that is being tested until the patient hears the ticking. While the watch is only a rough measure, it will often serve the otologist who has not a large instrumentarium, for if he uses the same watch on all patients, he obtains a criterion of the relative hearing acuity. The watch, however, is not a very dependable apparatus in that the ticking of no two watches is alike and, therefore, the results obtained by one examiner are not comparable with those reported by others. Furthermore, the watch produces a great variety of noises and is, therefore, subject to the same objections as the clicking of the acoumeter.

The **voice** is largely used to test the hearing acuity, the unaccentuated whisper and unaccentuated conversation being employed. In the small rooms in which most otologists in the larger cities must carry out their examinations, it seems perhaps better to test the patient first with the whispered voice. If that is not heard, then recourse may be had to the conversational tone. It is important *not* to accentuate, as otherwise the sound is carried further and it is more difficult to get an accurate estimate of the patient's ability to hear. For this reason it is better to speak with the residual air, placing no emphasis upon any particular sound or syllable. We believe that the better hearing ear should be tested first. With one ear tightly occluded, either by means of wet finger, cotton, or noise apparatus, and the eyes closed or averted so that he is unable to read the lips of the examiner, approach the patient gradually and note the distance at which the numbers, words, etc. are properly repeated. The patient must not touch the walls, doors, etc., since the sound-waves may be transmitted from them to the ear by way of the bones of the arm or other parts of the body. Use

high pitched and low pitched sounds individually, or numbers combining the two, as for instance, forty-seven which includes both a low- and a high-pitched sound. In English all numerals are low pitched except six and seven. Note the distance at which the high or low or both pitches are heard. It is very important to approach the patient from the distance when examining him with a watch, with the acoumeter, with the voice, and the other appliances to note the distance of hearing. One should not produce the sound close to the patient and then step away from him, because it is a psychological fact that as we recede he will from memory often continue to say that he still hears a certain sound when he no longer does. If you approach him from a distance a much more accurate estimate is had.

In order to be quite certain that the patient hears the sound in the ear which is being tested a two or three meter or even longer tube is used, inserting one end into the patient's ear and whispering into the other end. The sounds are thus carried only through the lumen of the tube to the examined ear, and if repeated must have been heard by the patient by way of that ear.

Having thus tested the patient's hearing and noted the results, the ear should be inflated either with a Politzer bag or, preferably, by means of a eustachian catheter, and the degree of hearing again tested. The presence or absence of an improvement in hearing following the careful inflation is an important index in consideration of the prognosis. If there is a decided improvement it usually means that further treatment will greatly benefit the patient. If on the other hand no improvement whatever is noted (especially after inflation has been repeated two or three times on various days), experience leads us to feel that in most cases ordinary treatment will be of very little avail. The patient should be warned regarding the condition of his hearing and its dubious prognosis.

**Tuning-forks.**—Before considering the use of tuning-forks in functional testing, it may not be out of place to briefly mention some of the essential facts regarding the physics of forks, their qualifications, etc. The prongs of the fork move in transverse vibrations and the stem in longitudinal vibrations for the same length of time. The prongs have a wide amplitude and little intensity, while the stem's vibrations show small amplitude with great intensity. All forks show a certain definite decrement of vibration after having been excited; the intensity rapidly diminishing after the first few seconds, then more slowly through a long curve of logarithmic progression. This decrement is different in various forks of the same pitch, but is practically the same for any one fork unless there is a great variation in the force with which the fork is struck. Every fork has a certain fundamental tone and then a great number of overtones or harmonics. In order to remove the latter, and bring out only the fundamental tone, clamps or weights are often placed on the prongs. While this has the advantage of eliminating the overtones, it has the disadvantage of diminishing the time for which the fork will vibrate. Edelmann found, however, that it is possible to eliminate the overtones and especially the first one, which is usually the most pronounced one, by forging the fork so that the first overtone and the so-called "plate tone" have the same pitch. It is then possible to do without clamps or weights. When a fork is lying on wooden ledges and is struck at certain points, the "plate tone" is produced instead of the sound emitted when the fork is struck in the ordinary manner. If by any chance

the "plate tone" and the prong tone happen to be of the same pitch, the fork will be entirely silent when excited in the usual way.

Among the requisites which we believe forks should have in order to function best, are (1) that they be made of one piece of metal; (2) that the handle should be large enough so that it can easily be held and yet loosely, without stopping the vibrations or damping them; (3) that the fork should sound for a long time; (4) that there should be weights for the lower pitched forks to eliminate the overtones; (5) that forks should not be nicked for when peeling occurs, adventitious sounds are produced by the loose pieces of metal; (6) and that rustless metal, if feasible, should be employed to prevent changes in pitch.

The last-named problem seems to be solved, for in the early part of 1927 Mr. B. E. Eisenhour of the Riverbank Laboratory, Geneva, Illinois (property of Col. George Fabyan) constructed a complete series of forks consisting of a magnesium alloy. The composition is stated to be magnesium 95.6 per cent., manganese 0.4 per cent., and aluminum 4 per cent. This alloy is rust proof and has the further advantage that it is very light in weight. When one recalls that the specific gravity of steel is about 7.7, that of aluminum about 2.7, and that of magnesium 1.7, it will readily be seen that the alloy has a specific gravity about one-third that of steel. This allows functional testing for considerable periods of time and the holding of larger without discomfort. We are informed that the cost of producing these forks is practically that of first-class steel forks.

So far as duration by air conduction is concerned, these forks are about as good as the steel ones except  $c^4$  (2048 double vibrations) and the  $c^5$  (4096 d. v.). At some of the pitches the magnesium forks are even better than the steel ones. On the other hand, some of the medium low-pitched steel forks vibrate a little longer. So far as bone conduction is concerned it was at first thought that the duration would be less with magnesium forks. However, some tests done by the writer showed that for bone conduction the magnesium forks served very well; in fact, practically as well as the heavier steel forks. It will, of course, require longer experience to see whether magnesium alloy forks will definitely supplant the steel ones.

The great advantages of the two qualities of resistance to rusting and the lightness of weight seem so desirable that I believe the magnesium alloy forks should be thoroughly tested out.

The range of forks usually employed is from  $C_2$  (16 d. v.) to  $c^5$  (4096 d. v.). It is essential that all forks be properly designated, namely, whether single or double vibrations are being used; otherwise a difference of a whole octave in pitch may result. Forks of twelve double vibrations have been made, but they are very large and difficult to manipulate. Forks higher than  $c^5$  have also been constructed, but not for general otological practice.

There are several sets of forks of which the two best ones are perhaps the Hartman series consisting of five forks,  $c$  (128 d. v.) to  $c^4$  (2048 d. v.) and the Edelmann set of continuous tuning-forks from  $C_2$  (16 d. v.) to  $c^5$  (4096 d. v.). Besides the latter series we also have the two Bezold forks: the unweighted A (108 d. v.) used very largely for the Weber and Schwabach tests, and the  $a^1$  (435 d. v.) extensively used in making the Rinne test. It is desirable to have as large a series of forks as possible, but it is not necessary to have the continuous series except for complete examination for

possible islands and defects of hearing. For routine examination, four or five forks; preferably a fairly low one, one or two of medium pitch, and one of high pitch are essential. The Committee on Standardization of Tuning Forks and Hearing Tests of the American Academy of Ophthalmology and Otolaryngology, of which the writer is Chairman, has recommended a minimum of three forks, namely, C-1 (32 d. v.),  $c^1$  (256 d. v.), and  $c^4$  (2048 d. v.); but preferably five forks: C-1 (32 d. v.), c (128 d. v.),  $c^1$  (256 d. v.),  $c^2$  (512 d. v.), and  $c^4$  (2048 d. v.), which should be in the possession of every otologist. A continuous series of forks, especially the Edelmann series, is quite expensive, rather bulky, and is not absolutely essential for the usual testing.

**Methods of Exciting the Forks.**—The general rule is that the lower the pitch of the fork the softer should be the material used for striking it. For the very lowest tones the side of the hand (hypothenar eminence) may be used; for those of medium pitch, a rubber-covered pleximeter; and for the higher forks, a metal hammer. A rubber weight attached to a string and used in the form of a pendulum also gives a rather accurate excitation. A pendulum consisting of a metal bar covered with rubber has also been used. It is best to strike forks at the "percussion center" which is approximately at the junction of the middle and distal thirds of the prongs, as this gives less overtones and a longer period of vibration.

It is essential that some very definite method should be adopted by otologists so that all excitations of forks would be quite uniform, and the results thus obtained be comparable. We have as a rule excited some of the lowest forks, like the unweighted A (108 d. v.), by allowing them to fall from a perpendicular position to a horizontal one, striking the knee. The medium pitched forks like  $a^1$  (435 d. v.) and  $c^1$  (256 d. v.) are usually held at right angles to the body with the outer, broad surface of one of the prongs uppermost. A small pleximeter, such as used by neurologists for eliciting reflexes, is permitted to fall of its own weight from a perpendicular to a horizontal position striking the prong. In this way a uniform excitation of the fork can be obtained. For the highest forks, instead of striking them with a metal hammer, a simple means of excitation which admits of three degrees of intensity has been used: First, lightly rub the prongs of the fork with the fingers; if the sound is not heard, strike one of the prongs with the fingernail; and then if still not heard, strike the prong with a metal hammer.

It was previously assumed that the greatest intensity proceeds from the flat surface of the prongs and the least from the angles, as here we have interference of the sound-waves. The writer conducted experiments with the Webster phonometer which seem to prove that the greatest intensity of sound proceeds from the broad or outer surface of the prongs, less from the two narrow surfaces held parallel with the ear, still less from the ends of the prongs, and least from the angle between the two surfaces of the prongs. It seems best, therefore, to hold the broad or outer surface of the prongs parallel to and near the ear without permitting contact with the vibrissæ or auricle. Koenig rods are used by some otologists in place of, or as an adjunct to, tuning-forks, but we believe the forks are preferable because with them both air and bone conduction can be determined.

Certain difficulties arise in all tuning-fork tests. In the first place we must rely so much on the subjective statements and impressions of the

patient. Secondly, it is difficult for him to distinguish between feeling and hearing the fork especially with the very low-pitched ones. Thirdly, patients often attempt to correct statements with reference to their sensation, which seem to them paradoxical. For instance, the Weber is referred to the worse ear in conduction impairment, but the patient believes that the result must be incorrect, as he assumes that he would hear all sounds, whether by air or bone conduction in the better ear, and he, therefore, says he must be hearing the fork in that ear.

**Testing the Range of Hearing.**—The range of hearing for the normal individual is said to be from 12 double vibrations to 20,000 double vibrations per second. The so-called "speech area" was placed by Bezold at about  $c^1$  (256 d. v.) to  $g^2$  (800 d. v.). While this is in the main true there are some tones in speech much higher, one of the overtones of the vowel "e" being about 3000 double vibrations.

Begin with the lowest possible forks, have the patient close his eyes so that he will not be influenced by the sight of the fork or its proximity to the ear, and have him state when he hears a certain sound. Do not ask him, "Do you hear this?" but have him tell you when he first appreciates the tone. If the lowest tone is not heard, proceed up the scale until you find the point at which he begins to hear the forks. After that, continue to use the forks in ascending octaves until the highest ones are reached. In testing with the low forks it is not necessary to close the opposite ear. (N. B.—Patients must be instructed in the difference between hearing very low tones and merely feeling the impact of the sound-waves against the auricle.)

When using the higher-pitched forks it is necessary that the opposite ear be tightly closed so that the sound is not carried over and appreciated by the ear which is not being examined at the time. It is, however, almost impossible to exclude high-pitched tones from that ear, no matter what method is used, especially if the fork is struck with a metal hammer. After exciting one of the high forks ( $c^4$ , 2048 d. v., or  $c^5$ , 4096 d. v.) in the manner previously described, and having the patient close the opposite ear, the vibrating fork is brought close to the examined ear. If heard at all, the fork is held steadily until the sound dies away, when it is removed for a second or fraction thereof, then returned to the ear. Often it is again heard and the process of removing and bringing back the fork is repeated until it is no longer heard. This whole period of time shows the comparative hearing for the high tones. The failure to hear after a time, with return of hearing when the fork is again brought back to the ear after a very short removal, is the "fatigue symptom." The temporary removal of the fork permits the auditory nerve to recover from the fatigue.

After examining with the highest pitched forks, the upper limits are further tested either by means of the Galton whistle or its modification, the Edelmann-Galton whistle, or still more recently, the Schaefer-Galton whistle. With the original Galton whistle many shrill overtones are heard and no chart showing the exact pitch produced when the whistle is set at various points is furnished. The Edelmann-Galton is superior to this model as an accurate chart is furnished, but it is very difficult to have the whistle perfectly adjusted because both the aperture and the pipe length should be set at a certain point and it is sometimes impossible to produce

an audible whistle at the very high pitches. In the new Schaefer modification only the pipe is moved, the aperture being fixed. With all whistles it is difficult for the patient to distinguish between the blowing sound and the actual, high-pitched, shrill whistle. Furthermore, the whistles are easily disarranged and if the sharp edge of the aperture or pipe is altered in any way by handling roughly, etc., the accuracy of the whistle is greatly impaired. However, for practical purposes and rapid results, in getting a general idea of the upper tone limit, the whistles when properly used are of considerable value. The Struycken monochord modified by Prof. Karl L. Schaefer has proved to be a most valuable instrument in that it is possible to test the upper tone limit not only by air, but also by bone conduction. This apparatus consists of a steel wire in a frame which is calibrated to show the pitch at the various points at which the wire may be clamped. The wire is caused to vibrate either by rubbing it with cotton or other substance moistened with alcohol, etc., producing longitudinal vibrations, or drawing a violin bow across it giving transverse vibrations. Now we have always assumed that the very highest tones are heard better by air than by bone conduction, but it has been shown with the monochord that this is apparently not true. To explain the fact that with bone conduction the higher tones are better heard than by air, Kalähne offers the hypothesis that as long as the amplitude of vibration is the same, the hearing for tones is better by bone than by air conduction. With the monochord it is quite possible to do this because the amplitude will be the same; not so with the tuning-fork, since the amplitude of the excursion of the stem of the fork is about one one-hundredth of that of the prongs. The monochord has the further advantage that it is not easily disarranged and that it can be easily manipulated. With the whistles or monochord, begin at the highest point, bring the apparatus near the ear, and have the patient state when he hears the high-pitched, shrill sound. If the highest tones are not heard, proceed to the lower tones until a pitch is reached at which the patient definitely hears the characteristic sound; this point is then noted as the upper limit of hearing.

**Air and Bone Conduction.**—Sound is conducted to the ears by air conduction, or by bone conduction, or both. Air conduction is by way of the external auditory meatus, tympanic membrane, and ossicular chain to the internal ear; or it may go by way of the nose and pharynx through the eustachian tube to the middle ear and then to the inner ear. Bone conduction also has two pathways; either the craniotympanic, by way of the bones of the skull to the middle ear, and then to the internal ear; or craniolabyrinthine, through the cranial bones via petrous portion of the temporal bone to the inner ear. According to Bezold<sup>1</sup> the difference between air and bone conduction lies in the fact that by way of air the sound waves strike the flat surface of the drum-membrane and ligamentum annulare, and by way of bone, they impinge upon the edge thereof.

As a rule it is observed that the hearing for most sounds is much longer by air conduction than by bone conduction. Bezold calls attention to the fact that the usual hearing by air invariably occurs through the medium of the conduction apparatus; he says that "even the small fraction of sound waves which in cases of intensive tones in the air strike the entire surface of the skull, are perceived only to the extent that they set the conduction apparatus in transverse vibration." In case of direct transmission

of vibrations of rigid bodies to the skull, as occurs with contact of tuning-forks on the head or various parts of the skeleton, the labyrinth as well as the conduction apparatus is caused to vibrate. In all probability our perception of sound is limited to waves (whether by air or bone conduction) which traverse the conduction apparatus before reaching the labyrinth; those vibrations which reach the labyrinth directly without the aid of the conducting apparatus are said to be unperceived by us. "The function of the conduction apparatus in sound perception consists largely in transferring the longitudinal air waves (including those which directly strike the skull), into transverse vibrations of this mechanism (as a whole, together with the column of labyrinthine fluid)." "The ossicular chain is most essential in transmitting the lower tones of the musical scale (tones produced by vibrations of large amplitude but little force), which the ossicular levers convert into vibrations of smaller amplitude but of great intensity. The *higher* tones, however, are produced by vibrations of small amplitude, whose relatively greater intensity is capable of producing a wave in the labyrinth fluid without increase in force by means of the leverage system of the ossicles."

**The Weber Test.**—This test is employed for determining the presence of the lateralization of sound. As a rule, one of the heavier low-pitched forks is chosen for this purpose. We are accustomed to use the Bezold A (108 d. v.) unweighted fork. After exciting it in the usual manner by allowing it to fall from a perpendicular to a horizontal position, with the broad surface of one of the prongs striking the knee, the fork is held as loosely as possible, so as not to interfere with the vibrations of the stem, and placed on the median line of the vertex. Some employ the forehead, the root of the nose, the teeth, or the chin. When placed in one of the two latter positions, the tone is usually heard louder because the mouth and nasopharynx act as resonators. The patient is then asked whether he hears the sound of the fork louder in the head itself or in one of the ears. The degree of lateralization is noted by shifting the fork away from the median line of the head. For instance, if the fork is heard louder in the left ear when held in the median line, move it to the right side of the head, and see how far from the median line the fork may be moved and the tone still be lateralized to the left ear. If heard in the left ear, with the fork held far over on the right side, it means either a very marked conduction impairment of the left ear or a very decided nerve or end-organ involvement of the right ear, with lateralization to the left, or in this case, the better hearing ear.

*Interpretation of this Test.*—Normally with both ears in the same condition the sound is usually heard "in the head" as the patient expresses it. Where there is a conduction apparatus impairment, the sound is usually heard in the poorer ear, but if there is a bilateral conduction impairment it is lateralized to the worse of the two sides. When on the other hand, an inner ear or nerve lesion is present, the sound is heard in the better hearing ear, but if there is bilateral inner ear involvement, the sound goes to the better of the two ears. Often, however, there are exceptions to these rules.

One difficulty with this test is the fact that ignorant patients give wrong answers since they think the lateralization must occur to what seems to them the better hearing ear. Furthermore, it must be remembered that pus in the maxillary antra or involvement of the eustachian tube, in cases of infection of the pharynx, may cause a lateralization of the sound.

Dr. George Mackenzie has described the so-called paradoxical Weber test. If for instance, an acute otitis media is present in the left ear, the sound is lateralized in that ear since we have a conduction apparatus involvement, but if mastoiditis is present, the bone conduction may be less than in the normal ear (right), since the pus and granulations in the mastoid give poor transmission of sound.

It is known, as we<sup>2</sup> also found in a series of cases examined some time ago, that the position of the fork does have some influence upon the lateralization. As regards the different forks which may be used, it does not seem to make a great deal of difference in the lateralization, as long as forks of approximately the same pitch are employed. It is better to use forks of the middle octaves, preferring those of lower pitch to the higher ones, since with the latter it is difficult at times to tell whether the sounds are conducted to one ear or the other by bone or by air.

A sudden change in the lateralization of the Weber is of great significance. If, with the fork heard for instance in the left ear (which has a conduction impairment due to an acute otitis media), there is a sudden shifting in lateralization to the right side, it usually means that an involvement of the inner ear (labyrinthitis) has occurred in the left ear, and the Weber is, therefore, heard in the opposite or better ear.

*Conclusion.*—We may perhaps safely say that the Weber test is of value only when used in conjunction with and agreeing with the results obtained by other functional tests. It is of no aid in some cases, and it may at times cause uncertainty owing to its great variability and tendency to contradictory results. It should, however, we think, be employed in all cases, because there are many in which the lateralization if present is a great aid to diagnosis, as for instance, in deciding between otitis externa and otitis media.

**Schwabach Test.**—This test is used for determining the duration of the bone conduction of the individual as compared with a normal standard. A fork of medium pitch somewhere between A (108 d. v.) and a<sup>1</sup> (435 d. v.) is employed, different authorities showing preference for various forks. We<sup>3</sup> like to use the large, unweighted Bezold A fork (108 d. v.) for this test. After exciting the fork in the usual manner, it is placed in the median line of the vertex; or, as some otologists do, at the root of the nose with the head bent backward. Others place the fork on the mastoid process, the same as in the Rinne test. When no longer heard, the fork is set upon the head of the examiner, or some other person whose bone conduction is supposed to be normal. In order to make this test more objective, and not dependent upon some other person for comparison, we have determined the average duration of hearing (at certain ages) for the particular forks employed, in a large number of normal individuals. Having obtained this average we simply compare it with the length of time which the fork is heard by the patient, and thus see at once whether the bone conduction is normal. If it is lengthened or shortened, note whether moderately or greatly changed.

Several rules should be observed in performing this test. Care should be taken that the hairs are separated so that the fork rests directly upon the scalp when the vertex is employed. Secondly, the patient should be cautioned against confusing the feeling of the vibration of the fork with the hearing of the actual tone thereof. Thirdly, the fork should be allowed to rest if possible of its own weight, since an increase in the pressure with which

it is applied changes the amount of bone conduction. Fourthly, the stem of the fork should not be held too tight, as the vibrations are thereby damped or stopped. The usual manner is to have the patient state when he no longer hears the sound, then remove the fork for a second, because of the "fatigue" symptom, then replace it on the head and note whether it is again heard. Even in normal individuals the duration of bone conduction varies with the age of the individuals (at or after middle age, it is considerably diminished), although it may differ in persons of the same age. It may also vary with the thickness of the cranial bones, the size of the air spaces such as the mastoid cells, as well as certain changes in the skull, such as depressions, results of traumatisms, etc. According to Wanner and Gudden,<sup>4</sup> adhesions between the dura and the bone, as well as other traumatic or pathological changes influence the Schwabach by giving a greatly shortened bone conduction despite the normal hearing by way of air. The amount of the hair, the tension of the skin, contact with the auricle, and the pressure with which the fork is applied, all may cause variations in the bone conduction. Politzer<sup>5</sup> believes that the Schwabach alone is only in rare instances of great value in differentiating middle- from inner-ear disease. When however, the bone conduction is found prolonged and the Rinne is decidedly negative, the Schwabach aids in making a diagnosis of interference with the sound conduction apparatus; where the Schwabach is shortened together with a positive Rinne and impaired hearing, it greatly assists in the diagnosis of an inner-ear or nerve affection. It is well to remember that if hearing for conversation is less than 1 meter the Schwabach is of practically no value because in these cases there is usually a severe lesion of the inner ear, and patients cannot distinguish between feeling and hearing the sound. If one ear is deaf and the Schwabach is tried by way of the mastoid, one may apparently get some bone conduction, but it is really by way of the opposite ear. Carrying these various factors in mind, one may say that lengthened bone conduction usually means impairment in the conduction apparatus, and diminished bone conduction usually signifies involvement of the inner ear or auditory nerve. It is necessary to remember that a slight increase or a slight diminution does not have any definite significance since there are certain variations even in the normal.

It is Mach's theory that bone conduction is increased by any disturbance in the external or middle ear because the normal outflow of sound is thereby hindered and a certain reflection of sound toward the labyrinth is produced. A disturbance of the sound conduction mechanism, caused by plugs of cerumen, stapes ankylosis, fluid in the middle ear, dry perforation of the membrana tympani, etc., may give lengthened bone conduction. Randall has demonstrated the retention of sound by means of a fork on the head and tubing in the ear; when the tube is compressed so the sound-waves cannot escape, the individual hears the fork louder than when the tube is open.

Bezold states that for proper sound transmission by way of air, the conduction apparatus must be in perfect equilibrium, a slight disturbance of which, such as a few tubal adhesions, is sufficient to produce a decided diminution in air conduction and an equally definite increase in bone conduction. This equilibrium is likewise affected by large perforations of the membrana tympani with a loss of some of the radiating fibers and a resulting overaction of the tensor tympani muscles.

Oscar Beck, of Vienna, has called attention to the diminution in bone

conduction so often noted in cases of lues with otherwise good hearing and no aural symptoms. He says that this lowered bone conduction may be found in 80 per cent. of all syphilitic subjects and that it appears mostly in the secondary, and only rarely in the first stages of lues. The diminished bone conductivity may be due to increased cerebrospinal pressure, to changes in the meninges, etc., but no entirely satisfactory explanation has as yet been offered.

**Rinne's Test.**—Dr. A. Rinne, in 1855, first described his test for comparison of air with bone conduction in the same individual. He placed the fork on the upper incisors and when the sound was no longer heard the hearing by air conduction was determined. As now performed the test is usually made by placing the vibrating fork upon the mastoid bone in the region of the antrum, and when the sound is no longer heard, holding the broad or outer surface of one of the prongs near the external auditory meatus and observing how much longer the sound is perceived by air. (N. B. The fork must not be allowed to come in contact with the auricle or vibrissæ.) In a normal individual, the air conduction is very considerably longer than bone conduction; that is to say the Rinne is *positive*. In those cases where bone conduction is longer than air, the Rinne is designated as being *negative*. Bezold holds that usually the air conduction is about thirty seconds longer than the bone in a normal positive Rinne when the a<sup>1</sup> fork (435 d. v.) is used. If the fork is held only at the meatus, without having been previously placed on the mastoid the duration of air conduction is longer than when the usual Rinne test is done; in fact, it lasts seventy to eighty seconds. This is due to the fact that when the stem of the fork is pressed against the bone the vibrations are damped and the duration of the sound is thus shortened.

We have at least seven or eight varieties of the Rinne<sup>6</sup> reaction:

(1) There are two forms of the positive Rinne. First that in which the air conduction is much longer than the bone conduction, but both factors are normal in duration. This is the normal positive Rinne found in cases of good ears. Second, there is a positive Rinne with air conduction longer than bone conduction, but both of them shortened as compared with the normal. This form is found in inner-ear disease with impairment in hearing.

(2) There are three forms of the negative Rinne. First, where bone conduction is much increased and longer than the air conduction, which latter is moderately diminished, this form is found in the ordinary acute middle-ear disease. Second, bone conduction longer than air conduction, but both diminished as compared with the normal; here we usually find a combination of inner- and middle-ear diseases. Third, bone conduction is longer than air conduction but both greatly diminished. This form occurs in very far advanced ear diseases; the air conduction is then lost more rapidly than bone conduction. Fourth, the so-called "infinite Rinne." If not heard at all by air, but somewhat by bone we have the "infinitely" negative Rinne; this is often found in cases of far advanced inner-ear disease. It is probable that the bone conduction here comes by way of the opposite ear.

(3) "Indifferent Rinne," that is to say, the plus-minus form. Of this there are two varieties. First, that in which the air and bone conduction are of equal length, because the air conduction is slightly diminished and the bone conduction slightly increased; the hearing is good in this form in which a slight middle-ear affection is usually the condition present. Sec-

ond, air and bone conduction are equal in duration, but both much shorter than normal; in this form the hearing is very poor, as this reaction is usually found in cases of serious inner-ear disease.

The Rinne test is of little value if the hearing is much diminished, so that conversation is heard less than 1 meter. With total deafness on one side one may have the "infinitely" negative Rinne because the bone conduction comes from the opposite ear as previously mentioned. Practical experience has shown that the Rinne is one of the most valuable of the tuning-fork tests, and that it gives us a very good idea of the location of the lesion, whether it is in the middle ear, in the inner ear, or in both. However, objections have been raised to this test because it measures the difference between two factors, namely, the vibrations of the fork stem and its prongs, which are physically so different that they are not comparable. While the prongs move in transverse vibrations of large amplitude, but slight intensity, the stem at the same time for just as long a period shows longitudinal vibrations of small amplitude but great intensity.

Dr. J. P. Minton and myself<sup>7</sup> endeavored to overcome these objections by using the stem of the fork for both air and bone conduction. After exciting the fork in the usual manner and placing it upon the mastoid until it is no longer heard by bone, the end of the stem is inserted into a piece of rubber tubing which at its other end has a hard-rubber, olive-shaped, perforated tip which is placed in the ear. Physical experiments show that the sound-waves are hardly if at all, transmitted through the wall of the tubing, but are conducted mainly along the air column in the lumen, and the sound is heard by air conduction. It is thus very easy to use the stem of the fork for both air and bone conduction, and the theoretic objections to the Rinne test are, we believe, eliminated. (Although the literature was completely searched for us at the time of our work, no reference to the use of the stem for both air and bone conduction was found. Lately we discovered a brief communication by Bonnier<sup>8</sup> in 1899, in which he advises the use of tubing applied to the ear. He, however, does not connect this with an olive-shaped tip, but places the fork *on*, not *in* the auscultation tube, a few centimeters from the auricle. He does not give experimental data, etc., to show that the modification of the test is physically correct. We also found a modification by Stefanini,<sup>9</sup> but his was a much more complicated one.)

With reference to the comparison between bone and air conduction, E. R. Lewis has recently stated that the stem of the tuning-fork is heard louder and longer, when placed on the tragus than on the mastoid, in all cases except those of stapes fixation. While we have not seen enough cases to thoroughly test this out, we believe that there are instances with conduction by way of the mastoid longer than by the tragus where apparently no stapes fixation is present, as, for example, in cases of early acute otitis media which we have observed. This is a very interesting communication and should be further investigated.

Summarizing we may say that the Weber is used to determine whether lateralization by way of bone is present in one ear as compared with the other; the Schwabach is employed to determine the length of bone conduction of the patient as compared with a normal control, or more objectively by comparing with the average duration of hearing for the forks employed; the Rinne test compares air with bone conduction in the same individual. The Schwabach test is the one that is most easily affected subjectively,

as it is often difficult for the patient to distinguish between the hearing the tone and feeling the vibration of the forks, especially when the large, low pitched ones are used.

**Gellé Test.**—This test is used for determining the mobility of the foot-plate of the stapes. A Politzer or other bag is connected by way of tubing to an air-tight-fitting, olive-shaped tip placed in the external auditory meatus. With the vibrating tuning-fork resting on the vertex, mastoid, or Politzer bag, the bulb is compressed. Thus the foot-plate of the stapes is pushed further into the oval window, the hearing is impaired for the time being, and we have a positive Gellé. When, however, the stapelial foot-plate is fixed, and cannot be further pressed toward the labyrinth, the hearing is not diminished by the compression of the air, thus giving a negative Gellé. Bárány modified this test by using a T-shaped auscultation tube, two ends of which had ear pieces, the third a mouth piece. The former are placed in one ear of the patient and of the examiner, and the latter held in the examiner's mouth. The fork is placed at about the middle of the rubber tubing, and when the air is compressed by the physician he can note whether or not hearing is diminished by the patient, using himself as the control. Gatscher proposed a modification with compression by way of a catheter in the eustachian tube, thus eliminating any influence due to changes in the tympanic membrane.

Unsatisfactory results are sometimes obtained with the Gellé test, due to the fact that the patients often fail to thoroughly understand what is being asked of them, or do not pay close enough attention to the possible change in hearing during the time that the bulb is compressed. It easily happens that the ear piece does not fit tightly in the meatus so that there is an escape of air. If properly performed, however, this test should give valuable information especially in those cases of typical otosclerosis with marked fixation of the foot-plate of the stapes.

A good many other functional tests have been used from time to time, such as the Politzer test for patency of the eustachian tube, Bing's test, and the Lucae-Dennert test. These tests, however, are not extensively used, and space does not permit their discussion here. If the three great tests (Weber, Schwabach, and Rinne), already described, are carefully done, sufficient information will be obtained so that these less used tests will not be needed.

**The Unmasking of Simulated Unilateral Deafness.**—As was mentioned earlier in this chapter, doubt exists as to whether a patient hears the whispered voice with the affected ear when it is only heard a distance of less than 1 meter. There is then the possibility that the sound is being conducted either through the bones of the head or by way of air to the opposite ear. The use of the 2- or 3-meter speaking tube, as previously mentioned, is then very valuable. If the whisper is heard by way of this tube, then there is certainly hearing in the examined ear; and if not heard there is probably complete loss of hearing in that ear.

For the detection of simulated total unilateral deafness a number of tests have been devised. We believe that the Stenger test is not only very reliable, but easy to perform. The principle upon which it rests is the fact that when two sources of sound of the same pitch and intensity are presented to the ears, and one source is nearer to one ear than the other one is to the opposite ear, the hearing in the ear from which the source is more distant will be drowned out by that entering the ear from the nearer

source. In performing this test it is essential that a pair of forks of exactly the same pitch be employed, and that the patient does not know that two forks are being used simultaneously. For this reason it is best to have him blindfolded. We usually employ the  $a^1$  (435 d. v.) forks. These are struck at the same time, and it is first determined at which distance the patient admits hearing the fork in his good ear; let us say ten inches from the left ear. Since he pretends to be totally deaf in his right ear, he denies hearing the same-pitched fork no matter how closely held to the right ear. With one fork kept vibrating let us say 3 inches from the right or supposedly deaf ear, and the other fork approaching within 6 inches of the left ear, the patient will state that he does not hear any sound at all, even though he previously admitted hearing the fork with the well (left) ear at a distance of 10 inches. The reason is this: The fork held 3 inches from the right ear will drown out the sound of the fork held 6 inches from the left ear, but of whose presence the patient does not know. In view of the fact that he claims to be deaf in the right ear, he will say he hears nothing at all; whereas, if he were really deaf in his right ear he would not hear the tuning-fork held near that ear, but would certainly hear the one vibrating in the vicinity of the left or well ear. Thus the diagnosis of malingering is established.

Dr. W. A. Wells<sup>10</sup> recently described an ingenious modification of this test. He uses a piece of rubber tubing of  $\frac{1}{4}$ -inch caliber, 30 inches in length, and of a firmness to give good conduction of sound. In one end of the tube is an ear-piece which fits well in the auditory meatus, while in the other end is inserted the stem of a tuning-fork of about 120 double vibrations per second. Applied in the ear in the case of a malingerer, the principle of the procedure "lies in the fact that with the intensified sounds in the alleged deaf ear one is deprived of the power of recognizing the weaker sound in the good ear, which is not true in a real deafness. While the earpiece is in the supposedly deaf ear, the vibrating fork is conducted stealthily to the meatus of the opposite ear to a point at which it has been previously ascertained that he could distinctly hear it; the really deaf person will now report hearing the fork as soon as it comes into the range of hearing in his good ear. The malingerer, unconscious of any hearing in his good ear because of the lateralization of sound to the alleged deaf ear, continues to report no hearing." This is the same finding as with the Stenger test.

The reading test, especially with the noise apparatus in one or both ears in order to see whether the patient will raise his voice or not, has also been used in the effort to discover simulation of unilateral deafness.

The simulation of bilateral deafness is sometimes extremely difficult to unmask. Often these patients have schooled themselves so thoroughly that they do not respond in any way to the voice, to noises, or other happenings in their vicinity. It is sometimes necessary to observe such a patient for a long period of time or to have him carefully watched by some one who can catch him off his guard. Occasionally the making of a slurring remark in his presence will cause him to suddenly react in anger, thereby displaying the simulation. It is a well-known fact that persons who are entirely deaf usually respond to tactile sensations. If, for instance, a tremendous concussion is produced, as by dropping a large dishpan on a hard floor, a person entirely devoid of hearing will feel the concussion and will suddenly turn to see the cause of the commotion. The malingerer

usually controls himself so that he does not make any move if this test is carried out.

**The Recording of Functional Tests.**—It is essential that a proper record of all tests be made; but it is difficult to devise a method whereby the data will be readily accessible to and understood by readers throughout the world. Each one is inclined to work out a scheme of his own, but we have employed for years the acoumetric formula proposed at the Eighth Otological Congress in Buda Pesth in 1909. For our own purposes we have added to it certain factors that seemed convenient, such as recording resonator tests, etc. Another method which is very good for recording the tests is that used in the University Clinic of Vienna, which we have on our own cards combined with the acoumetric formula just mentioned. We submit, herewith, illustrations of these two methods of recording functional tests.

This shows in brief the formula adopted at Buda Pesth at the Eighth International Congress:

A D W S

..... a<sup>1</sup>M( ) a<sup>1</sup> Air ( ) R ( ) H.

A S A ( )

P.... V ( ) v ( ) LI LS (G) A. C. V.... A. C.  
A. P. V.... A. P. v.....

Explanation of abbreviations used above:

A D Aurum dextrum

R Rinne

A S Aurum sinistrum

H Horlogium (watch)

W S Weber-Schwabach { A fork 108 double variations (d. v.)  
a<sup>1</sup>M (fork 435 d. v. on mastoid)  
a<sup>1</sup> air (fork 435 d. v. by air)

P Politzer acoumeter

V Voice (conversation)

v Voice (whisper)

L I Limen inferior (lower limit)

L S Limen superior (upper limit)

(G) Galton whistle

A. C. After catheterization

A. P. After politzerization

The other method is the one used largely by the Vienna Clinic which we have also adopted with slight modifications. The combination of the two gives a very complete and concise record which may be easily read at a glance.

R

L

C<sub>2</sub>

C<sub>1</sub>

C

c

c<sup>1</sup>

c<sup>2</sup>

c<sup>3</sup>

c<sup>4</sup>

c<sup>5</sup>

Gellé

Stenger

Fistula Symptom

Spon. Nyst.

Spon. Past Pointing.

R, right ear.

L, left ear.

**Resonators and Their Possible Use as Aids in Functional Testing.**—For a number of years, the author<sup>11</sup> has been experimenting with resonators, especially those devised by Prof. Karl L. Schaefer, in order to see whether their use would be of value in the functional testing of hearing in conjunction with tuning-fork findings. Resonance or sympathetic vibration depends upon the principle that a number of slight impulses properly applied will finally create a considerable momentum, as seen in the well-known fact of giving impulse to a swing or pendulum at the proper phase of the oscillation. It is easy with the resonator to test the actual duration of vibration of the various forks. In determining the presence of actual deafness for certain tones, resonators are of great aid; for if a fork, especially one whose pitch lies in the so-called "speech area" is not heard at all when reinforced by the resonator, the hearing for that tone may be said to be absent for all practical purposes. Furthermore, it is possible to determine the pitch of a tinnitus aurium from the patient's own observation when the resonator is attuned to the various sounds in the surrounding air. Together with Dr. John P. Minton<sup>12</sup> the author conducted some studies on the reinforcement of sound by means of the Schaefer resonators. Our conclusions were that the sound intensity amplification depends on the manner in which the resonators were used. The maximum value of this amplification, as observed in a closed room by the examiner was 273; by the patient, 44; and, as it appears in an open, unconfined space, 10,700. The efficiency of the Schaefer resonators as well as of other types, decreases rapidly when approaching the higher tones. The same is true of the lower ones except when in an open unconfined space. It has always been known that resonators show their greatest efficiency in the middle octaves. Resonators may be used to test certain definite tones, for instance, A (108 d. v.),  $c^1$  (256 d. v.),  $a^1$  (435 d. v.),  $c^3$  (1024 d. v.) which latter is the highest tone which will be reinforced, with any degree of efficiency by the ordinary resonators. If a fork is struck with definite intensity and held before a resonator you can measure the distance heard. This method gives more even distribution of sound than by holding the fork before the ear, since it may turn in the fingers or touch the auricle; there is also less interference with the sound waves from the fork. Work with resonators indicates that their use may have some significance, but to really decide their actual clinical value, if any, in otology, such as an aid to diagnosis, prognosis, etc., will require further extensive investigation.

**Audiometers.**—In order to obtain as accurate an apparatus as possible for the testing of hearing and at the same time to cover a comprehensive range of pitch, a number of audiometers have been devised. Perhaps those best known are the following four: (1) The one invented by Prof. Seashore and modified and employed by Drs. L. W. Dean and C. C. Bunch of Iowa City, Iowa. This apparatus has a frequency variation from 30 to 7070 double vibrations per second. The latest model, I believe, has 10 to 14,000 double vibrations. (2) The apparatus of the Western Electric Company in which the frequencies available are separated by intervals of an octave. (3) The Knudson audiometer in which the frequencies available are the octave intervals from 64 up to 8192 double vibrations per second, with a continuous frequency variation from 7000 up to 20,000 double vibrations per second. (4) The Kranz audiometer in which the frequency variation is continuous from 250 to 6000 double vibrations per second, this

being divided into six ranges of about an octave each. The frequency variation is under the control of either the operator or the subject. The three latter types of the apparatus all use the audion or vacuum tube oscillator for the amplification of sound. The greatest advantage of the audiometer seems to lie in the graph and curve of the state of hearing which is obtained. This may then be compared with that secured by another otologist or may be compared with one's own record later on. In order, however, that the plan may be universally applied it is necessary that the various audiometers be standardized since the result shown on one graph should correspond definitely with those shown on others obtained from other apparatus.

As constructed at present, the audiometers are very expensive and it is doubtful whether the average otologist would be willing to make the investment required in order to have one of these appliances for functional testing. Some of the other objections which have been raised to the use of the audiometers are, that with most of them it is impossible to have as wide a range of testing as with the tuning-fork and monochord, that with some of the types of instruments one cannot get a continuous tone series in the pitches below 7000 double vibrations, and lastly, one cannot test the bone conduction properly.

An attachment for the audiometer for the testing of bone conduction has been devised by the Western Electric Company. Whether this will serve entirely satisfactorily further experience must show.

We have not yet had sufficient experience to pass judgment upon these appliances. Considerable experimentation, the testing of many cases, and the analysis of the results obtained must first be done to allow us to draw definite conclusions.

The Bureau of Standards at Washington, D. C., has agreed for a very nominal charge to determine the so-called damping "constant" or decrement of the forks submitted to them. It is possible even without audiometers to get a very definite graph of the state of hearing by the use of forks whose "constant" or decrement is definitely known. By using a uniform method of excitation of the fork, and subtracting the time that the forks are heard by the patient, from the time that the normal individual is supposed to hear them, and then multiplying the remainder by the "constant" of the fork, one gets its figure regarding the degree of hearing shown by the patient. Having a fair number of good forks whose "constant" is known, it is possible to make very accurate tests of the state of hearing without the use of very expensive apparatus which some men are disinclined or unable to obtain.

**Differential Diagnosis of Middle- and Inner-Ear Disease.**—It is impossible here to go very deeply into this subject, but as stated at the outset, one of the principal purposes of functional testing of hearing is to determine the localization of the impairment. To summarize, one may say that in middle-ear involvement, there is diminished hearing of the lower pitches, that is, the lower tone limit is raised. In a nerve or inner-ear lesion, the hearing of the high-pitched sounds is first impaired, but if there is a marked involvement, both the high and low pitches are poorly heard. Tuning-forks from  $c^3$  (1024 d. v.) and upward are poorly heard, that is to say, the upper tone limit is lowered. With the Galton whistle and its modifications, the Edelmann-Galton and the Schaefer-Galton, as

well as with the monochord, the highest tones are markedly reduced. The Weber test is usually lateralized to the worst ear in conduction impairment, and usually lateralized to the better ear with inner-ear disease. (N. B. At times the Weber remains in the median line of the vertex despite a lesion in one or the other ear.) With middle-ear infections (acute otitis media) one may have the inner ear also involved due to hyperemia or collateral edema of the labyrinth, so that atypical findings may be had. In the Schwabach test the bone conduction is usually found lengthened in middle-ear disease and usually shortened in inner-ear disease. With the Rinne test we usually have a negative reaction in middle-ear disease and a positive one with inner-ear affection. (N. B.: The lower the pitch of the fork used, the more likely is the Rinne to be negative.) After catheterization of the eustachian tube, the hearing is often improved at least temporarily in middle-ear disease, but it is not affected when there is inner-ear disease present. The outstanding features are that the typical conduction apparatus impairment usually shows a diminution in hearing for the low tones, lengthened bone conduction, and a negative Rinne, whereas in inner-ear disease we are more likely to have shortened bone conduction, a positive Rinne, and noticeably reduced hearing for the high tones.

**Recapitulation of the Principal Hearing Tests.**—I. Observation of the patient. Character of the voice (usually loud in severe inner-ear disease; subdued in marked middle-ear disease).

Close attention and evident lip reading on part of patient with often appearance of anxiety in effort to hear.

Inclination of the head to one side.

Hand held at auricle to improve hearing.

II. Otoscopic examination:

Inspection of:

(1) Auricle.

(2) External auditory meatus.

(3) Tympanic membrane, thickening, scars, retraction, etc.

(4) Mucosa of tympanic cavity if perforation of drum-membrane is present.

(5) Mastoid region.

III. Nasal, nasopharyngeal, and pharyngeal examination.

IV. Testing with speech:

With the patient's eyes closed or averted and the opposite ear closed, use:

(a) Unaccentuated whisper or, if necessary,

(b) Unaccentuated conversation, employing high- and low-pitched numbers or words, and combinations of high- and low-pitched sounds. Designate in feet or meters or subdivisions thereof the distances heard, or state if *ad concham*, or not at all. If whisper is heard 5 or 6 meters away, need not use conversation.

V. Inflation with Politzer's bag or by catheter, and again test hearing with speech (whisper or conversation).

VI. Tuning-fork tests.

Range of hearing:

Lower limits from C-2 (16 d. v.) upward—*i. e.*, C-1, C, c, c<sup>1</sup>, c<sup>2</sup>, c<sup>3</sup>, c<sup>4</sup>, c<sup>5</sup>.

Upper limits:

(a) Higher forks,  $c^4$  (2048 d. v.) and  $c^5$  (4096 d. v.).

(b) Galton whistle (preferably Edelmann-Galton or Schaefer-Galton).

(c) Monochord.

1. Weber test for lateralization. Fork placed on median line of vertex, forehead, or root of nose.

(a) Normally heard in vertex ("in the head").

(b) Usually lateralized in worse hearing ear in conduction apparatus impairment. If both ears have middle-ear affection, sound goes to worse of the two ears.

(c) Usually lateralized in the better ear if disease of perception apparatus is present in other ear. If both ears have perception impairment, sound usually lateralized in the better ear.

2. Schwabach test for duration of bone conduction in the individual as compared with the normal, using living control or, better, comparing with average hearing for the particular fork employed. Fork usually placed on median line of vertex or forehead; may be set on mastoids. Note whether bone conduction is normal, lengthened, or shortened; a slight increase or diminution is of no significance.

The age of the patient, the thickness of the hair or bones, the manner of application of the fork, firmness of contact of fork, etc., may give variation in length of bone conduction.

A definite lengthening of bone conduction means impairment of the conduction apparatus (adhesions, fixation of the stapes, etc.).

A definite shortening of bone conduction means involvement of the perception mechanism (inner ear or auditory nerve).

A decided change in bone conduction is in many ways the key to diagnosis and prognosis in ear disease.

3. Rinne test, for comparison of air with bone conduction in the same individual. The stem of the fork is placed on the mastoid (avoiding contact with auricle), and when no longer heard the prongs are held close to the meatus (without touching auricle or vibrissæ), parallel with the ear, and the duration of hearing by air noted.

If a negative Rinne is suspected—*e. g.*, if the Schwabach was found lengthened, test air conduction first and then bone conduction.

Normally the Rinne is positive (air conduction longer than bone). There are about eight varieties of Rinne (two forms of the positive, four varieties of the negative, and two of the indefinite or plus-minus type).

4. Gellé test, for determining mobility of the foot plate of the stapes. Compressing the air in the external auditory meatus gives diminution of hearing in normal cases by pushing stapes into oval window; where fixation of stapedia foot-plate is present no change in hearing occurs with increase in air-pressure.

5. Stenger test, for unmasking simulation of total unilateral deafness. Two forks of exactly the same pitch used; the patient is unaware that more than one fork is sounding. The fork nearer one ear drowns out sound of fork at other ear.

6. Audiometers, resonators, and other appliances for special tests.

ROBERT SONNENSCHIN.

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### THE TREATMENT OF DEAFNESS

**General Consideration.**—Obviously before undertaking the treatment of a case of impaired hearing, a most comprehensive examination is necessary to determine the type of deafness present, the primary cause, and all complicating factors. Careful hearing tests are made and recorded for diagnosis in the first place and as a check on the future progress of the disease. A very careful history is important, and should embrace not only the previous general and aural ills of the patient but also the family history in so far as it relates to ear disease. In all cases of chronic or progressive deafness, at least a blood Wassermann test is indicated, and it is often desirable, if the former is negative or doubtful, to check up with a spinal fluid test for syphilis, irrespective of the history obtained. Careful inquiry should also be made into the patient's habits in regard to eating, use of alcohol, tobacco, or narcotics, and whether or not large doses of quinine or similar drugs have ever been taken. The history of traumatic injury may also be significant. A careful report upon the teeth in all cases and a Roentgen-ray examination by an expert should not be neglected.

Retesting of hearing acuity should be made at definite intervals and properly recorded. While the use of the audiometer is satisfactory and important for this purpose and undoubtedly is less subject to variation due to the personal equation of the examiner or examiners and to different surrounding noise conditions, it is questionable whether the older methods of testing should not also be employed and the results recorded at least occasionally.

In most acute and subacute cases where the family history is not unfavorable, the prognosis is good, and the anxiety of the patient may be frankly relieved. In the case of chronic and progressive forms of deafness, particularly those well advanced when first examined, the prognosis varies from doubtful to grave or hopeless. In suggesting treatment to a patient it is usually best to state frankly that much (or little) can probably be done to improve the hearing (or only to arrest or retard the progress of the disease), that the aurist's directions as to habits, examinations, regular

attendance, and medication must be carried out faithfully, that no definite promise can be given, that full restoration of function is extremely unlikely, that even in the event of marked improvement taking place the patient should report at intervals of six months or a year for re-examination to determine if impairment has again set in. The morale of the patient should not be needlessly shattered by brusquely giving a bad prognosis, for much depends upon the co-operation of the patient, but if a hopelessly deaf individual presents himself he should be kindly and frankly told that the time for possible improvement has passed, he should be aided in the selection of a suitable hearing apparatus, if such can be found, and he should be told to take up lip or speech reading, directed to a properly qualified teacher, and encouraged to join a club for the deaf or a local League for the Hard of Hearing.

**Acute and Subacute Cases.**—Deafness due to obstruction of the external auditory canal from cerumen, foreign bodies of many kinds, inflammation of the soft parts or bony growths can obviously only be relieved by thorough removal of the cause, in which case prompt restoration of hearing may be confidently expected in the majority of uncomplicated cases.

Where the impairment is of short duration and is due to drugs, such as quinine, alcohol, or tobacco, recovery should follow removal of the causative factor.

When deafness follows "an acute cold" due to inflammation of the pharyngeal orifice of the eustachian tube and the tubal lining mucosa, with or without exudate in the tympanum, treatment is directed first to the nose and nasopharynx as outlined in a previous chapter. Topical astringent applications to the tube mouth and an oily spray of ephedrine sulphate aid in restoring the ventilation of the middle-ear cavity, and later, when the acute stage of the infection has passed by, gentle inflations of the middle ear with the Politzer bag or eustachian catheter accelerate the return to normal. Judicious massage with a hand- or engine-driven masseur is also of benefit, these two procedures tending to restore the normal air equilibrium, to remove the products of inflammation, and to break up beginning adhesive bands of connective tissue in the tympanic cavity.

If these acute attacks recur a thorough search for the cause should be made and foci of infection in the mouth, fauces, nasopharynx, and nasal accessory sinuses removed. Tonics and alteratives are indicated, exercise and natural or artificial sunshine, proper personal hygiene and clothing, at times the restriction of sugar and starches, and often the addition of fats to the dietary. Shurly's prescription of a wineglassful three times daily of the juice obtained by pressing two raw potatoes, two raw carrots, two raw tomatoes, two raw beets, and a bunch of celery is often of value in children, particularly those who suffer from enlargement of the cervical lymph-nodes with every cold, even after the tonsils and adenoids have been removed.

**Chronic Cases (Progressive Deafness other than Otosclerosis).**—Careful hearing tests serve to determine the type of deafness, whether the middle ear or internal ear element predominates. Whereas these cases were formerly sharply divided into conductive deafness and perceptive deafness, the modern tendency is to group both types under the general term of *progressive deafness* since Emerson has shown that in most, if not all, cases of conductive deafness of long standing there are distinct signs of

impairment of the perceiving apparatus also. These are the cases that were formerly diagnosed as "mixed deafness." Emerson has taught us that most cases of deafness originate in and are perpetuated by a focus of infection, either repeated acute attacks, or a slow absorption of toxins from a chronic infection; and the nasal accessory sinuses, particularly the maxillary, the tonsils, and the teeth, are the more common causes, although the intestinal tract, the appendix, the gall-bladder, or the colon, must not be overlooked.

In instituting treatment then, it is important first of all to survey carefully these various foci and estimate their probable effect on the hearing function. Badly deviated septa, effectually blocking one or both nasal passages, should be straightened, not only to secure better ventilation and reduction of inflammation in the nasopharyngeal mucosa, but also as an aid to the elimination of sinus infection. The sinuses should be carefully studied clinically and by Roentgen ray, the latter to be supplemented by injection of radiopaque substances, such as lipiodol. Direct injection may be made in the case of the maxillary sinuses, and the Proetz method of displacement irrigation by posture and suction in the sphenoid and posterior ethmoid cells. The mere absence of secretion does not necessarily absolve a given sinus from blame since Emerson claims that an atrophic mucous membrane in the antrum of Highmore is still an infective focus. The infected sinuses, then, should be so treated as to remove them from consideration as a possible cause of deafness, even to the extent of radical operating with the removal of *all* infected mucosa. Definitely infected tonsils should be removed, as well as the adenoids where present; and all teeth that can be considered infective. Many dentists now believe that all devitalized teeth come under this category. Even the lingual tonsil should not be overlooked, as it usually is, since French has demonstrated that definite focal infection not infrequently originates here. If, after careful study of the gastro-intestinal tract, no infection can be demonstrated and the Wassermann test is negative for syphilis, the case is ready for the trial of local measures.

It is obvious that a damaged perceptive element is not amenable to local treatment other than the removal of the causative factor, which has already been done. If the damage is not irreparable or the case of too long standing, some improvement, at times even a considerable one, may result with no other treatment. Emerson, indeed, recommends that treatment stop at this point. Others, however, when the eustachian tube is stenosed by hypertrophic mucous membrane, prefer some form of local treatment. Simple inflation with the cold-air douche gives little permanent improvement in these chronic cases, but if a hot vapor of iodine, menthol, or camphor, or a combination of these drugs, can be introduced into the tube or middle ear at sufficiently close intervals, some improvement takes place at times, and the distressing tinnitus may be allayed. There are electrical heaters obtainable by which hot air under pressure may be passed over the crystals of iodine, etc., the resulting vapor being driven through the silver catheter into the tube and middle ear. Care should be taken not to overdo these inflations, and they should always be controlled by the diagnostic tube from the operator's ear to the ear being treated. Treatments should be at semi-weekly or even shorter intervals for a period of weeks, depending on whether or not improvement can

be noticed. Where the tube is of such small caliber that the vapor will not enter readily enough, or at all, the use of eustachian tube applicators and bougies is indicated. After careful shrinking of the tube mouth, an applicator of the Yankauer type is wound with a small amount of cotton for about 2 inches from the end. This is saturated with 10 per cent. cocaine solution or, preferably, with 3 per cent. ephedrine, or butyn and ephedrine combined, and passed carefully through the catheter to and beyond the tubal isthmus, where it is allowed to remain for fifteen minutes to half an hour. It is then withdrawn and replaced by another applicator loaded with silver in some form which remains for fifteen minutes, or a bougie may be passed using the largest size that will penetrate the isthmus without undue force and trauma. The bougie should remain for a like period of time and inflation should *not*, as a rule, follow its withdrawal owing to the danger of carrying infection into the middle ear through the now widely open tube. The patient should also be cautioned against forcible nose-blowing. There is little danger of infecting the middle ear by these procedures if there is no acute infection present in the nose and throat, and if ordinary attention to aseptic technic is adhered to. Gentle massage of the ear-drum with a hand- or machine-driven masseur, is usually grateful to the patient, as is massage of the external auditory meatus with a cotton-tipped applicator carrying yellow oxide of mercury ointment as practised by Walter Roberts.

For the atrophic type of middle-ear deafness, where the tube is widely patulous and the tympanic membrane relaxed, tubal inflation and massage are contraindicated. Here the patient must be carefully instructed in the art of blowing his nose without inflating his ears to cause further relaxation of the already too-mobile drum. Applications of collodion to the drum cause temporary improvement at times by tightening up this structure and making it more vibrant. Cantharides in collodion has also been used for this purpose by Heath and others in order that the resulting inflammation may cause permanent thickening and tightening of the membrane. The method is not without danger, however, as the destructive action of the cantharides upon the anemic, atrophic structure may result in perforation and middle-ear infection. Tightening of the drum may also be obtained, sometimes with marked improvement in hearing, by myringotomy, the scars resultant upon healing tending to tighten up the drum-membrane. As these usually relax after a greater or less interval, repeated myringotomies become necessary, and often sooner or later, infection takes place, the devitalized drum-head melts away and a permanent perforation results. The hearing, however, may be improved even if this happens.

As adjuvants to the treatment outlined above may be mentioned the high-frequency current and diathermy. Hays strongly recommends the former using a glass vacuum electrode of the general size and shape of the eustachian catheter, wrapped with adhesive to the tip in order to prevent injury to the patient from splinters of glass in case of breakage. The tip is passed into the eustachian tube orifice by the usual catheter introduction technic and the current applied for five to ten minutes. This is said to materially reduce the congestion of the tubal mucosa and, therefore, to aid in ventilating the middle ear.

Diathermy applied to the middle ear through the external canal by

the use of a suitable electrode seems at times to have a beneficent action upon the mucosa of the tympanum and to aid in the absorption of exudate and fibrous adhesions.

There is no evidence to prove that the Roentgen ray has been of certain value in restoring hearing in spite of extravagant claims to that effect. This method of treatment is, however, effective in reducing lymphoid hypertrophies elsewhere, and it is at least within the bounds of possibility that its use may, by causing atrophy of the glandular elements of the mucous membrane about the pharyngeal orifice of the eustachian tube, contribute in some cases to partial restoration of hearing.

**Impaired Hearing in Cases of Chronic Suppuration of the Middle Ear.**—Attempts to improve the hearing in this class of cases should not be made until the suppuration is under control and the ear dry. Failure to wait for this may lead to increased discharge, renewed activity of granulations, and possible damming back of pus into the recesses of the mastoid process.

When the ear is dry and no granulations are present hearing may often be appreciably improved by the application of an artificial drum to the perforation in the membrana tympani. The commercial drums are occasionally of use for this purpose; they are worthless for their advertised purpose of improving the hearing in cases of middle-ear catarrh where the drum is intact. A far better artificial drum, however, can be made in the office by the aurist in a moment by cutting from a piece of glazed writing paper a disk slightly larger than the perforation to be covered. When lightly moistened this disk can be applied by means of a cotton-tipped applicator to the perforation and snugly fitted over it until the margins are all in close contact with the paper. The glue in the sizing of the paper holds the patch in apposition to the skin of the drum and may continue to so hold it for weeks, after which it will have to be replaced. Hearing is often much improved and there is seldom enough irritation established to cause renewed suppuration. If the latter occurs the paper patch is loosened by the moisture from the middle ear and soon washed out.

Another, and often a better, method consists of the use of a cotton ball, cylinder or cone, applied to or through the perforation. The cotton acts best as a sound conductor or amplifier when moist. This type of hearing aid, which can barely be classified as a drum, may be used when there is still a slight amount of moisture in the middle ear, but great care in its use must be exercised as it is often irritating and may readily act as a plug to dam back the discharge. It must be removed at frequent intervals and replaced by clean cotton. When no discharge is present to supply moisture for the cotton plug it is dipped in glycerine for this purpose. By the use of this method the hearing is often sharpened to a remarkable degree, especially in those cases where the drum-membrane and ossicles have been largely destroyed and the cotton plug can be fitted closely down over the stapes or its remnants in the niche of the oval window. The patient can, as a rule, readily be taught to make and apply these plugs himself, and usually becomes proficient in adjusting them to the exact spots where they do the most good. The ear so equipped should, however, be inspected at regular intervals by the aurist to see that no undue irritation is being caused. These remedies, of course, are merely palliative. Their aim is to improve the hearing while they are in use, but

they are in no sense to be considered as attempts to cure or permanently improve the deafness.

The cotton plugs may be wound upon an applicator being shaped as a ball, a cylinder, or a cone. In the latter case it is the broad base that is applied to the inner wall of the middle ear. Ease of insertion and removal of the cone may be increased if the point of the cone, almost to the base, is dipped in collodion which is allowed to harden. This stiffening makes for ease of introduction by either the aurist or the patient and also simplifies extraction.

**Prevention of Deafness.**—There is little doubt that many, if not most, cases of progressive deafness that become manifest in adult life have their inception in neglected eustachian-tube and middle-ear inflammations in infancy and childhood. It, therefore, behooves the pediatrician and aurist to watch carefully and treat all ear complications of the acute exanthemata and infections of the upper respiratory tract and to keep them under observation until they are reasonably sure that normal hearing has been re-established. To accomplish this the ears of children suffering from such acute infections should be frequently examined and appropriate treatment directed, during the acute stages, to the nose and nasopharynx. These measures consist in cleansing the nasal passages with warm alkaline solutions introduced with a medicine-dropper, shrinking solutions such as ephedrine, and antiseptic solutions such as mercurochrome, 2 per cent., or argyrol 5 to 20 per cent. Instillations of bland oil into the nose, either plain or containing ephedrine or menthol and camphor in minute quantities, are also useful. Prevention of repeated attacks of tubal inflammation calls for careful surgical cleaning up of the nasopharynx, particularly in the region of Rosenmüller's fossæ, and appropriate treatment of nasal accessory sinus disease, as well as meticulous attention to the child's general health. After the acute attack is passed, ventilation of the middle ear should be aided by inflations with the Politzer bag and gentle ear massage. Vaccines for prophylaxis of repeated "colds in the head" often have a most beneficial effect; the prevention of these "colds" usually meaning prevention of loss of hearing at the time as well as in later life.

**Incurable Cases.**—When a very advanced case presents itself, or when all measures have been tried without success, it becomes the duty of the aurist to gently but frankly inform the patient that his hearing is irreparably damaged, that nothing further can be done to improve it, or even to retard further deterioration, and that resort must be had to sound-amplifying devices for his own comfort. In many cases it is wise to use such apparatus before this stage is reached—their use does not militate against hearing improvement and may, in fact, even aid it by stimulating the hearing apparatus and hearing centers. The final resort is speech- or lip-reading. Anyone can learn to read lips proficiently, but it requires in the first place a good teacher and long patient application on the part of the patient. The aurist should be in touch with such teachers and be able to direct his patient to such a one and encourage him to join the work of one of the speech-reading clubs or leagues for the hard of hearing where he will find much that will help him keep his morale and bear his affliction cheerfully. In some cases it is advisable to continue tube and ear treatments even after all hope of improvement has gone by and the patient has been so informed. Though hearing may not be improved by

treatment, the patient can frequently be made more comfortable by periodic ventilation of the middle ear, being relieved of the "fulness in the head" so often and bitterly complained of; and the distressing tinnitus aurium can often be relieved. In addition, by maintaining close touch with the physician in this manner, he can be encouraged, cheered, and helped in many ways.

GEORGE MORRISON COATES.

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## AIDS TO HEARING

Politzer tells us that hearing-trumpets were in use in the times of Asclepiades, 96 B. C., so it is very evident that deafness was appreciated to be such a serious handicap, even in those times, as to need relief. The endeavor to improve the action of these trumpets has led to the construction of a great many appliances of varying form and shape. In looking through the literature for the past hundred years, there is little new that is really efficient and of value until the invention of the microphone for the use of the telephone. The application of that principle for an apparatus for the deaf has stimulated an increasing interest in devices for aiding the hearing. It is very strange that there has been comparatively little that is new discovered as to the method of the propagation of sound-waves. Astonishing discoveries in other fields have been made—different pathways for the conduction of energy—and still we are supposed to depend upon waves of the air for the conduction of sound-waves. It is reasonably sure that the time is not far distant when there may be another method of reaching the center of hearing besides that of the usual middle-ear mechanism. It is generally the experience in life that when a great need arises for some device or other to alleviate suffering or to aid the general welfare, someone comes forward with just the right appliance. The profession is awakening to the fact that the thousands of deaf and deficient in hearing cannot be cured by the ordinary means at the command of the aurist; it has not, however, realized that even now there are methods for improving the hearing to a great degree. There is an increasing number of hearing instruments seen in public. An interest in deaf guilds and in the education of the deaf or hard-of-hearing child has been aroused.

When is a person deaf, or in other words, when does a person require an artificial aid? It is Politzer's opinion that when a person does not hear a spoken voice, with the aid of the lips, at a distance greater than  $1\frac{1}{2}$  meters, some assistance is necessary. It is evident that the degree of deafness which one requires depends entirely upon the individual in question; one's profession, work, or environment must be taken into consideration. Edison is said to be better able to concentrate upon his problems because of his deafness, and it is very evident that a teacher in a public school requires better hearing than an upholsterer or an artist. Then there is the stenographer, who depends upon her profession for her support, whose employer dictates with his head turned from her and there is the mother who is beginning to miss the voices of her children.

In determining the best artificial aid one should advise for a certain patient it is necessary to know whether the deafness is due to trouble in

the sound-conducting apparatus or in the sound-perceptive apparatus; if due to the former, whether low tones are cut off or only the high, with the medium voice-tones still existing. A hearing test, as accurate as possible, must be made. This brings up a subject of the greatest importance, one which has been discussed time and again, that of a uniform standard method of testing the hearing which the profession as a whole accepts. If the forks are used, and most of us do use them, they are of various makes and of various duration-vibration periods, etc. When the audiometer, as devised by Dean, Seashore, Wegel Wilson, and others, becomes perfected, both as to efficiency and ease of operation there will be a method of determining not only the hearing for the entire musical scale, but also the pressure or energy necessary for the perception of tone by the ear.

Mechanical aids can be classed as internal and external. The former are only applicable where there is a defect in the drum-membrane and where there is a movable stapes. These aids are in the form of artificial drums. Artificial drums have been used for several centuries, being mentioned as far back as 1640. Yearsly, in 1848, suggested small balls of cotton, and Toynbee, in 1852, constructed a small disk of rubber to which was attached a silver stem. There are many modifications of Toynbee's drum. Even drums of gold were used by Mueller in the ear of an American heiress. The cotton ball or flat pad, to which is applied vaseline to make it adhere, seems to be the most practical form. The patient, with a suitable pair of forceps, can soon apply the drums himself, adjusting them with great precision. Unfortunately, such aids to hearing often cause irritation in the middle ear and a discharge occurs which must be overcome before a drum can be reinserted. If the use of such a device is persisted in Nature often gives up in despair and allows the offending foreign body to remain. If the ossicular chain is intact and the perforation is not too large a paper patch or splint, as first used by the late Dr. C. J. Blake, placed over the opening often increases the hearing power. Also, a patch placed over the stapes, when the other ossicles are destroyed and the stapes is visible, has been used. Curiously enough, at times a cotton ball placed in the niche of the round window is more efficient than elsewhere. There are several rubber drums upon the market, duly advertised as curing all forms of deafness. There is no doubt that the person who invented this particular type may have been helped as there are conditions which are greatly benefited by them. The unfortunate part is that they are used indiscriminately by the public and the result is that many doctors have had the opportunity of fishing for a small piece of rubber buried in a thick discharge. There is no question but that a great many people might be helped by a little more attention and patience being paid to the use of artificial drums. It must be remembered that a very little improvement often means a tremendous comfort to some patients.

The greatest number of people who need some aid to hearing are those whose deafness is due to a non-suppurative change occurring in the middle ear and this class must require some external device to be of any real use. With the exception of a very few aids, all of which are very inefficient, such an appliance must necessarily be conspicuous. This brings us to the most difficult subject with which a physician has to deal, viz., the overcoming of pride and sensitiveness in the average patient. Some are ready and willing to co-operate, doing all in their power to help themselves,

while others have become discouraged, heedless, and actually too lazy to try any form of appliance. Education is the first step toward success; education toward the realization that everyone owes a certain amount to society, that while their deafness is a great handicap, still they have duties and responsibilities which they must assume just the same. They must be taught that their "Cross" is nothing to be ashamed of and although a device is slightly conspicuous, it is no more so than tortoise-shell glasses which we are now so accustomed to wearing.

The following tests\* were confined to tones one octave apart, over a frequency range from 128 to 4098 double vibrations. It is unnecessary to give the details of the method. The measure of the efficiency of an instrument depends upon its power to increase the intensity of the energy of sound waves and this intensity varies directly with the square of the amplitude of the vibration for that particular tone. The efforts, therefore, are directed to the best method of increasing sound amplification. Nearly all of the ear-trumpets are more or less complicated modifications of the open conical horn. The principle is that of a collector of sound waves, introducing a greater number into the ear than can be introduced by the external ear. Recent investigations have shown the horn to be a sound resonator as well, with its own natural pitch and has the power of amplifying a wide range of tones higher in pitch than its own fundamental tone. As might be expected, the larger the opening and the longer the instrument, with certain limitations, the more efficient it is. These charts all show there is a fairly sharp rise at  $c^3$  and a more sudden dropping off when the vibration period of  $c^6$  is reached. Upon the charts, along the base, are the vibration frequencies, and the figures upon the perpendicular line show the increase or decrease of the loudness produced by the particular instrument over the loudness perceived by the unaided ear. This is called zero.

Figure 283 is a chart of a simple horn "A" with an opening of about  $2\frac{1}{2}$  inches and about 10 inches long. The line marked "B" is that of an oval metal trumpet with an opening about 3 by 5 inches and about 20 inches long.

Figure 284 is a chart which shows the intensity of sound conveyed to the ear by a complicated horn having a reflecting surface. The efficiency falls off suddenly at  $c^3$ . The line marked "B" shows the efficiency of the bare hand held back of the ear. It is really quite an efficient resonator and is as efficient as many of the smaller horns.

Figure 285 is a chart which shows the efficiency of two speaking tubes. One is a large tube with an opening of about  $2\frac{1}{2}$  inches in diameter and the other is a small flexible metal tube, the mouthpiece being about 1 inch in diameter.

Figure 286 is a chart which shows the efficiency of a curved horn containing a vibrating diaphragm. The line marked "B" indicates the efficiency of a hard-rubber fan which is curved and held between the teeth. Of course the vibrations are conveyed to the inner ear by bone conduction.

It may be seen from these charts that the most efficient horns are those with the largest openings. Because of their appearance and size their limitations are soon reached. Their use, therefore, is confined to almost personal intercourse, and they are of but little value as a real aid to the

\* In discussing the efficiency of the various devices to aid the hearing, I am indebted to the work of Dr. Paul E. Sabine of the Riverbank Laboratories, Geneva, Illinois, whose publication appeared in the *Laryngoscope*, 1921.

hearing. Speaking tubes of fairly large diameter are of value for the very deaf as they eliminate the necessity of shouting into the person's ear. An ordinary pasteboard mailing tube is really an excellent speaking tube. The fan, and one can use an ordinary Japanese fan, with a piece of celluloid upon the edge against which the teeth are placed, is very efficient where there is a very strong negative Rinne test.

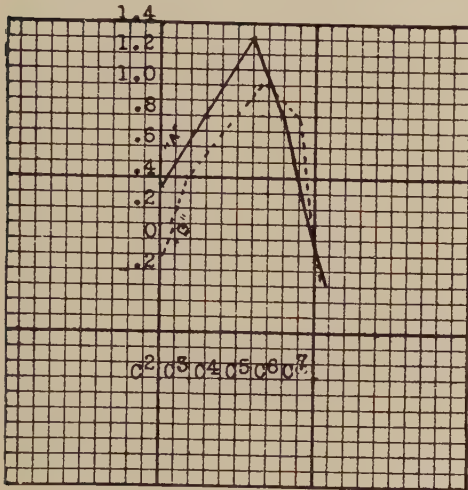


Fig. 283.

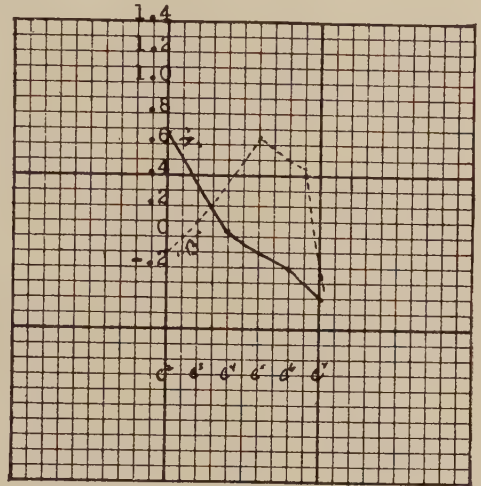


Fig. 284.

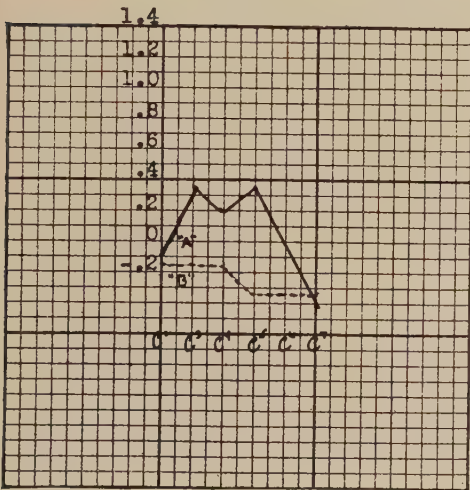


Fig. 285.

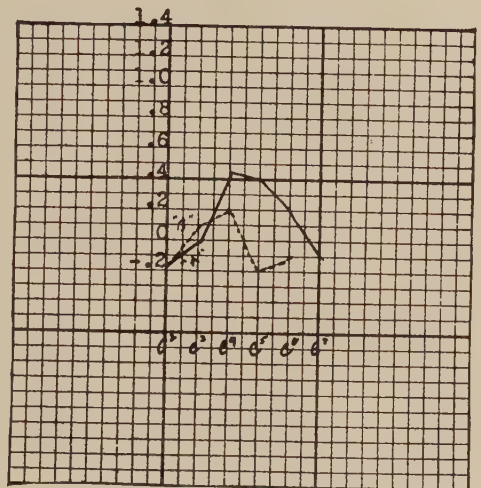


Fig. 286.

The most efficient hearing appliances we have at the present time are of the telephone type. They are far from perfect, but the strides which have been made since the first microphone was placed on the market for the deaf are so great that almost anything is possible. The fact that the physicist appreciates the necessity of the co-operation of the otologist is encouraging. There are several excellent instruments available for the average deaf person if he will only "put his pride in his pocket" and try to learn to use them. If patients could appreciate the great change in the

lives of many people, occasioned by the use of some instrument, they all would take courage and "go and do likewise."

In advising the use of an instrument, the patient's sensitiveness to outward appearance must be overcome. He must be made to understand that by continuing in his present condition he is his own worst enemy. He makes his own life miserable and also causes intercourse with his friends most difficult. If a patient can be persuaded to try an instrument, usually the result is so startling that he immediately appreciates the help and finally educates himself in the use of it. He must be told that it is an artificial ear, subject to many defects in the proper amplification of the various voice tones, that one must concentrate upon the particular voice or thing he wishes to hear, and learn to exclude extraneous sounds. The re-education possible with an instrument is often very marked. A great many persons give up the use of one ear, using the better one entirely. It is often possible to increase the hearing power in the other by an ap-

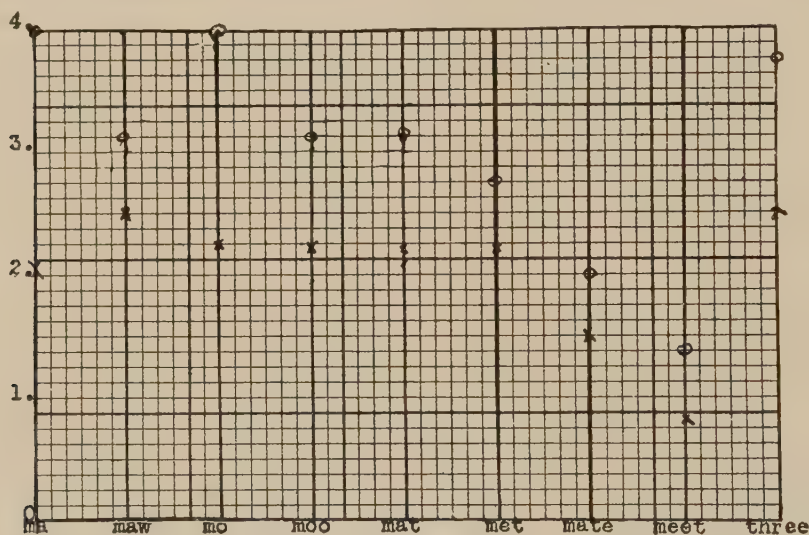


Fig. 287.

pliance. The only objection to the use of such a hearing device is the fact that they are more or less conspicuous, but in time the vast numbers seen will accustom everyone to them, so they will not be noticed.

The question of fatigue has been mentioned. Any deaf person suffers from the "strain of trying to hear" and if an instrument is used at too long periods, fatigue will result, but it is only temporary. It is usually customary to use an appliance for the first few times only for a few minutes each day and thus gradually accustom themselves to it.

There are two systems of instruments upon the market. The application of the amplifier, so commonly seen in radio sets and used for wireless telephony, has made possible the amplification of sound-waves to almost any extent desired. The problems of weight and expense are being solved. At present one can procure an elaborate stationary set with several amplifiers and a double receiver, or the small portable set. Then there are a large number of small pocket devices of the carbon transmitter type with which you are all familiar. These vary in design more or less but all are

built upon the same principle. Several firms manufacture instruments of different sizes and of varying intensities. Some are intended for home use, others for office, and still others for lectures, theater, etc. Nearly all give an unpleasant noise, which is inherent in the instrument. This noise may vary in different forms and also vary at different times in the same instrument. The following chart (Fig. 287) shows the efficiency of one amplifying instrument (marked O) and a composite of three of the well-known makes of carbon transmitter type (marked X). It is seen that the efficiency for the different vowel sounds varies greatly. This is due to the fact that the frequencies of voice sounds vary tremendously and are the most complicated of all sounds. A microtelephone has its own fundamental tone at a frequency say of 1000. Sounds about that frequency will be transmitted clearly, but other tones and combinations of tones will suffer from distortion.

The Bureau of Standards at Washington has examined a number of the standard makes of non-electric and electric aids to hearing. This most valuable work was made possible through the efforts of Dr. Douglas Macfarlan, chairman of a committee appointed to survey the instrumental aids to hearing. It is impossible to give even a summary of the report here, but the results can be obtained from any of the various Guilds for the deaf and from otologists. The full report can be found in the October number of the *Volta Review*, 1927. At the present time it is unnecessary for the really deaf person to be resigned to a life of silence, unless it be a type of advanced nerve deafness. There are instruments of both the non-electric and electric type which will aid nearly every case to a certain degree. These instruments can usually be seen and a test made in any one of the Guilds for the deaf. It is to be remembered, however, that such a hearing device, without lip-reading, will not be entirely satisfactory. I cannot urge too strongly the necessity of everyone learning lip-reading, even if it be only a little.

D. HAROLD WALKER.

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## OTOSCLEROSIS

**Definition.**—Otosclerosis is a new osseous formation affecting primarily the bony capsule of the labyrinth. Clinically, the term is commonly applied to that type of the disease with stapes ankylosis, characterized by progressive deafness, usually bilateral, severe tinnitus, normal drum-membranes, patent eustachian tubes yet demonstrating the Bezold triad.

**Synonyms.**—The name adopted is the one in general use. Others are: Stapes ankylosis, dry catarrh of the middle ear, hereditary deafness, spongification of the labyrinth capsule, chronic metaplastic otitis of the labyrinth capsule, osteitis vasculosa.

**History.**—Stapes ankylosis was first observed by Valsalva, in 1724, and later by Morgagni, in 1761. Toynbee, in 1857, completed his wonderful dissections and preparations illustrative of diseases of the ear. Four years later, Moos, in 1861 demonstrated that the condition could occur without disease of the middle ear mucosa. Bezold, in 1883, proved the accuracy of his diagnosis by microscopic examination, and Politzer shortly after-

ward stated that it was a primary affection of the bony labyrinth capsule. Later, Katz, Siebenmann, Manasse, Denker, Brühl, and Pierce laid the foundation of our present histological knowledge.

**Etiology.**—The numerous theories advanced as to the etiology of otosclerosis prove that not one is wholly satisfactory. It is commonly a disease of the young adult. The first symptoms of tinnitus or deafness generally appear between the fifteenth and thirtieth years. Females are affected more frequently and usually more severely than males. Over 50 per cent. give a family history of non-suppurative deafness, and, like other conditions of a hereditary nature, it tends to be transmitted through the female line. The best known hypotheses as to its nature are, viz.:

1. It is due to a biological variation or degeneration of the otocyst or of the auditory nerve causing an abnormal growth of the labyrinth capsule.

2. It is a primary affection of the bony capsule of the labyrinth due to (a) abnormal continuance of the process of growth in the petrous bone, or (b) lack of proper vitamins during the embryonic growth or in early childhood, or (c) abnormal vascular disturbances in the capsule.

3. Others hold it is secondary to a chronic inflammatory process of the mucoperiosteum of the middle ear, either catarrhal or suppurative.

4. It is due to toxemia, circulatory disorders, traumatism, aseptic infarcts, or anemia.

5. It is a non-malignant congenital new-growth, activated by numerous conditions.

6. It is due to an endocrine anomaly.

7. That three factors are called into action: (a) Constitutional, such as mesenchymal disturbances secondary to disfunction of the ductless glands, or heredity. (b) Unstable bone lying between the primary cartilage canals and the secondary radiating bone vessels. (c) An exciting component such as the female disturbances of adolescence or pregnancy.

**Symptomatology.**—The chief symptoms of otosclerosis are progressive deafness, tinnitus, and paracusis willisiana. Vertigo, and pressure or pain in the depths of the ear may be present.

1. *Deafness.*—The loss of hearing in otosclerosis is usually bilateral, insidious in onset, and is first noticed in early adult life. It is difficult to ascertain when the deafness first appeared. The patient may maintain that the head noises interfere with otherwise normal hearing. Testing with the audiometer, however, will show loss of hearing already present. Periods of quiescence in the progress of the deafness are not uncommon. Repeated exacerbations induced by pregnancy, colds, chills, overfatigue, and anemia cause marked temporary deafness. These losses are never completely regained and the deafness continues to increase. One ear may be affected before the other, but symmetry in their hearing curves is commonly present. The weather conditions seem to have no effect upon the hearing.

2. *Tinnitus.*—The head noises are even more disturbing than the loss of hearing and may precede it by months. They are present in 80 per cent. of cases and vary with the severity of the case. The tinnitus may be of three types: (a) A hissing or buzzing due to slight changes in labyrinthine pressure; (b) a humming or pulsation due to vascular sounds transmitted from vessels in the new-formed bone or from vessels close by because of the increased bone conduction; (c) a roaring or thundering

due to mechanical or chemical changes in the labyrinthine fluids or interference with its blood-supply.

3. *Paracusis Willisiana*.—The apparent ability to hear conversation better in a noise, especially on a train, is constantly present in stapes ankylosis. Noises of low vibration being shut out by the rigidity of the stapes disturb the otosclerotic but little. The normal-hearing individual, compensating for these noises, will raise his voice and will be heard more distinctly by the patient. Its presence signifies middle-ear disease with an unfavorable prognosis—usually stapes ankylosis.

4. *Vertigo* is present in 10 per cent. of cases of stapes ankylosis. Unsolved cases of primary vestibular vertigo may be explained by small otosclerotic foci of the semicircular canals.

**Pathology.**—The essential pathology of otosclerosis is absorption of the normal bone of the labyrinthine capsule and its replacement by a new

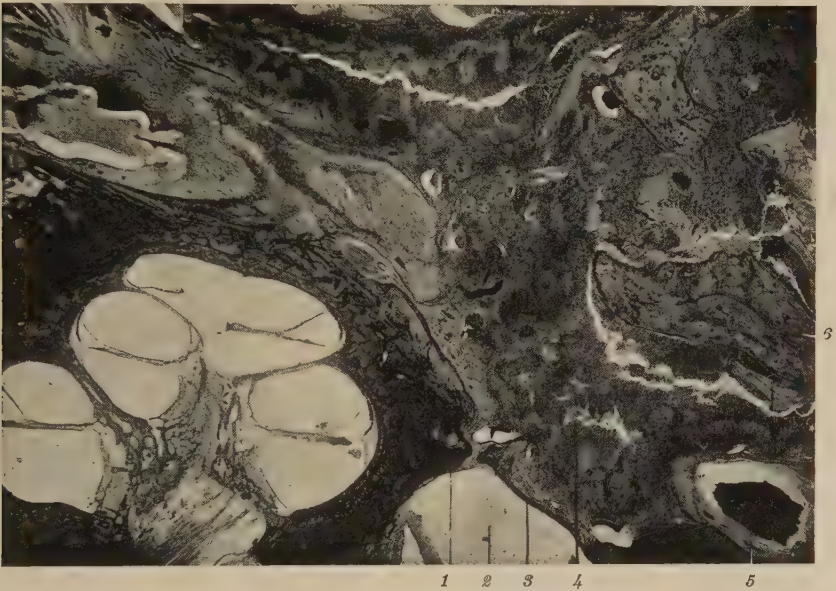


Fig. 288.—Tuberculosis of the middle ear in an infant. Horizontal section. 1, Cartilaginous rests in fissura ante fenestram. 2, Utricle. 3, Foot-plate of the stapes. 4, Middle ear. 5, Facial nerve. 6, Incus.

type of bone formation. Any part of the labyrinthine capsule may be affected, *e. g.*, the bone about the oval window, especially in the area adjacent to the fissura ante fenestram,\* or the round window, or the walls of the cochlea, or of the semicircular canals, or even of the internal auditory meatus. The sites of these foci, however, bear a close relationship to the fissures which separated the primary centers of ossification in the embryo.

The bony changes may affect the capsule in one or more small foci, or diffusely, and are usually symmetrical in both labyrinths. This is true not only when the foci are near the anterior margin of the oval window, but also when present in the walls of the internal auditory meatus, or at the apex of the cochlea, or in the areas about the semicircular canals. The

\*The fissura ante fenestram (Siebenmann) is also known as Cozzolino's zone or the fissura anterior.

point of predilection is the anterior margin of the oval window. With any marked new-bone formation in this area, involvement of the annular ligament and fusion of the stapes foot-plate to the margin of the oval window

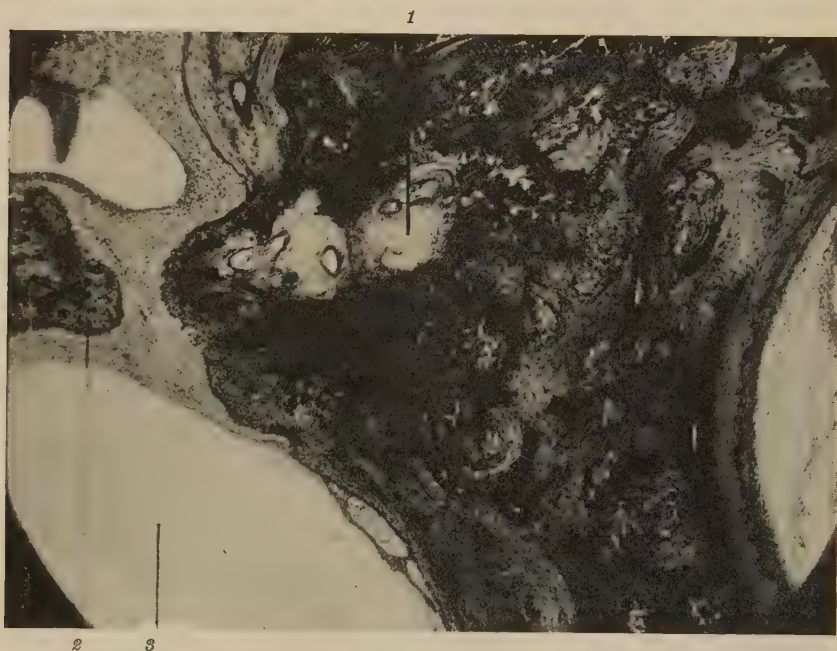


Fig. 289.—1, Cartilaginous rests in fissura ante fenestram. 2, Foot-plate of the stapes. 3, Utricle.

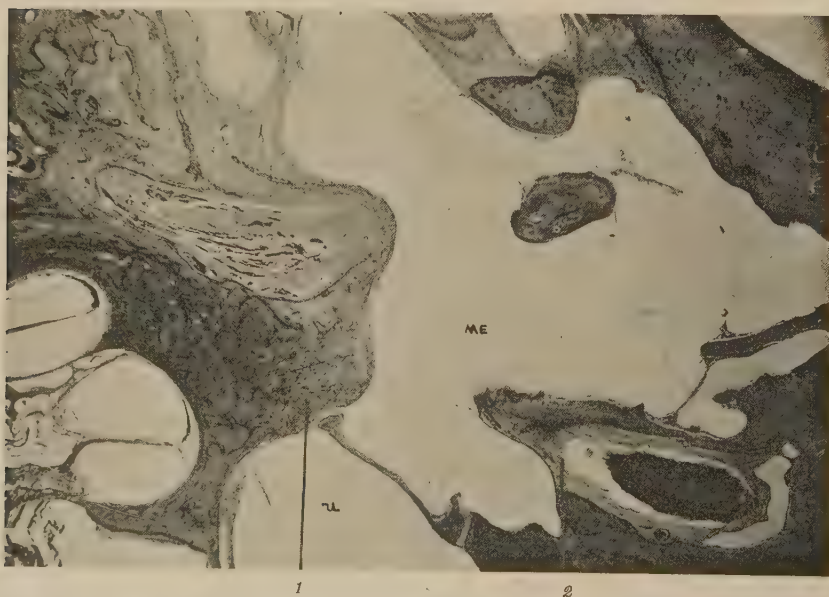


Fig. 290.—Horizontal section showing otosclerosis of anterior margin of oval window without stapes ankylosis: 1, Otosclerotic bone. 2, Facial canal showing dehiscence of bony wall. U, Utricle. ME, Middle-ear cavity.

must result. Unless this involvement and fusion take place, otosclerosis of the middle-ear type, the common clinical form of the disease, will not

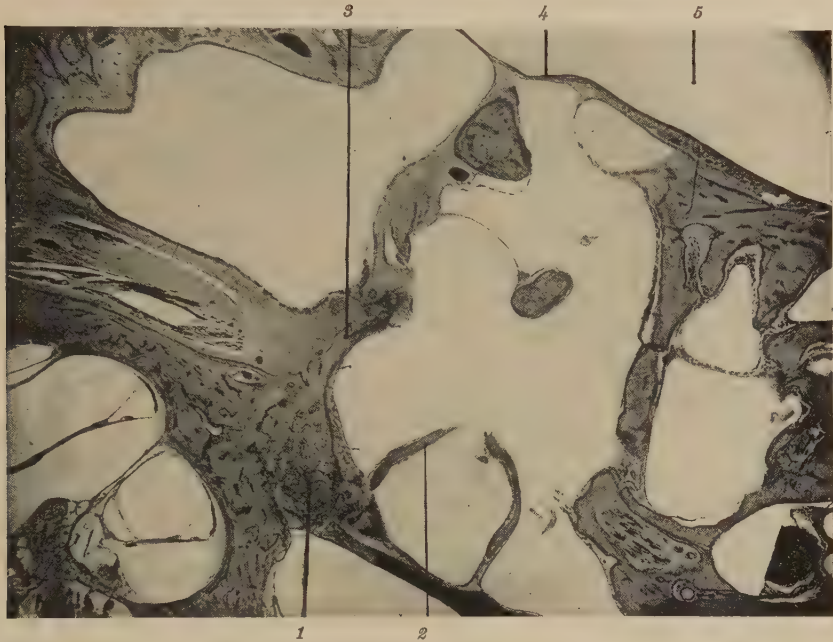


Fig. 291.—Horizontal section: 1, Otosclerotic area. 2, Anterior crus of stapes. 3, Processus cochleariformis. 4, Drum-membrane. 5, External auditory canal.

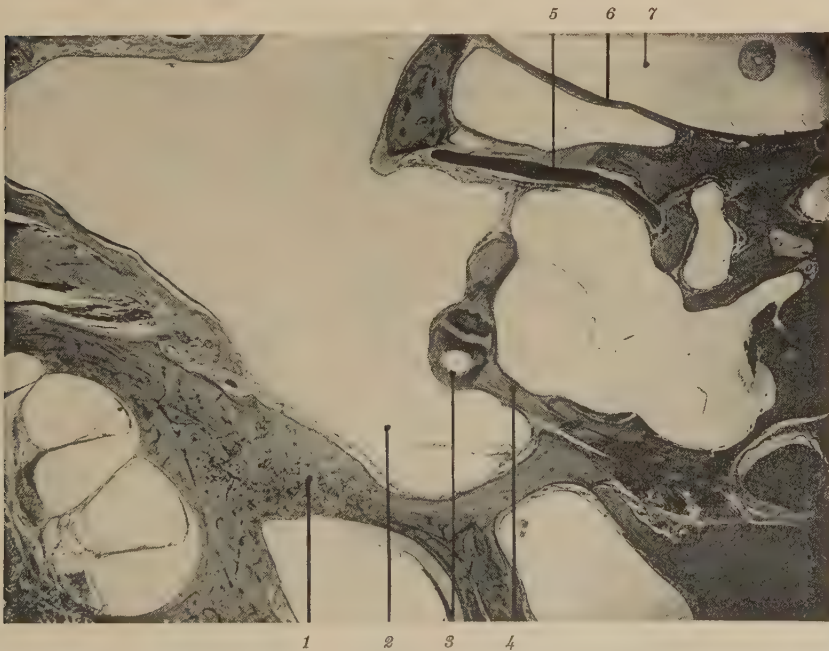


Fig. 292.—Horizontal section: 1, Otosclerotic bone. 2, Middle-ear cavity. 3, Head of the stapes. 4, Tendon of the stapedius muscle. 5, Chorda tympani nerve. 6, Drum-membrane. 7, External auditory canal.

be present. Yet stapes ankylosis is only an accident in the disease complex and takes place because of its anatomical position.

This area about the fissura ante fenestram is one of the late points of ossification and bilateral cartilaginous foci, or rests, are found here in 5 per cent. of normal individuals. These rests are composed of non-calcified cartilage and are quite different from the interglobular spaces of calcified cartilage found throughout the normal labyrinth capsule. This is also the point subjected to constant irritation both from the pull of the tendon of the tensor tympani above and the movement of the anterior crus of the stapes, posteriorly.

Microscopically, the new bone manifests two distinct types of osseous growth: (1) A pink, rose-colored bone, due to an affinity for eosin, with thick osteoid beams and small marrow spaces; (2) a deep blue hematoxylin-staining spongoid bone consisting of a network of bony beams with large marrow spaces.

In slow advancing cases of otosclerosis the pink-colored compact bone with little cell activity predominates, while in the rapidly progressing cases, especially in the young, the blue-staining bone with large tissue spaces is found. Neither type exhibits the interglobular spaces of calcified cartilage so characteristic of the normal labyrinth capsule.

The bone changes group into four cycles: 1. A primary new-bone formation. It consists of a network of blue-staining osteoid beams with small triangular or irregular-shaped bone corpuscles. The cavities between these beams contain young connective tissue embedded with round, fusiform, and cubical cells; capillaries; osteoblasts, and occasionally a giant osteoclast, but no large blood-vessels. Nucleated red cells, myelocytes, and fat cells, so characteristic of the red bone marrow, are not found in these spaces. This type is found in the very young or in very rapidly progressing forms of the disease.

2. A secondary absorption of the normal capsular bone of the labyrinth. The advancing new bone by its pressure on the normal capsular bone and its blood-vessels produces a simple atrophy of the old bone without signs of cell activity. Howship's lacuna resorption with giant-cell osteoclast is rarely found at the boundary zone of the normal old capsular bone and the new-growing bone.

3. A resorption of the new blue-staining bone. This resorption takes place through the joint action of a canaliculization process in which the bone cell cavities distend, communicate, and form canals in the bone. Giant-cell osteoclasts with Howship's lacuna formation appear and aid in the resorption.

4. A replacement of the new blue-staining bone by a new pink-colored bone. This pink, eosin-staining bone is laid down by osteoblastic activity. It consists of a network of thick bony beams with nearly normal bone corpuscles. The marrow spaces contain connective tissue with lymphocytes, spindle-cells, and small blood-vessels. In the older areas of pink-colored bone, the bony beams become thicker and more compact, with lamellar systems developing around blood-vessels.

These different types of new bone may represent different intensities, or rates of growth in the new forming bone, rather than cycles in its development. The four changes may take place simultaneously in different parts of the affected area.

**Diagnosis.**—A diagnosis of otosclerosis is ordinarily made in the most common type, *i. e.*, with stapes ankylosis. Yet any part of the labyrinth capsule may be affected by the new-bone formation. The signs and symptoms in these types differ, but a family history of progressive non-suppurative deafness is a vital link aiding in the diagnosis of all.

Five types of otosclerosis must be considered:

*Type I.—Otosclerosis with Stapes Ankylosis.*—The diagnosis of otosclerosis with stapes ankylosis is based upon a history of progressive loss of hearing with tinnitus, usually bilateral, of insidious onset in early adult life, a practically normal tympanic membrane, patent eustachian tubes yet demonstrating the Bezold triad. This triad consists of lengthened bone conduction, a markedly negative Rinne test, and elevation of the lower limit. Paracusis willisiana, or apparent ability to hear conversation better in a noise, and the pinkish drum reflex, due to hyperemia of the mucous membrane over the promontory, are pathognomonic of this type. The former is, in part, a measure of the stapes rigidity, while the latter is a sign of intense otosclerotic activity. Both the Rinne and Gellé tests are negative. The upper tone limit may appear normal with the Galton whistle and the audiometer curve be of the middle-ear type, yet it usually shows beginning involvement of the upper limits.

*Type II.—Otosclerosis with Stapes Ankylosis and Nerve Involvement.*—As the new-bone formation in Type I penetrates the capsular wall so as to affect the endosteum of the labyrinth, there is a marked decrease in the previously lengthened bone conduction of Type I, and a decided lowering of the upper tone limit. This is the usual second stage in the course of the primary stapes ankylosis. The tinnitus is usually increased. The audiometer will show a combined middle-ear and nerve disease curve. The Gellé test is negative and the negative Rinne is decreased and may become positive.

*Type III.—Otosclerosis with Primary Nerve Deafness.*—Primary cochlear involvement will be diagnosed when primary nerve deafness, without apparent cause, is found in a young adult of an otosclerotic family group. The onset may be insidious or abrupt. Tinnitus is usually present. A pink promontory glow, if evident, would clench the diagnosis, even in the absence of a family history, and rule out toxemia and tumor of the nerve fibers. The audiometer curve will show pure nerve involvement. Both the Rinne and Gellé tests are positive.

*Type IV.—Otosclerosis with primary cochlea involvement* and late stapes ankylosis is a second stage of Type III. The late appearance of a negative Gellé test with raising of the low limit and possibly an increase in bone conduction in a case of pure nerve deafness would typify this group.

*Type V.—Otosclerosis with primary vestibular involvement* may explain many cases of vertigo without apparent cause. Nerve deafness is almost a certain later development.

In all types, the common point is the triad of progressive deafness with tinnitus in a young adult of an otosclerotic family group.

**Prognosis.**—The prognosis in otosclerosis is very unfavorable, as the pathology indicates. This is particularly true (*a*) when the symptoms appear before the twentieth year; (*b*) when the loss of hearing is rapid and the tinnitus severe; (*c*) when the heredity tendency is strong; (*d*) when the red promontory reflex is marked; (*e*) when early involvement of the

internal ear is present. If the loss of hearing has been slow in recent years, and the bone conduction still increased, the process may have limited itself. These patients retain some conversational hearing and ability to use the telephone. Such a prognosis has an excellent psychological effect.

**Prophylaxis.**—This disease appears to be an ossification anomaly. The onset of an otosclerotic change must take place at least five to ten years before it develops sufficiently to produce the first symptoms of deafness or tinnitus. Ossification in the residual fissures which in earlier embryonic life bounded the primary centers of this process in the labyrinth capsule is not always complete at birth. Hence a latent, if not an active, state of the disease is present at birth. The importance to an expectant mother and offspring of a proper diet as to vitamins and calcium is obvious. The vitamins in cod-liver oil, green vegetables, milk, together with sunlight, so essential to all children, would favor normal completion of this ossification in the young with an otosclerotic family tendency.

The advantage of informing the young woman with otosclerosis of the dangers of marriage with probable pregnancy is an open question. The mental depression already present due to the deafness and tinnitus is much increased and, moreover, the condition commonly progresses to a severe degree even without pregnancies.

**Treatment.**—Local therapy, unless the condition is complicated by middle-ear catarrh, is useless. The improvement obtained at times by use of the Roentgen ray, vaccines, the mercury quartz light, and similar lamps is due to their quieting action on the lymphoid tissues about the eustachian tube rather than any possible effect on the otosclerotic foci.

Excessive zeal in treatment may result in much harm. The value of intratympanic injections of warm air, pilocarpine, dionine, pepsin, etc., is practically negligible. Passive movements as exemplified by various methods from simple tragus massage and Lucae's probe to the various electrically driven masseurs have been of little aid.

Operative measures, including mobilization and extraction of the stapes, removal of the drum-membrane, the malleus, and the incus have not proved of lasting value.

Infection shows a tendency to activate the process. This is more evident in middle-ear suppuration. Hence, tonsillectomy and adenectomy must be considered upon the slightest provocation.

No hard-of-hearing patient is quite as deaf as the hearing test indicates unless total hearing is lost. Suppression or inattention deafness is always present in addition to the organic loss of hearing. Whatever improves the general condition enables this latent hearing to be utilized. It may be the mental stimulation of hope advanced by the charlatan of finger massage, or the general well-being brought about by regulated diet and exercise.

Any metabolic abnormality due to endocrine malfunction should receive the proper attention of the expert. Accurate diagnosis in such cases is essential and only available in the properly equipped laboratory. In a series of over 100 otosclerotic cases, Drury<sup>1</sup> found endocrine disturbance in two-thirds of the number, including the thyroid, pituitary, and the gonads. Lawrence<sup>2</sup> states that 14 out of 19 cases, or over 73 per cent., were improved by endocrine extracts. The patients with thyroid disturbances showed the most improvement. The pituitary cases were

slower in responding and the ovarian cases least satisfactory under treatment. Although a large percentage of otosclerotic patients show evidence of malfunction of the ductless glands, the incidence of otosclerosis in endocrine pathology is small.

Anemic conditions should receive all the advantages of a liver diet, ultraviolet radiation, iron medication, and cod-liver oil.

The most commonly used drugs have been phosphorus, calcium, and sodium or potassium iodide. The good obtained by their use may be due to improving the general health as well as any specific action on the bony process.

The severe tinnitus at times demands the aid of luminal or bromides. The patient should be advised of the fact that the head noises will gradually decrease in time and are never the forerunner of insanity. Companionship and a hobby to fill up unoccupied moments are its best therapeutic agencies.

The ear-trumpet and speaking-tube are of aid in all types of middle-ear and nerve deafness. Their bulk and appearance prevent their more general use. The electrical devices are many. The middle-ear deaf, with increased bone conduction, seem to profit more by their use than do patients with pure nerve deafness.

Lip-reading should be recommended at an early date. It is of great value in the prevention of both the mental depression and inattention deafness common to this disease.

HARRY P. CAHILL.

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#### TREATMENT OF CHRONIC SUPPURATION OF THE MIDDLE EAR

The accumulation of a serous exudate within the tympanic cavity affords an excellent culture-medium for bacterial invaders. After such infection occurs there is marked inflammatory reaction of the tympanic lining; and the turbid transudate, containing exfoliated epithelium, polymorphonuclear leukocytes, and bacteria, is released from the drum-cavity by rupture of the tympanic membrane or by myringotomy. Swelling of the eustachian orifice commonly prevents spontaneous drainage into the nasopharynx. Suppuration thus becomes established, and if it continues for more than three to six weeks it is classified by most authorities as chronic.

Treatment of middle-ear suppuration which persists longer than a few days includes some of the most tedious problems of otology. Tremendous difficulties are combined in the management of this cavity, set deeply in the bone among structures of the most vital importance, yet exposed to bacterial invasion along two distinct avenues. Reinfection of healing mucosa is frequent, and the relatively pure cultures disclosed on myringotomy will usually be found changed to a very different and varied flora after the lapse of a few weeks.<sup>19</sup>

The virulence of certain bacterial strains, notably of the hemolytic types of streptococci and staphylococci, *Streptococcus viridans*, and *S. mucosus*, is an important factor in the rapid invasion and prolonged course of such infections. Impaired physical resistance, general or local, must always be reckoned with in the treatment of aural suppurations. Such loss of resistance may be due to direct blood-stream invasion, or to the circulation of soluble toxins from other foci, with definitely destructive effects upon the phagocytic power of the blood, and even with a distinct loss of red cells and a marked loss of hemoglobin. Toxins of scarlet fever, measles, diphtheria, whooping-cough, and those of pneumonia, typhoid, tuberculosis, and syphilis, as well as the metabolic interference of diabetes, have each a special influence upon intratympanic invasion. Treatment of such general conditions requires occasional examination of the ear, with a view to the institution of prophylactic measures in time to avoid serious involvement.

Anatomical conditions which favor chronicity of aural suppurations are those relations which impede free drainage, notably the pouching of the tympanic lining and its reduplication by transverse bands and folds.

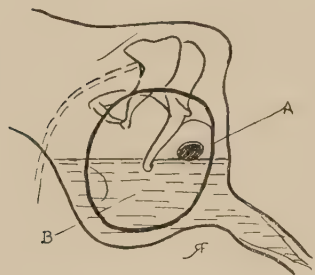


Fig. 293.—Schematic drawing of right middle ear viewed from without. A, Small high-placed perforation; B, exudate filling lower half of drum.

Across this involved series of cavities the ossicular mechanism swings in its ligamentous attachments. Treatment must take cognizance of these remote interstices, which may readily be closed off by swelling of their mucosal linings. Bacterial invaders grow in a thick turf upon such ill-drained soil, producing gas and toxins which carry the infection further. Maceration of the epithelial lining, cell death, and exfoliation deprive the mucosa of its original power of phagocytosis.

Objects to be attained by treatment include: Securing and maintaining free drainage, determination of the type of infection, aseptic cleansing, antiseptic treatment, removal of local or general causes of reinfection, drying up of discharge, and restoration of hearing.<sup>3, 4, 10, 12</sup>

The type of tympanic perforation existing in a given case affords a rather important index to the ease with which drainage may be effected.

Very frequent is the small opening, especially in the upper part of the membrana tensa, too small to permit inspection of the drum-cavity (Fig. 293). Such perforations occur in subacute suppurations rather than in long-established chronic processes. Granulations are not in evidence. Continuation of the discharge is favored not only by the small size and relatively unfavorable location of the high-placed perforation, but often, also, by an inferior general physical condition of the patient. Such perforations generally require enlargement, or the substitution of another incision better placed for drainage.

A second type includes those large openings with visible granulations, which may assume elongated or polypoid forms (Fig. 294). Here treatment must be directed toward removal of the granulation tissue, as a prerequisite to smooth healing. Such treatment, usually surgical, must be completed by caustics, antiseptics, and as soon as possible by dry swabbing

in place of fluids. Relapses are not infrequent, and are usually traceable to areas of granulation not readily accessible through the existing perforation.

Large perforations through which polyps or granular tissue protrude, arising from regions inaccessible to ordinary means of removal, form a third and very trying group (Fig. 295). These inflammatory tissues may spring up from the excoriated tympanic floor, far below the level of the auditory meatus. They may betoken involvement of hypotympanic, zygomatic, or eustachian groups of cells. More often they spring from the mucosal pouches about the heads of the major ossicles, from the roof of the attic, or from the aditus. If right-angled curets cannot reach and destroy such foci, drainage by ossiculectomy or by radical procedures through the mastoid will become necessary.

Perforations of the membrana flaccida are often very tiny, occurring above or near the short process of the malleus; but they often extend into

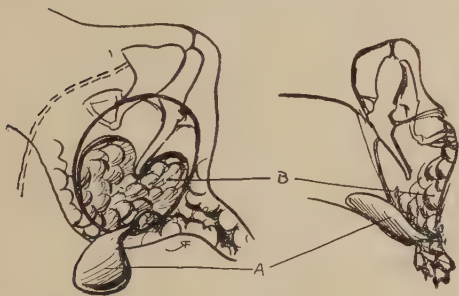


Fig. 294.—Right middle ear viewed from without, and from before backward. Large kidney-shaped perforation below; the promontory, hypotympanum, and tube mouth filled with granulations. (B) A small polyp (A) protrudes into the meatus.

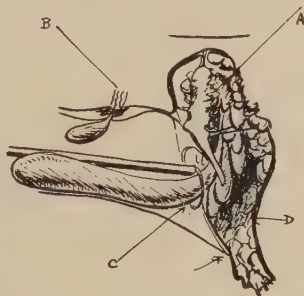


Fig. 295.—Right ear from before backward. Caries of head of malleus, incus, and inner wall of attic (A). Granulations and polypi line the drum (D); one large polyp extends along canal, but cannot be removed entire by snare loop (C). Superoposterior polyp from mastoid fistula shown for comparison at (B).

the outer wall of the epitympanum (Fig. 296). Such openings, often masked behind an apparently innocent flake of cerumen, are the portals of the most chronic and perhaps the most dangerous and insidious form of ear suppuration. They are most frequently responsible for invasion of the crowded and complicated region of the attic and aditus by cholesteatoma.

Large perforations through which a smooth-walled tympanic cavity is readily visible, filling up rapidly with a copious discharge, give rise to the suspicion that other cavities besides the drum are involved. In such cases the middle ear simply forms part of the fistulous tract through which pus from the mastoid, or rarely from the tegmen tympani, is being drained (Fig. 297). Comparative roentgenograms of the two temporal bones will throw much light on suspected cases of this type.

Multiple perforations, with free discharge and slight reparative reaction, are suggestive of tuberculosis. Occasionally multiple splits and tears may be found subsequent to explosive accidents, where secondary infection may readily bring about the usual inflammatory changes.

Drainage, the first object of treatment, will be hampered by any obstruction in the external canal, as by cerumen plugs, or by exostoses. Block-

ing of an existing perforation by inspissated pus, or by epithelial scales, or, in the process of continued discharge, by wads of macerated skin-flakes, will bring about temperature rise and necessitate prompt measures for cleansing. Close to the drum, such detritus needs careful softening by hydrogen peroxide solution, and removal by syringing, or by careful use of dull ring-curets and forceps.

Uphill drainage, in consequence of high position of the perforation, especially when located in the membrana flaccida and through Prussak's space, is a most potent factor in spreading the infection through delay in healing and retention of secretions. Such perforations in subacute cases may well be supplemented by incision in the pars tensa as elsewhere directed. Pouting out of the intratympanic mucosa through these upper perforations may bulge downward over the short process and even the manubrium. Teat-like perforations and those presenting a throbbing bead of exudate are highly dangerous, and may call for repeated myringotomy, suction, and other methods of removal of pus.<sup>14</sup>

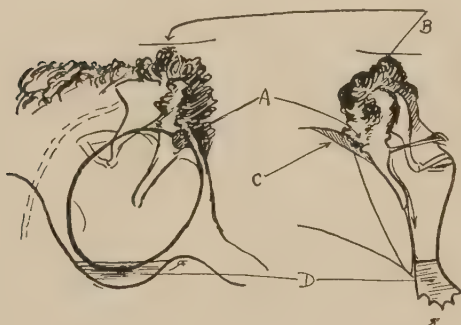


Fig. 296.—Right ear, from in front and outward. Small perforation above short process (A), draining an extensive carious process of attic and ossicles (B). Perforation hidden by dried exudate (C). Liquid seepage has collected in hypotympanum (D).

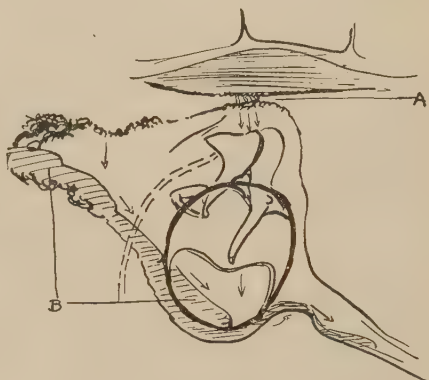


Fig. 297.—Right ear from without. Dehiscences through the tegmen tympani with an epidural abscess are shown (A). Pus flowing down from antrum and aditus involvement (B). Tympanum not affected, walls smooth.

External suction is a valuable adjunct to drainage if cautiously applied.<sup>5, 6, 7</sup>

Swallowing, with nostrils closed, after Toynbee, is often useful in starting exudates backward down the eustachian tube. Catheterization, or the procedures of Politzer and Valsalva, should not be used unless a free opening is present, and under the personal responsibility and supervision of the otologist. Too frequently the enthusiastic beginner is tempted to blow pus out of an ear by compressed-air pressure, with gratifying immediate results, but with disaster later from coincidental blowing of infectious material into the aditus and thus to the mastoid antrum. Indeed, many otologists and pediatricians hold that blowing the nose should be forbidden in many cases of subacute aural suppuration; removal of postnasal secretions should preferably be done by hawking and spitting, until the aural condition is better.

Swimming should be forbidden also to persons with open ear-drums or old suppurations, even if greased cotton or rubber stoppers be worn.

Again the eustachian tube must receive the blame for rekindling quiescent or mild infections. Obviously too, any treatment or procedure for nasal irrigation which might place the tympanic cavity lower than the level of fluid passing through the nasopharynx must be avoided. It will be remembered that nasal irrigations, save for the opened sphenoid, are now commonly advised with head bent forward, breathing through the mouth. Spasm of the soft palate is unlikely to force fluid up the tube, with the head held forward.

Determination of the type and virulence of the infection may often be made from the character of the exudate. If it be reddish or blood-stained, rather watery, appearing in great volume within a short time, there will usually be a disproportionate febrile reaction, and hemolytic organisms will be found. Complications may often be anticipated in the stormy course of this type of infection. The discharge is sufficiently acrid to precipitate an acute meatal eczema in many cases.

Mucoid or mucopurulent white or yellowish opaque discharges will disclose the variegated non-hemolytic flora of the nasopharynx and mouth. Delay in drainage may be caused by the tenacious consistency of the exudate. Unless the pneumococcus be found, such discharges offer a relatively benign prognosis.

In the more chronic types of middle-ear infection varying degrees of tissue destruction, with the presence of ingrowing meatal epithelium in small flakes or masses, lead to saprophytic and mold infection, with the production of odors ranging from mustiness to carrion stench. The latter is usually more or less indicative of bone denudation, either in the attic or along the ossicular chain. Such discharge is usually thin, whitish or brownish or flecked with grayish scales, and dries readily to adherent scabs or crusts. These heap up to a considerable size if undisturbed and may form accurate casts of the open tympanic cavity and adjacent meatal walls, lying upon a foul bed of moist grayish exudate.

The very copious pale-yellow discharge of diabetes seems to melt away the tympanic structures with little inflammatory reaction. Tuberculosis has a grayish, thin, rather scanty exudate through multiple perforations. Ichorous or serosanguineous discharge, from rapidly recurring areas of fungating granulations which tend to encroach upon the canal, would in mature persons suggest epithelioma. Ordinary granulation tissue or polypi recur but slowly, and are bathed in ordinary thick pus, whose drainage they impede.

After removal of external crusts which may be masking perforations of Shrapnell's membrane, the tympanic cavity is sometimes seen to be filled with a pearly white or gray, rather shining substance. Careful prying and softening, with a minimum of hydrogen peroxide and sometimes with dehydration by glycerine or alcohol drops, will disclose the laminated moist tissue-paper-like substance known as cholesteatoma. Such accumulations may be very extensive, and their removal will require several sittings. Much caution in syringing and curetting the site of a cholesteatomatous mass should be employed, because of pressure erosions through the tympanic walls which may cause fistulous involvement of the dura, facial nerve, or internal ear.

An intermittent discharge of clear mucus from the mouth of the eustachian tube is often observed in cases of old perforation where the tympanic

mucosa remains clean and uninfamed. Often due to faulty nasal hygiene, this tubal catarrh is directly related to similar nasopharyngeal moisture, needing treatment only when and if it becomes copious and thickens with the onset of an acute ethmoiditis or pharyngitis.

Occasional and rare contaminations of the tympanic region, which are in general agents coming from the external meatus rather than from within, include *Bacillus pyocyaneus*<sup>24</sup> (blue pus); *B. coli*<sup>20, 21</sup>; the gonococcus; actinomycosis (sulphur granules); blastomycosis; aspergillus, and other mold infestations<sup>33</sup>; and the bacillus of leprosy. Parasites of various kinds also invade the tympanic cavity and cause violent suppuration and very severe general symptoms. Grubs of the common house fly are perhaps the commonest of this disgusting type of middle-ear invasion. Killing of the parasite by chloroform, acetone, or alcohol, and instrumental removal followed by powerful bactericides, have been found wise in these cases. The rarer bacterial and fungoid suppurations require local bactericidal measures, while the associated systemic condition is attacked.<sup>18, 19</sup>

Aseptic cleansing measures include those precautions elsewhere noted respecting the toilet of the external auditory meatus. Proneness of the meatal skin to fissures, furuncles, and eczema should suggest caution in the use of caustic measures in the middle ear, or in long-continued moist treatment.

Considerable partisan argument has arisen at times regarding the merits of the so-called "dry" and "wet" methods of treatment of cleansing suppurating ears. In general, American opinion upholds the value of irrigations with normal saline or boric solutions, used at 100° to 115° F., in subacute cases (especially children) with copious discharge. The isotonic character of these solutions is helpful in avoiding maceration. To avoid softening, 1 : 600 formaldehyde irrigations have been suggested; but considerable burning is reported after use of this agent. Many clinicians prefer from 1 : 4000 to 1 : 10,000 bichloride of mercury, but its use is not general, nor is that of the stabilized chlorine solutions of the Carrel-Dakin type, probably because of increased likelihood of maceration of the meatal skin.

Washing out the ear may be done with a douche bag or irrigator 1 or 2 feet above the patient's head, using a blunt hard-rubber or medicine-dropper tip; or, a small all-rubber bulb syringe may be used. The stream of water must be directed along the meatal walls rather than directly at the drum; pain and vertigo will surely follow carelessness in this regard. From a pint to a quart of solution may be used at an irrigation, depending upon the amount and tenaciousness of the discharge. Such washings may be made half-hourly, and the interval diminished to twice daily, as the discharge diminishes and changes in character. To avoid maceration, the meatus externus should be dried carefully with several long, firmly rolled, narrow cylindrical cotton pledgets, after each irrigation.

Suction, as pointed out by Haskin,<sup>6, 7</sup> may either be applied directly by the attic cannula, or in connection with irrigation by the apparatus of Lucae or of Fowler. Care must, of course, be taken to see that Fowler's ingenious glass suction-bell is moistened at the rim so that suction will be effective, and also that the narrow rubber-tube irrigation cannula does not touch any inflamed area of the external auditory meatus.

If the external canal be straightened by pulling up and back in adults,

and down and back in young children, drying will be complete and painless. Otherwise, water will be left internal to the isthmus of the canal, and maceration will be probable, while drying of the tympanum will not be effected at all. Intelligent attendants may be instructed in the technic of preparation of such long cylindrical cotton swabs as are mentioned above, and advised by actual demonstration that such cleansing is both painless and free from danger to the drum-membrane. Their aid in carrying out such dry swabbing will be found to abbreviate considerably the duration of most aural suppurations.

The dry treatment, on the other hand, presupposes personal attention by the otologist or a highly trained professional assistant at such frequent intervals that for practical reasons it is rarely available in the average case. It is carried out by the insertion of wicks of the narrowest selvedged gauze packing, just long enough to reach the drum-membrane; these wicks are required to be changed as soon as they become saturated with the fluid from the middle ear. After the discharge has lessened so that the wick does not require to be changed oftener than once or twice a day, no method is easier or less troublesome. Preparation of solutions and sterilization of syringes and accessories are obviated, and swabbing and drying of the canal are minimized. For both dry and wet methods of cleansing a proper understanding of the preparation and safe use of cotton swabs is essential; and their frequent employment by the patient or his attendant will assist the clinician greatly by diminishing the amount of pus which requires removal at the regular times for treatment. Gauze wicks should never be inserted until the meatus is entirely free of exudate, dead skin or pus, and should the secretion upon them adhere to the canal wall, it must be softened with hydrogen peroxide or normal saline solution to obviate possible excoriation of the meatal skin.

As elsewhere suggested irritation of the skin of the canal requires drying, and touching with 10 to 20 per cent. silver nitrate solution on fissured areas, followed by a mineral-oil base zinc oxide ointment, or an ointment of lanolin containing boric acid.

Antiseptic treatment of aural discharges varies considerably with the type of suppuration and the chronicity of its course.

Dehydrating agents have held high place for years in intratympanic therapy. Alcohol, 70 to 95 per cent., with or without the addition of finely powdered boric acid, is an agent of venerable repute. Somewhat painful, its use as an astringent and penetrating antiseptic is frequent where a fairly large perforation exists, especially when the aural mucosa is covered with indolent granulations. If boric powder be used, care must be exercised to see that no crust accumulates to obstruct the perforation. Salol and dithymoldiiodide (aristol) have also been used with alcohol or by insufflation. Iodoform powder, because of its odor, is rarely used at present except in surgical packings, or in a paste with bismuth subnitrate and petrolatum.<sup>35</sup>

Ether, which boils at 95° F., is almost entirely anhydrous, and therefore dries the tympanic walls more rapidly and thoroughly than either alcohol or its occasional substitute acetone. Intratympanic aqueous syringing should be done but once, and after thorough drying the ether should be dropped directly into the ear.<sup>36</sup>

The more powerful antiseptics should only be administered by the

otologist, and this is especially true of the caustics (chromic acid; trichloroacetic acid; silver nitrate), whose main use is the destruction of the bases of inflammatory granulation tissue and polypi. Applied on cotton applicators of minute size in saturated solution, or fused upon the tip of a tiny silver probe, these agents act primarily by dehydration and destruction of vascular supply, and must be used only in very circumscribed areas. Danger to the facial nerve or labyrinth must not be ignored.

The action of all of the milder antiseptics is prolonged by the use of glycerine. Phenol, in  $\frac{1}{2}$  to 1 per cent. solution, though a mild escharotic, is found to alleviate pain if glycerine be the solvent; but its continued use provokes maceration of the canal walls. Phenol with tincture of iodine  $\bar{a}\bar{a}$  500 mgm., to 30 c.c. of alcohol is an admirable desiccant antiseptic (Beck).

Antiseptics based upon the organic dyes have found much employment during recent years because of their non-corrosive, non-coagulant penetration into the outer layers of the tympanic mucosa. In this group, mercurochrome,  $\frac{1}{2}$  to 5 per cent., is a powerful antagonist to the streptococcic infections.<sup>45, 46, 47</sup> Its solutions must be not more than three or four days old. A less irritant and practically colorless member of the same group, meroxyl, may be used by irrigation, 1 : 500. As in the nose, mercurochrome is occasionally very irritating and causes burning and swelling of the canal. Its high color may readily be removed by slightly acidulated alcohol. The scarlet dye in the throat is an accurate index of the permeability of the eustachian tube. Metaphen, 1 : 5000 solution, is a still more recent, non-irritating mercurial of high germicidal potency, without staining disadvantages.<sup>47</sup>

Mercuric chlorid, 1 : 2000 to 1 : 10,000, is often used in aqueous solution for irrigation, though it is a coagulant of albumin. Occasionally, bichlorid-alcohol, 1 : 4000, is used by swabbing.

Acridflavin neutral, 1 : 2000 to 1 : 5000, though slightly more irritating than mercurochrome, is equally effective.<sup>40</sup>

Gentian violet (pyoktanin),  $\frac{1}{4}$  to 1 per cent., is highly destructive to staphylococci; but its violent purple color is a bar to its use save in the hands of the otologist for intratympanic application.<sup>38</sup> Similar comments may be made upon methylene blue and crystal green in the same strengths, and upon basic fuchsin, 1 : 30,000 in alkaline aqueous solution.<sup>37</sup>

Specific indications obtain for certain infections, glacial acetic acid on the indolent pale spongy tissues of the *Bacillus pyocyaneus*<sup>24</sup>; an emulsion of salvarsan in glycerine in the rare case of infection with the symbiosis of Vincent (*B. fusiformis* with Vincent's spirillum); 5 per cent. solution or even the fused chromic acid bead may be very helpful in clearing the deep ulceration of such cases.<sup>29, 30, 31</sup> Lactic acid, 20 per cent., or formaldehyde, 1 per cent., are useful aids in staying local tuberculous manifestations while systemic treatment is under way.<sup>28</sup> Mercurial antiseptics are obviously indicated when suppuration complicates syphilitic involvement of the eustachian tube and tympanum. Pneumococcic infections yield quickly to optochin (ethylhydrocuprein), 1 per cent.; but this quinine derivative is so toxic to the auditory and optic nerves that it must be used with the greatest caution by local application, never by instillation nor for long periods of time.

Silver salts, notably the nitrate  $\frac{1}{2}$  per cent., are instilled as mildly

astringent antiseptics. Lacking the coagulant action of the nitrate, hence less active and less likely to cause preliminary irritative increase of the tympanic exudate, are the 5 to 25 per cent. solutions of various silver albuminates—argyrol, protargol, silvol, neosilvol, etc.<sup>39</sup>

Double exposure of the suppurating ear to infection from the open drum and from the swollen eustachian tube may often change its bacterial flora in a few hours from one specific invading agent, adding multi-form saprophytes, molds, and even yeasts.<sup>18, 19, 23, 25</sup> Such agents add fermentative and digestive changes, with the production of bad odors. Delicate syringing of the drum-cavity with Carrel-Dakin solution, or the instillation of 1 : 1000 formaldehyde solution, or the insertion into the attic of a little chlorazene paste or dichloramine, 2 per cent., in chlorcosane oil, will speedily destroy such odors. Of course if death of bony tissue is occurring, with caries of an ossicle or sequestration of portions of the tympanic wall, the odor will promptly recur and other measures must be undertaken. The chlorine antiseptics require care in covering the meatal skin with zinc oxide ointment, to obviate possible irritation.<sup>43</sup>

Animal and insect parasites, larvæ, or maggots, occasionally find an excellent breeding place in the pus of a neglected ear-drum. After cleansing as far as possible, instillation of chloroform will usually kill the invaders, after which the violent suppuration must receive careful treatment.

Introduction of chemical antiseptics in the nascent form by electrical "ionization" has recently gained some enthusiastic advocates. The aural cavity is first cleansed with peroxide and alcohol, then filled with a zinc sulphate solution, 1 per cent. A zinc terminal connected to the positive pole is so placed in the external meatus that it cannot come into contact with the skin or drum-membrane; the negative pole is on the arm, and a current of  $\frac{1}{2}$  ma. is used to begin with, working up gradually to 3 ma. for ten- or fifteen-minute periods every other day. Iodine, ionized from Lugol's solution, with the negative pole in the ear, has also been used. Marked increase in discharge occurs for the first two or three treatments, and a grayish exfoliation of meatal and tympanic epithelium occurs. Pain and vertigo are marked as the current strength increases, and will determine the length and interval of treatments. The procedure is of no value unless the perforation in the drum is big enough to let the electrolytic fluid into all parts of the suppurating area.<sup>41, 42, 43, 44</sup>

Removal of local causes of reinfection includes a wide range of procedures: Enlargement or modification of tympanic perforations; removal of granulations or polypi; curettage of carious areas; ossiculectomy; and the various radical and semiradical methods of surgical drainage of the mastoid antrum and other cellular structures in the temporal bone. Hygiene and removal of septic foci in the nose, throat, accessory sinuses, and teeth are elsewhere considered. All such factors must be considered in estimation of the prognosis of an aural suppuration. Prophylactic removal of adenoids and tonsils in children is advocated by some, even during the progress of an acute otitis media.

General conditions tending to cause reinfection include tuberculosis, diabetes, and syphilis, and also the chronic metabolic disturbance brought about by a diet lacking in the water- and fat-soluble vitamins,<sup>15, 17</sup> or by a diet poor in iodine and lime. No organ so complex as the ear should ever be considered save in the light of its manifold relations, vascular, neural,

and orificial, with the entire body. Sepsis in the teeth, the tonsils, the accessory sinuses, the abdominal organs, or the genito-urinary tract, may lessen resistance elsewhere, and, as in the ear, permit invasion by germs against which the individual is normally safe. Soluble toxins carried in the blood-stream, without the occurrence of actual bacteremia, may be responsible for some of this lost protective reaction in the tympanic mucosa. Variable results from use of vaccines in aural suppurations may be traced perhaps to the influence of remote foci. Vaccines are most effective if autogenous, freshly made, and started in small doses. Reactions must be watched and avoided by making the succeeding injection without any increase in amount. Doses should rarely exceed 0.5 c.c., and are best given twice weekly. They are of greatest value when made from the hemolytic cocci; other forms give lesser duration and degree of immunization. Changed bacterial flora will indicate renewed culture and revaccination at least every six weeks while this method is under trial.<sup>32, 33, 34, 34a</sup> Stock vaccines are probably of most value from the alien-protein protective reaction which they provoke. Diphtheria antitoxin is, of course, indicated for invasion by the Klebs-Löffler bacillus.<sup>22</sup> As an aid to the treatment of tuberculosis of the ear, tuberculin may be used, O. T. (old tuberculin) preferably, but always in minimal doses and with strictest regard to the patient's pulmonary and laryngeal condition. Such measures may well be managed by the colleague in charge of the patient's general condition, since overdosage has not infrequently rekindled healed lesions elsewhere.<sup>26, 27, 28</sup>

Lessening saprophytic involvement by instillation of active cultures of *Bacillus bulgaricus*, or by use of dried mixtures of intestinal and glandular digestants, has been tried without startling results.

Diet, in children especially, should include plenty of antiscorbutic materials—citrus-fruit juices, carrots, fresh vegetables of the leafy sorts. Sea foods, notably shell-fish, are of value. Cod-liver oil has an exceptional vitamin content and may be freely given, either unrefined or emulsified with malt extract.<sup>15, 17</sup> Thyroid and parathyroid deficiencies, with corresponding loss of calcium content in the blood, may be considered as factors in lowered resistance to the chronic infections.<sup>16</sup> Guarded administration of thyroid alone, or of parathyroid with or without calcium, may, therefore, be tried in sluggish cases.

Cessation of discharge takes place under medical treatment of aural suppurations in 50 or 60 per cent. of cases; the rest require local or neighboring surgical relief. Unless an emergency exists, even cases with a copious discharge from remote parts of the tympanum should not be condemned to undergo radical surgery until medical measures have received a fair trial for several weeks.<sup>3, 4, 7, 10, 12</sup>

The patient and his attendants must receive careful instruction in the technic of cleansing the ear.<sup>10</sup> The otologist must see his case sufficiently often to make sure that his orders are regularly and completely carried out, or modified to fit changes in the situation. Nowhere is this more essential than in the care of children. Firm control by an assistant, extreme caution in avoidance of painful manipulations, a soothing voice, will gain confidence and co-operation from the young patient. Nothing but dissatisfaction can arise from forcible and often brutal efforts at inspection and treatment, and parents are often alienated by the otologist whose activities are thus ill-advised. If the necessity for firm control is explained

to the parent, valuable help is at once enlisted, especially in managing the pampered or invalid youngster.

Restoration of hearing after chronic suppuration depends upon the amount and extent of tissue loss, and the resultant scarring about the ossicular chain and tympanic membrane. Therefore, if a discharge can be cured with a minimal amount of operative interference and of trauma during medical treatment, the prognosis for hearing will remain good. Irrigation and swabbing may be cut short by the use of electrically-heated hot-air douches, though care must be exercised to avoid vertigo from excessive heat. Diathermy from moderate use of powerful electric lamps is also employed to assist in terminating the discharge and restoring normal circulation.

But resolution of a suppuration may drag for months and years unless treatment is carried on. Hearing suffers, therefore, so far as its transmission across the tympanum is concerned, not only from local obstruction by retained secretions, but from the gradual substitution of fibrous connective-tissue for the normal mucous and ligamentous structures. This interchange may take years, but it remains one of the most frequent causes of incurable deafness. Intratympanic operations for the relief of this condition are subject to the defects of operations upon scar-tissue elsewhere; increased inflammatory reaction is followed by scarring often more extensive than at first.<sup>2</sup>

Reinfections from outside sources, as from water in swimming by way of the external ear or the eustachian tube, or from faulty blowing of the nose, improper nasal irrigation, or from intercurrent rhinopharyngeal infections, all add to the burden of scar-tissue which binds the tympanic membrane and ossicles and dulls their vibratory function.

Similar scars of inflammatory origin, webbed across the labyrinthine apertures and the promontory, may immobilize the stapes and impede endolymphatic impulses between the oval and round windows.

Moistening and softening of such scar-tissue bands by mucopus often seems to favor temporary improvement of hearing; hence the reported benefit to some patients when the ear "starts to run." Dry crusts are dislodged and some motion is resumed. Dull hearing in the "dry" ear may also be due to pads of epithelial scales, closely applied in the tympanomeatal angle.

Ears which after cleansing seem to reap benefit from moisture of the tympanic structures may be helped by glycerine drops, or by the various types of oiled-cotton or membranous artificial ear-drums, as noted elsewhere, but, unless such prostheses are frequently changed under aseptic precautions, renewed suppuration will be provoked.

Where aural discharge lasts but a few weeks, diminishing gradually to a thin sticky mucus, hearing will probably be unaffected whether permanent perforation remains or not. Frequently the entire membrana vibrans is lost with virtually no impairment of hearing. Recurrent discharges, which become copious and thick upon short notice, and disappear after a moderately uncomfortable course only to flare up with the next severe cold, may wreak their will upon the tympanic structures for years, with a loss of hearing so gradual that it passes unnoted by its victim. Such a condition should call for the sternest measures of cure and of prophylaxis against renewed infection.<sup>2, 10</sup>

## INDICATIONS FOR SURGERY OF THE MIDDLE EAR

Indications for surgery of the middle ear may be considered in two groups: The first includes cases wherein improvement of hearing, or relief from tinnitus, may be effected by operative means; the second comprises those cases in which inflamed or diseased tympanic structures require surgical drainage, removal, or obliteration.

No intratympanic procedure for the improvement of hearing should be undertaken without careful and repeated testing of the auditory range. Eighth-nerve involvements, as commonly seen after basal fractures, particularly the recent crushing and hammering fractures from motor accidents, must be excluded from the possibility of aid from surgery of the conduction apparatus.

The upper tone limit may be raised through the contraction or shortening of the tensor tympani muscle, or when increased tension of the mobile ossicular chain is produced by adhesions, and also when the tympanic membrane is in direct contact with the mobile incus or the stapes.

Lowering of the upper tone limit occurs in solid fixation of the ossicular chain, accumulation of exudates, and acute or chronic swelling of the intratympanic soft parts, as well as in ankylosis of the stapes within the oval window.

Exploratory tympanotomy is occasionally done in an effort to discover by direct inspection the exact reason for ossicular immobility. It must be admitted that far less work of this delicate nature is being done at present than during the decade from 1895 to 1905. Certain clinicians have failed to cultivate the refined technic necessary to this field, while others consider the division of scars or thickened ligamentous bands to be often a futile expedient. Nevertheless, if the sectioned surfaces can be drawn far apart by resetting the ossicular chain in relations approaching the normal, and if an increased number of adhesions can be prevented from re-forming at the point of operation, the orthopedic problem is solved. There is no possible method of applying fixation-apparatus to retain the parts in a position of exaggerated correction while they heal; there is a constant tendency to recurrence of the faulty alignment. Only rarely can membranous substances be interposed between sectioned tympanic adhesions<sup>8</sup>; and as with septal turbinate synechia, recurrence is prompt unless a considerable distance separates the underlying structures.

Advantage may be taken of the elasticity and essential balance of the ossicular chain as it swings between the drum-membrane and the oval window, to draw apart adhesions about the incus and stapes. Regard must be paid to the possibility of distortions due to adhesions of the inner surface of the drum to the inner tympanic wall, and to the existence of thickened bands of fibrous tissue in the drum-membrane drawing the malleus forward and upward, and especially to increased thickness and tension in the transverse mucous folds about the heads of the malleus and incus. This latter transverse diaphragm-like structure tends to hoist the major ossicles up into the attic, and interferes also with the normal drainage of the epitympanic mucosa.

When the drum-membrane is largely gone, the tensor tympani remains unopposed. Contractures of the unopposed tensor interfere greatly with the normal hearing range. The malleus is pulled inward, and practical fixation may take place.<sup>13</sup>

By far the most important indications, however, for intratympanic surgery, arise from the necessity for improving drainage. Given a persistent discharge from remote regions of the drum, the succession of pathologic changes makes betterment in drainage almost prerequisite to a cure.

First among surgical hindrances to drainage comes the relative location and size of the perforation. Low and large perforations of the membrana tensa are less likely to call for surgical enlargement than are small perforations of Shrapnell's membrane, cluttered up by mucosal folds, by the major ossicles and their ligaments, and by epithelial ingrowths (Fig. 308, B).

Next among the surgical obstructions are the inflammatory swellings—granulation tissue at the rim of a perforation, or bulging toward the drum opening from the promontory, the tympanic floor or its roof, or from a carious ossicle. Such sessile granulations, swimming in exudate, elongate readily into myxomatous or fibromyxomatous polypi, which may not only fill up the perforation and thrust aside the remaining tympanic structures, but may even protrude as far as the external opening of the auditory canal (Fig. 295, C). Removal of such new-growths is imperative before diagnosis of intratympanic damage can be determined; but it is equally important that their origin be accurately mapped out. Shrinking with adrenaline and removal of surrounding exudates will usually permit a decision. Occasionally polyps will be found to arise from a fistula of the posterosuperior meatal wall, pointing to mastoid involvement (Fig. 295, B). Careless traction, or blind and forcible probing, may cause extension of the purulent process to the labyrinth, the meninges, or the jugular bulb.

Obstruction to drainage, caused by changes in previously normal intratympanic structures, must next be considered. Swelling and stiffening of ligaments and mucous folds, followed by ulceration, and later by perioritis of the ossicles, will dam up fluids within the attic and its communicating pouches. Removal of the malleus, or of both malleus and incus, is often indicated in such cases. It should be recalled that long-continued carious processes will go far toward destroying these ossicles, and that the normal processes and articular surfaces of the little bones will not be found; a mere shred or knob of roughened irregular aspect, easily lost and out of line with its usual landmarks, is all that the observer will locate.

Drainage is perfect after such removal, making postoperative annoyance rare after ossiculectomy. A widely opened cavity has been substituted for one half-full of swollen structures whose normal physiological function is no longer exercised.

Stapedectomy, total or partial, is unnecessary for the promotion of drainage. Its only indication has been for the improvement of hearing. Many observers question the value of substitution of a fibrous membrane in the oval window at the cost of danger from labyrinthine infection, from trauma to the facial nerve just above, or from fracture of the crura of the stapes, with resultant bony ankylosis of the foot-plate.

Resection of the outer wall of the attic is specially indicated when caries of this region has begun, and is often a necessary complement to ossiculectomy. Where cholesteatoma has invaded the epitympanum through high perforation, complete cure is rare unless this or a radical mastoid be done.<sup>11</sup> Removal of the attic wall is rarely sufficient to destroy

a cholesteatomatous or carious process which has extended back into the aditus or antrum. Copious welling out of pus with the pulse-beat, after preliminary care in cleansing, will discourage the prognosis of cure from intratympanic surgery alone. Indeed, no patient suffering from chronic aural suppuration should ever be told that intratympanic operations will cure him. These procedures should be recommended rather as conservative of hearing, as avoiding long hospitalization and after-treatment. They do not in any sense replace the radical tympanomastoid exenteration for destruction of all purulent foci.

To keep out pharyngeal infections, rasping out the eustachian tube-mouth, and covering it in with Thiersch grafts or flaps from the neighboring remnant of the tympanic membrane, have been recommended. Also the ingenious procedure of threading in and burying a strand of knotted catgut at the isthmus of the tube (Pierce) may be tried. Such procedures are more properly a part of the radical operation, where room is established for carrying out all steps. More important are the prophylactic nasopharyngeal operations elsewhere discussed.

Fissuring, furunculosis, eczema, or other irritative conditions of the meatus should delay intratympanic procedures of moment, such as ossiculectomy. Removal and cauterization of granulations and polypi may, however, be a necessary preliminary to cure of the otitis externa.

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## INDICATIONS FOR SURGERY OF THE MASTOID PROCESS

## INDICATIONS FOR THE SIMPLE MASTOID OPERATION

**In Infancy.**—The mastoid process in infancy is quite rudimentary, the cortex thin, and the antrum correspondingly superficial. The periosteum is not so firmly adherent in the superior part, which explains the tendency for pus passing through a perforated cortex to follow the line of least resistance and accumulate in the temporal region. Under such circumstances the mastoid process *per se* may not present the classical symptoms—redness, tenderness, and swelling—and the condition is frequently overlooked. The stylomastoid foramen carrying the facial nerve lies almost on the surface in infants and it is here, just as the nerve emerges from the foramen, that facial palsy is likely to occur, either through mastoid disease or as the result of injury at operation.

The mastoid process approaches the adult type in size and conformation only at the beginning of the fourth year.

**Types of Mastoid Process.**—In considering indications for operation on the mastoid process, it is necessary to recognize three and sometimes four distinct types of processes. The pneumatic type, in which the cortex is relatively thin, favors rather early perforation, especially in children. Danger of intracranial complications in this type, particularly if the direction of the inflammation is external, is reduced to a minimum. It is found most frequently in acute pathology, without exacerbations.

In the diploic type of mastoid, which is characterized by multiple small cells, the cortex is usually thicker and, therefore, does not as readily present localizing symptoms of mastoid disease. This is frequently observed in patients suffering repeated attacks of otitis media.

The cortex in the compact type is probably always the result of sclerotic changes and often becomes very thick and dense. In some instances the entire mastoid cavity except the antrum is completely filled with hard, ivory-like bone. It can be readily understood that localizing symptoms of mastoid disease could not possibly present themselves under such circumstances. Here the inflammation will follow the line of least resistance and may invade the interior of the skull. This condition is found almost exclusively in the presence of chronic otorrhea and mastoiditis. It is reasonable to assume that the dense type of mastoid cortex is pathologic, the result of tympanic inflammation in infancy, which interferes with the usual absorption of the diploë and subsequent pneumatization.

It is not unusual to find a mixed type which represents a combination of the diploic and sclerotic. A few pneumatic cells may also be present.

**Method of Mastoid Involvement.**—With the exception of a very limited number of cases of mastoiditis that may occur as a complication of some general disease or trauma, acute mastoiditis is always secondary to an acute tympanic disease, and it is equally true that chronic mastoiditis results from neglect of the acute form.

The mastoid antrum is always involved to some extent in practically all acute suppurative inflammations of the middle ear. The early tenderness on pressure over the mastoid process is in most cases an external reflection of the involvement not of the osseous structure, but of the mucosal lining, which quickly subsides and never calls for immediate operation unless symptoms of some complication become manifest.

As the mastoid cells sometimes extend beyond the upper wall of the meatus and involve the zygoma, they are subject to pathologic changes and produce localizing symptoms separate and apart from the major cells. When the cells extend backward and communicate with those of the occipital bone, the disease is apt to involve this locality and produce characteristic symptoms.

An abscess may form posterior to the mastoid process if the infection penetrates the temporo-occipital suture, or the canal carrying the mastoid emissary vein. Below the zygomatic ridge and just back of the external meatus there are small foramina through which minute vessels pass and finally open into the superior petrosal sinus. Meningitis may develop by infection passing along this route. This is the favorite point for a fistulous opening in acute mastoiditis, especially in children, and calls for operation.

When the posterior zygomatic route becomes involved an abscess develops beneath the temporal fascia and is known as zygomatic mastoiditis, which sometimes becomes very extensive and painful. The swelling and discomfort are anterior to the auricle and above the temporomandibular joint and may extend over a considerable part of the front of the head, even exposing the dura covering the temporal lobe. In a limited number of cases the membrana tympani presents few, if any, signs of inflammatory changes and may not have been even perforated. Such patients state that they suffered from earache in the beginning, but have not at any time noticed a discharge. Some pain and difficulty are experienced in mastication, and there is slight edema of the eyelids. I believe that the source of involvement here is, as usual, the nasopharynx, the infection extending to the tympanic cavity and attic by way of the tube. The attic becomes walled off, the inflammation extending outward and reaching its destination via the cancellous bone, with but little involvement of the antrum. As stated, this picture is not one of the common types, but I have observed it not infrequently. This particular type of mastoid disease occurs when the zygomatic cells are large, and is found most frequently in young children. By the inexperienced eye its significance is often overlooked, as the condition is not obviously connected with the mastoid process, which may be little, if any, swollen.



Fig. 298.—Zygomatic mastoiditis, with absence of tenderness and swelling over the mastoid process, causing doubt as to presence of mastoiditis interna.

**Bezold's Mastoiditis.**—In the pneumatic mastoid an acute infection will readily find its way to the various cells, causing early perforation of the cortex, or may penetrate the inner aspect of the tip into the digastric fossa, producing what is known as Bezold's variety of mastoiditis.

Bezold's mastoiditis is favored where there are large pneumatic spaces

at the apex and where the cortex is dense and fairly thick. It is rarely found in very young children. In this condition there may be an entire absence of pain on pressure over the process and a cessation of other symp-



Fig. 299.



Fig. 300.

Figs. 299, 300.—Anterior and posterior views of zygomatic mastoiditis in child six years old. Acute otitis media with mastoid involvement extending over two weeks. Spontaneous rupture followed by myringotomy one week later. Swelling and fluctuation over mastoid process disappeared when pus found lodgment above and in front of auricle, giving the impression mastoid was not involved.

toms temporarily, because the pus has found new lodgment. The neck below the mastoid becomes swollen and quite tender on pressure, due to the accumulation deeply situated underneath the muscles, which precludes a diagnosis by fluctuation until the abscess has attained a considerable



Fig. 301.—Unusual illustration of Bezold's variety of mastoiditis in a child eighteen months old, complicating acute otitis media.

size. On account of the edema the finger cannot be placed underneath the apex of the mastoid, as is the case in the normal process. This is a valuable diagnostic point.

Bezold's mastoiditis may be confused with an involvement of the glands of the posterior cervical triangle insofar as swelling and tenderness are concerned. In the latter case no pus formation actually exists unless the glands break down, but there is a rise in temperature and considerable pain on moving the head. This type of Bezold's mastoiditis may develop rather quickly and almost invariably calls for mastoidectomy.

A thick cortex, especially if the cells are of the diploic type, provides an effective barrier for resistance to external carious erosion. The direction of the inflammation, therefore, is internal, favoring the formation of a cerebral abscess or meningitis by extension through the tegmen antri or tegmen tympani; or it may extend backward, involving the lateral sinus or the base, producing a sinus thrombosis or involvement of the cerebellum.

The most frequent complication of an acute tympanic abscess is chronic suppurative otitis media and consequent chronic mastoiditis, both of which are too often the result of neglect or inefficient treatment, and therefore preventable.

When there is pus in the middle-ear cavity it is generally present in the mastoid antrum, hence it is reasonable to assume that suppurative tympanitis and antritis are usually coexistent. The infection readily finds its way to the various mastoid cells and sets up a mastoiditis of a mild or virulent type, depending on the character of the micro-organism, the destruction being especially rapid and extensive when complicating influenza, scarlet fever, measles or diphtheria, as here bone necrosis and serious inflammatory penetrations are likely to develop *very early*.

**Primary mastoiditis** does occur, though rarely, as a result of trauma. It may develop also, complicating

a low grade of disease, by way of the blood-stream. Otherwise I believe the so-called primary mastoiditis develops in the usual way, originating in the nasopharynx and extending up through the tube to the tympanic cavity and then to the mastoid antrum and cells. There are unquestionably cases in which the middle-ear involvement is so mild and transitory, on account of efficient drainage through the eustachian tube, that on examination the membrana tympani and middle ear do not present any pathology, but the mastoid involvement which occurred simultaneously may slowly continue and finally present symptoms of mastoiditis, even the classical symptoms, as shown in the illustration (Fig. 303). Mastoiditis of this type is necessarily surgical.

**Appearance of the Membrana Tympani.**—There are two distinct observations on the appearance of the membrana tympani which must be kept in mind in arriving at a diagnosis of surgical mastoiditis. The first



Fig. 302.—More ordinary type of Bezold's mastoiditis, in a child six years old, which does not so clearly show the collection of pus below the tip. Clinical symptoms wholly disappeared with the perforation of the tip.

is when the membrane is reddened and swollen, with obliteration of the landmarks and a nipple-shaped perforation; if, in addition, there is drooping of the adjacent posterosuperior osseous wall and a persistent rise in temperature, even though slight, surgical intervention is necessary. The second is where the inflammatory changes in the membrana tympani are indicating resolution, as evidenced by a return of the landmarks to view, with little or no discharge present. Usually there is no involvement of the posterosuperior osseous wall, the disease being confined to the tip. Tenderness on pressure over the process, particularly the tip, will continue as a sequela of the acute tympanic disease, notwithstanding the subsidence of the latter. Operation is indicated here also.

**Myringotomy vs. Spontaneous Rupture.**—If the drainage is free and the

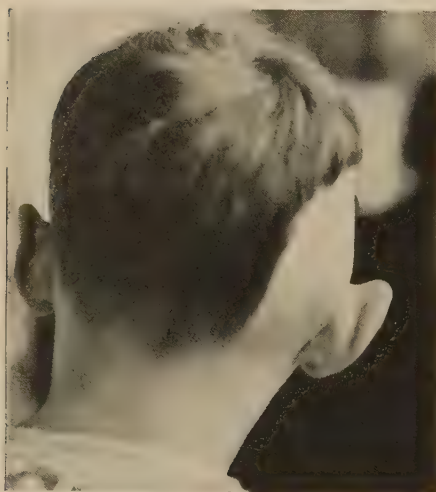


Fig. 303.—So-called primary mastoiditis. Patient aged seven years. Acute suppurative otitis media, left ear. No pathology observable in external canal nor membrana tympani of right ear. Carious erosion through cortex, producing fluctuating mass. Cells broken down and filled with pus.

infesting organism only mildly virulent, a subsidence and subsequent cure are likely to occur. If a virulent infection involves the osseous structure the tympanic and mastoid suppuration will become chronic unless relieved by operation. The severity of each type is greatly lessened and complications may be averted by *prompt* myringotomy.

A spontaneous rupture unquestionably favors surgical mastoiditis and should never be permitted if the case is seen in time to prevent it by myringotomy. It is much better to err on the side of a too early myringotomy than to wait for the complications that may arise following a spontaneous rupture.

When a repeated myringotomy becomes necessary the mastoid is the chief etiologic factor and usually will require surgical treatment. If, in the presence of an ample perforation,

the discharge continues abundant and fever persists, together with intermittent or continuous headache, and especially if there is persistent, though slight, mastoid pain, surgical intervention is required.

**Bacteriology.**—Various micro-organisms are found in the discharge from the tympanic cavity, the most virulent and destructive being the diplococcus and the various strains of the streptococcus. While the staphylococcus is not so virulent, it frequently is very difficult to abolish and is one of the chief factors in the continuance of the chronic state. Although the *Bacillus mucosus capsulatus* is not acutely virulent, it is the most insidious and one of the most destructive organisms and is the cause of some of the really formidable complications, these developing out of all proportion to the accompanying symptoms, which are frequently insignificant until the major complication becomes suddenly manifest.

Bone necrosis is invariably present and mastoidectomy is indicated when the insidious *Streptococcus mucosus capsulatus* (*Pneumococcus* Type

III) predominates in the discharge, accompanied by the following picture: Absence of pain and tenderness, normal pulse and temperature, apparent beginning resolution, *but a discharge persisting for a period of from four to six weeks*. If in the third week or even the second the pus is fairly copious and of a peculiar creamy character, the chief bacteriological factor is the *Streptococcus capsulatus* and operation is indicated, especially if the Roentgen-ray findings are positive.

Should the predominance of the *Streptococcus hemolyticus* or the *S. viridans* persist, mastoidectomy will usually be required.

**Blood Examination.**—When in doubt blood examinations are often of great value, but not uniformly so. In some cases of extensive destruction there is little change from the normal, which in all probability is due to the establishment of free drainage and little absorption of the toxins.

An increased leukocytosis shows the patient's resistance to the infection, whereas an increase in the polymorphonuclear percentage indicates its severity. Even though the polymorphonuclear percentage may be high, if the increased leukocytosis is correspondingly raised, the latter will largely balance the former. It will be plain, therefore, that an ascending leukocytosis and a descending polymorphonuclear percentage are usually a favorable indication, whereas the reverse is definitely unfavorable. In this latter condition mastoidectomy should be seriously considered, and is called for when supported by other indicative symptoms.

**Temperature.**—A rise in temperature, although usually persistent, is not high in uncomplicated cases of mastoiditis, ranging from 99° to 104° F. The higher temperature is common in children, but should be looked upon as possibly pointing to further complications in adults. The remissions between these extremes taken every two hours are fairly typical in the average case.

A sudden rise in temperature followed by a corresponding drop to about normal or below, accompanied by a chill and perhaps sweating, warrants suspicion of lateral sinus involvement and calls for mastoid surgery, as does also a steady high temperature without sudden remissions, which is suggestive of meningitis, septicemia, or pyemia.

On the other hand the temperature is frequently atypical and in no sense characteristic.

Careful elimination of all other causes of temperature, especially pyelitis in children and central pneumonia, must be sought before resort is had to aural surgery.

**Mastoid Tenderness.**—It rather frequently happens that there is considerable or even extreme tenderness on pressure over the mastoid process. This is especially true when the infection is active and the drainage somewhat obstructed. Under all circumstances the two sides should be compared in eliciting mastoid tenderness. Pressure should be exerted inward and somewhat backward to avoid manipulation of the cartilaginous canal, which is painful in the presence of a circumscribed otitis externa and serves in some instances as a point of differential diagnosis.

Usually the initial tenderness disappears within a few days under proper treatment. Should it continue unchanged, however, or if the symptoms subside and then recur, operation is indicated in the presence of either scanty or profuse drainage.

Pain may be almost wholly absent in processes due to the *Streptococcus*

capsulatus and yet great bone destruction occur. A danger signal is the sudden appearance over the process of tenderness which might not have been present heretofore. Should mastoid pain first appear after the tympanic suppuration has existed for a week or more and then persist, operation is indicated. Usually an accelerated pulse, rise in temperature, irritability, and general malaise accompany this picture.

The development of pain on pressure over the emissary vein is suggestive of a threatened sinus involvement and may call for mastoidectomy.

Mastoid tenderness on pressure over the process is most marked at the points especially vulnerable to carious erosion, over the antrum close to the auricle, and at the tip. Pressure underneath the tip is painful in a healthy mastoid and should be avoided when searching for points of tenderness. Cases have been pronounced surgical mastoiditis with such tenderness as the predominating symptom, all of which recovered without operation.

Ambulatory patients who complain of only slight tenderness on *deep* pressure over the tip, accompanied by a progressive loss of hearing, frequently go to operation even in the temporary absence of other suggestive symptoms.



Fig. 304.—Cortical perforation in acute mastoiditis, with fluctuation and forward displacement of the auricle.

A continuation of tenderness on pressure over any part of the mastoid process, especially after a subsidence of the acute symptoms, is a strong indication of surgical mastoiditis and is, therefore, our most reliable symptom, notwithstanding the fact that an occasional surgical mastoiditis does develop without appreciable pain.

**Percussion dullness** over the process has been pronounced a valuable diagnostic indication of early mastoiditis.

Much stress is laid on this lessened resonance by several authors, but I personally am unable to place much value on it.

**Mastoid Edema.**—A collection of subperiosteal pus over the mastoid process, recognized by fluctuation and forward displacement of the auricle, is due to a cortical perforation and requires operation. On the other hand, tumefaction over the process in young children rather frequently occurs without cortical perforation, the mastoid exudate finding exit through the Rivinian notch by way of the posterosuperior osseous wall.

An equally large, non-fluctuating swelling over the mastoid or adjacent parts may be indicative only of a periostitis and should not be operated upon until non-surgical measures have failed.

It will thus be seen that mastoid periostitis *per se* is not necessarily serious or surgical, but periostitis of the posterosuperior osseous wall must always be regarded as a danger signal. This may occur during an acute suppurative tympanitis or an acute exacerbation of a chronic otorrhea. The osseous meatus becomes narrowed by the drooping of the posterosuperior osseous wall close to the tympanic ring, which is definitely tender on pressure and is, in turn, secondary to an empyema of the mastoid antrum. The presence of this symptom is one of the most reliable indications

of surgical mastoiditis, as is also foreshortening of the external canal, notwithstanding the fact that there may be an entire absence of all external signs of mastoid disease over the process.

Bone destruction is very rapid in mastoiditis complicating influenza, the exanthemata, and pneumonia. In order to prevent complications it is imperative in these cases to open the cells very early. Cortical perforation is likely to occur, producing the classical symptoms—redness, tenderness and swelling—which are always late signs and should never be awaited.

In some cases mastoid swelling may be greatly reduced or even disappear, giving the impression that resolution has begun. As a matter of fact the infective process is actually increasing, the subsidence of the swelling being due to freer drainage through the tympanic perforation or the eustachian tube, or the development of a zygomatic or Bezold's mastoiditis. Under such circumstances mastoidectomy is indicated.

It frequently happens, particularly in the chronic form of disease, that the cortex is quite thick and the inflammation, following the line of least resistance, progresses inward. From the foregoing it will be seen that drooping of the posterosuperior osseous wall occurs only when the antrum is sufficiently involved to implicate the posterior osseous wall which separates it from the external auditory canal. Operation is indicated in all such cases. This important and very definite symptom will not be present, however, when the disease chiefly involves the lower mastoid cells, particularly the tip, and yet this condition may require an operation just as urgently.



Fig. 305.—Furunculosis of the posterior wall of the external auditory canal, producing the clinical symptoms of mastoiditis—redness, tenderness, and swelling—over the process, relieved by evacuation of the furuncular abscess.

The inception of an otitis externa is usually without pain; there is little discomfort in pulling the auricle or applying pressure to the tragus. Furthermore, the cartilaginous canal is equally involved. This condition should not be confused with drooping of the posterosuperior osseous wall secondary to mastoid antrum disease, as above described.

When a fistulous opening appears through the posterior osseous wall it is definite evidence of considerable chronicity and calls for surgical intervention. Cases of this kind may occur when the infection is virulent, especially when complicating the exanthemata.

**Otitis externa** of the circumscribed form (furunculosis) involving the posterior wall frequently produces swelling, redness, and tenderness over the process, which is confusing to the inexperienced. A differential diagnosis should not be difficult. The tenderness and swelling extend only a short distance back of the auricle and are especially marked at the anterior

ridge of the process adjacent to the meatus. Also, the edema definitely pits on pressure. An examination of the external auditory canal will further serve to clarify the diagnosis. Audition will be but little interfered with by a furunculosis, whereas it is considerably impaired in mastoiditis secondary to tympanic disease.

Should an otitis externa and an otitis media be present at the same time, it may be difficult to determine which is responsible for the mastoid symptoms. When the abscess is superficial it would indicate that the otitis externa is the causative factor, whereas the middle-ear disease is responsible when the cortex is involved.

If suppuration over the mastoid process takes place and on evacuation the abscess is found to be superficial, neither the periosteum nor the bone should be disturbed if the former is normally adherent to the latter. On the other hand, if the collection of pus is subperiosteal and the cortex is discolored or shows a tendency to bleed, mastoiditis interna is present and calls for mastoidectomy.

**Discharge.**—When suppuration is profuse and continues unduly, even though there is an absence of all other symptoms, operation is indicated, if only on the ground that such a quantity of pus could not possibly come from the tympanic cavity and must, therefore, have its principal source in the mastoid process. A case of this kind may eventually subside, but is almost certain to assume the chronic form of otorrhea with all its attendant discomfort and danger. Such procrastination and neglect are unjustifiable.

Furthermore, even though the discharge does eventually cease, a considerable destruction of the membrana tympani usually will have taken place, which subjects the patient to future tympanic infection and its complications, as well as an impairment of hearing, though this may develop slowly. We are not justified in taking such chances even though an occasional case will completely recover in spite of neglect.

When a rise in temperature coincides with a definite decrease in the amount of discharge, surgical intervention is usually required. Likewise, the sudden cessation of a profuse tympanic discharge is often the initial symptom of surgical mastoiditis.

In some cases the perforation may be large and the tympanic discharge slight or even absent, and yet the mastoid disease steadily progresses on account of interrupted drainage through obstruction of the aditus. The classical symptoms of mastoiditis are apt to be present in cases of this type, and when complicating the exanthemata or influenza the destruction is very great, with extensive osteomyelitis.

Barring complications, an acute suppurative otitis media in children may exist from four to six weeks without causing anxiety, but an extension of this time before resorting to mastoidectomy is dangerous, not only in its bearing on possible intracranial complications, but on the future audition of the individual, even though ultimately there may be a cessation of suppuration and a regeneration of the membrana tympani. This statement is made notwithstanding the fact that I have seen cases go on for a longer period and completely recover. These are exceptional and the risk is too great and an expression of bad surgical judgment, for although a few cases may recover, as above stated, the average one will develop an endocranial complication or else interrupted resolution results in a chronic otorrhea. The great difficulty is to decide on the necessity of an operation after all

of the acute symptoms have subsided, inasmuch as the patient has been relieved of all symptoms except, perhaps, a slight impairment of hearing, mild subjective noises, and an otorrhea that is more or less abundant. The duration of the suppuration and the partial abatement of the symptoms, although important prognostic signs, are not wholly dependable, for even though the acute tympanic disease may have run its course almost without symptoms, the possibility of a sudden intracranial complication cannot be definitely excluded, which illustrates the danger of delay.

Recurrent acute suppurative otitis media in children, when the nasopharynx can be eliminated as an etiologic factor, is due to a focus of infection in the mastoid and should be relieved by operation if the use of suction and other non-operative measures fail.

Hemorrhagic mastoiditis is indicative of influenzal infection and generally requires operation.

In the presence of acute tympanic disease, either with or without discharge, though there may be an entire absence of all mastoid symptoms, if indications point to a beginning endocranial lesion which cannot be otherwise accounted for, mastoidectomy is required.

**Primary bone tuberculosis** of the mastoid process in children unquestionably exists and is, perhaps, more common than is generally realized. It is characterized by slight pain at first, which rapidly becomes severe, accompanied by early perforation of the cortex, followed by the classical symptoms of mastoiditis. As in periostitis of the mastoid the hearing is usually unaffected and there may be no clinical evidences of the disease as revealed by examination of the external auditory canal and membrana tympani. Mastoidectomy is always indicated in such cases, notwithstanding perforation of the cortex may not have taken place.

**Relative Urgency of Operation in Childhood and Adult Life.**—From an anatomical consideration it is quite evident that we are justified, generally speaking, in postponing operation in children and young persons to the limit of safety, but this is the important question: When does safety merge into danger? As stated, the cortex in a child is thin and the direction of the inflammation is usually external, causing carious erosion of the cortex and consequent escape of pus under pressure. It is true that the tegmen antri and tegmen tympani are also thin and there may even be a dehiscence in children. Still the fact remains that in the average case nature is considerate and nearly always favors the external route. On the other hand, at the age of forty-five and thereafter the cortex is much thicker, becomes denser from year to year, and accordingly resists inflammatory changes, so that the direction of the inflammation here, in following the line of least resistance, will show a greater tendency to involve the interior of the skull. Furthermore, it has been shown that the labyrinth does not always take on sclerotic changes but under some circumstances becomes actually softened and somewhat spongy, which also favors intracranial inflammatory penetration. This will show the relative danger of operative postponement in the adult as compared with the child.

Acute aural disease is relatively infrequent in advanced years, but as age increases the danger of labyrinthine involvement becomes proportionately greater. For this reason it would seem unwise to defer operation beyond reasonable limits in patients of middle age or older.

**Facial Palsy in Acute Aural Disease.**—Facial palsy is not often seen in

acute aural disease, but when present it is quite as urgent an indication for surgical intervention as when found in the chronic form. In either case it must be determined that the aural disease is the etiologic factor.

**Primary Jugular Bulb Thrombosis.**—Mastoidectomy is indicated as a preliminary step in operating for the relief of primary jugular bulb thrombosis.

**Roentgen Ray.**—A Roentgen-ray examination forms an important part of the routine study of suspected mastoid disease, and when considered in connection with the clinical findings constitutes a valuable diagnostic aid in our endeavor to differentiate surgical from non-surgical mastoiditis.

In order to be of real service, however, the plates must be as near perfection as can be attained to demonstrate the finest possible detail, which requires both extensive experience and skilful execution. This being accomplished, the highly technical part of roentgenology—the proper interpretation of the plates—follows. To my mind this can be done with the necessary accuracy only by those specially trained in this line of work, and herein lies the value or uselessness of the findings, depending on the skill and experience of the roentgenologist. It is my intention to state only a few important general principles and not in any way to enter into a discussion of the technic of roentgenology. It is true that the interpretation of the plates is not always borne out by operative findings, but this should not deter us from employing this valuable diagnostic aid, as the expert interpretation is mostly confirmed at operation.

Bone defects are commonly pointed out with great accuracy. It is even possible to demonstrate the presence of granulation tissue, free pus, or air in the cells. A mastoid which consists of very small cells in the region of the antrum does not provide for as free drainage as one in which this structure consists of larger cells, the small cells becoming more easily blocked than the larger type, thus interfering with drainage and more often requiring operative intervention. The position of the sinus can be uniformly indicated, which is always valuable to the operator, especially in chronic cases.

As the Roentgen ray freely penetrates sanguineous media to the extent of indicating translucency or even transparency in the absence of cortical thickening, the interpretation of plates in hemorrhagic mastoiditis is apt to be misleading, even in the hands of an expert, as an index of the extent of the pathology present. This has been demonstrated rather often when the clinical picture has demanded operation that was not clearly indicated or even may have been contraindicated by the Roentgen-ray findings. I am indebted to Dr. Frederick M. Law, of New York, for his views on this phase of roentgenology: "The hemorrhagic type of mastoiditis occurring in a structure in which some previous process has not caused an ostitic thickening of the trabeculæ will show a more transparent mastoid than one filled with pus, the trabeculæ directly under the cortex appearing sharply outlined, while those of the deeper cells appear softened or thinned out; in other words, the plate does not show the apparent involvement which the symptoms and clinical evidence indicate. At first glance there does not seem to be much difference between the normal and the diseased side, but careful study of the stereoscopic pair will usually show the change in the trabeculæ of the deeper cells. If a hemorrhagic mastoiditis occurs in a mastoid in which ostitic thickening of the walls has taken place as a result

of a previous process, the thinning of the walls will not be so apparent and an error in interpretation is liable to occur. A Roentgen-ray report should contain a description of the type of cell structure."

Both mastoids should always be examined and compared. If the disease is unilateral it can be readily observed; if bilateral, comparison will serve to point out the pathologic changes in each, which may later, through additional pictures, assist in determining whether one or both become operative. Repeated Roentgen-ray examinations are of service in determining whether resolution has set in and subsequent recovery is likely. They are of value, furthermore, in aiding one to advise for or against operation after the cessation of clinical symptoms, for by this means we may detect an incomplete resolution which otherwise would assume the character of a chronic otorrhea, with all its potentialities. In other words, roentgenology is indispensable as a diagnostic aid in all cases of atypical mastoiditis.

The presence of a perisinus abscess or an extradural abscess has been pointed out frequently by roentgenology when it would not have been suspected by the symptomatology alone.

As valuable as the Roentgen-ray findings are, however, they should not, as a rule, be accepted as an indication of surgical mastoiditis in the absence of confirmatory clinical symptoms.

In addition to formulated rules and regulations as indications for mastoidectomy, there is an indefinable factor possessed by the surgeon of wide experience called intuition. This enables him to weigh the pros and cons of the patient's general condition and advise for or against operative procedure, even though to one of less experience the contrary may seem to be indicated. This faculty is especially valuable in atypical mastoiditis, which has become so common in the past few years that it is almost the rule.

#### INDICATIONS FOR THE RADICAL MASTOID OPERATION

It is generally conceded that a chronic suppurative otitis media is dangerous and that the radical mastoid operation is indicated in all cases that do not yield to non-surgical treatment, this notwithstanding the fact that in some instances it has continued for many years without symptoms except deafness and the inconvenience of a discharge. I have known an otorrhea to be continuously or intermittently present for forty years, when suddenly an endocranial lesion became manifest which may have existed for some time. If these seemingly innocuous cases do not yield to non-operative measures, continued procrastination eventually becomes hazardous, for, after all, our only definite assurance that chronic otorrhea is not a menace lies in its complete eradication and, *per contra*, I have never known an intracranial complication to develop following a radical operation that was not suspected or actually existent before operation.

Many rules and indications for performing the radical mastoid operation have been advanced from time to time, but they are not conclusive insofar as meeting the exigencies of every case, so the responsibility of final judgment as to the necessity of operating on a given patient must be assumed by the operator. More discrimination is used in the selection of cases than formerly and in consequence the radical operation is not performed so frequently.

**Objects Sought in Operation.**—There are two principal reasons why the radical mastoid operation should be done: First, for the relief of an annoy-

ing and at times offensive otorrhea; and second, the prevention of intracranial complications, which are always serious. This does not mean that the radical operation should be resorted to in all cases of chronic suppurative otitis media which do not wholly recover under conservative methods of treatment. Every case of chronic otorrhea does not present the same degree of potentially serious complications and each must be dealt with on its own merits. When the suppuration continues as the result of carious erosion or a necrotic process, intracranial complications are threatened, but on the other hand, if the disease is confined to the mucosal lining of the tympanum it is quite unlikely that intracranial complications will arise, thus contraindicating a radical operation. Furthermore, when the pathology is *apparently* limited to the tympanic cavity, but is not relieved by non-operative treatment or ossiculectomy, the radical mastoid operation is indicated because the mastoid area under such circumstances must be involved even in the absence of symptoms.

In cases where the membrana tympani is destroyed in the postero-superior quadrant and where the annulus tympanicus is denuded of its periosteum and covered with granulation tissue, operative intervention is indicated. This indication is emphasized where the tympanic end of the posterosuperior osseous wall has been destroyed by carious absorption. At times this effort on the part of nature to perform a radical operation provides for ample drainage and a spontaneous cure, but if the discharge continues from this opening a radical mastoidectomy is indicated and demanded in the presence of cholesteatoma.

**Character and Quantity of Discharge.**—A suppuration should be considered dangerous or only potentially so, depending on its amount and character as well as on the location of the perforation through which it finds exit. A marginal perforation, especially if situated in the postero-superior quadrant, is definitely of the dangerous type. This includes those cases in which the membrana tympani is practically all destroyed, as this uniformly indicates osseous involvement.

A continuous suppuration—mucoid, rather scanty and without fetor or subjective symptoms—indicates that the discharge is confined to the tympanic and antral regions, with no osseous involvement, and may be cured without operation. If intermittent and quite offensive, the indications are that the mastoid is involved in a necrotic process and may require operation, and here diagnosis is made more certain by the Roentgen ray.

In the presence of a long-continued, obstinate, foul-smelling discharge which has resisted all forms of conservative treatment, the radical mastoid operation must be resorted to for a cure. Although a brownish-yellow, foul-smelling discharge indicates extensive bone necrosis and, therefore, mastoidectomy, we must remember that purulent collections without odor are at times even more destructive than the extremely fetid.

When a sudden cessation of discharge is followed by pain, fever, chills, vertigo, or other untoward symptoms, the radical mastoid operation is indicated, as it is also in depression or other indications of abnormal mentality accompanying a chronic otorrhea.

**Acute Exacerbations.**—The advent of acute symptoms in a patient suffering from chronic otorrhea indicates some serious complication incident to obstructed drainage, the most common of which is a recurrent mastoiditis, and is a danger signal indicative of operation. In several instances that I

recall, patients were entirely without aural symptoms, including absence of discharge, for a period of several years before the occurrence of an acute exacerbation which was followed by brain abscess. The danger of perforation into the interior of the skull increases with each acute exacerbation.

The radical operation should be performed in the presence of a chronic otorrhea, whether scanty or profuse, continuous or intermittent, when there is persistent or recurring pain in the ear or in the mastoid, parietal, or occipital regions. When surgical intervention has been postponed until additional symptoms of an intracranial lesion become manifest, such as vertigo, drowsiness, stupor, or loss of consciousness, we are then operating not only for the relief of the mastoid disease, but its complication, thereby incurring a danger which could have been avoided by an earlier resort to surgery. Any procedure which more quickly and surely cures chronic otorrhea before complications arise is not only justified but is, after all, true conservatism.

**Granulation Tissue and Polypoid Growths.**—Slowly developing exposures of the dura and sinus through carious erosion are fairly frequent and usually harmless if not disturbed, they being walled off and covered with protective granulation tissue. This protection is also provided to a lesser degree by polypoid granulations of the tympanic cavity. Their imperfect removal through the external auditory canal, however, has caused serious intracranial complications. They may be cured oftentimes by conservative treatment when the tympanic cavity alone is involved. If they recur after several removals, the radical mastoid operation should be performed, even in the absence of additional indications. Otherwise fistulous openings involving dangerous localities are favored.

Meningitis has not infrequently followed the removal of a polypoid growth by traction, the pedicle being attached to necrotic bone which was removed by this procedure, exposing the unprotected dura to the ravages of the various micro-organisms. The radical operation, therefore, is indicated where obstructive granulation tissue or polypoid growth cannot be promptly eradicated by the more simple measures. Furthermore, we must always keep in mind that chronic otorrhea is subject to secondary infection, which may give rise to fatal septicemia either with or without actual bone involvement. In fact, a person suffering from a chronic aural suppuration is in danger of grave complications, notwithstanding an absence of symptoms for the time being.

The principal point is to rid the patient of a dangerous pathology by whatever means it can be accomplished and not to wait until there is evidence of sinus, labyrinthine, or other intracranial complications. It is, of course, advisable and necessary to employ conservative measures of treatment, but we must also be possessed of a clear surgical judgment that will dictate when operative intervention is called for to prevent the above-mentioned complications.

**Cholesteatomata.**—Ingrowth of epidermis through marginal perforations, especially those occupying the posterosuperior quadrant, is the *modus operandi* within the tympanic cavity and mastoid antrum of collections known as cholesteatomata. Generally speaking, these masses are a definite indication for complete mastoidectomy. However, in the presence of a subsiding tympanic suppuration the ingrowth of meatal epidermis does occasionally serve to cover with skin the formerly inflamed mucous mem-

brane, resulting in what is known as a spontaneous cure. Under such circumstances the osseous structure is not involved beyond repair.

On the other hand, when a suppurative inflammation is present this newly formed epidermis collects layer upon layer in the narrow spaces of the middle ear and antrum, forming a cholesteatomatous mass of varying size, which in turn sets up an irritation and establishes a suppuration of its own. These masses constantly increase in size and eventually, through pressure necrosis, an internal perforation involving the interior of the skull may occur or occasionally the accumulation protrudes into the external auditory canal.

Under the influence of pressure necrosis the surrounding osseous structure becomes actually invaded by particles of cholesteatoma. This has been shown by microscopical examination and in all probability accounts for the occasional recurrence of cholesteatoma after its eradication by means of a radical mastoid operation. In doubtful cases cholesteatoma should be suspected when, notwithstanding proper treatment, a vile odor somewhat resembling decayed cheese continues. In such cases a small quantity of foul-smelling discharge will collect at the innermost part of the meatal floor adjacent to the membrana tympani. Where the perforation is large the mass can be readily observed. In other cases shreds of epidermis will escape during syringing.



Fig. 306.—Preoperative Bell's palsy in a man fifty years old, recurrent suppurative otitis media since childhood. Cessation of discharge varied from few weeks to four years at a time; continuous for past two years. Scheduled operation unavoidably postponed, patient developing Bell's palsy in interval, making fifth case of preoperative Bell's palsy where operation had been postponed.

It has fallen to my lot to see a considerable number of cases where nature has performed an incomplete radical mastoid operation by perforation and absorption of part of the posterior meatal wall, in some instances exposing not only the tympanic attic, but also the mastoid antrum and adjacent cells.

I have been able to demonstrate that

the majority of these cases occurred as a result of the cholesteatomatous erosion and perforation. After removal of any remaining mass and appropriate after-care, it was found unnecessary to do any further operation on a fair percentage of them. In general it is a safe rule to follow that the radical mastoid operation is indicated if the diagnosis of cholesteatoma is made or even if there is a strong probability of its presence. Latterly some authorities regard the collection of a cholesteatomatous mass as malignant in character, this being an additional indication for its complete eradication so far as possible.

**Facial Palsy in Chronic Aural Disease.**—Bell's palsy or any manifestation that may indicate its development calls for mastoidectomy. I have seen cases where the membrana tympani and ossicles have been destroyed in which definite sensation in the tongue and twitching about the outer canthus of the eye of the same side were produced by wiping out the tym-

panic cavity. From repeated observations I am convinced that this twitching was wholly involuntary. These patients were few, but those on whom a radical mastoid operation was performed did not develop Bell's palsy, whereas palsy resulted in others where operation was not done. I have reported 3 cases of preoperative Bell's palsy, the operation on each being unavoidably postponed for twenty-four hours, during which interval palsy developed. The interesting question arises: Would Bell's palsy have occurred if the operation had been performed as scheduled? I should answer in the affirmative, as the facial nerve was evidently already involved and the operation would have been too late as a preventive measure. Two additional cases of this type have come under my observation within the past few months, one of which is herewith illustrated.

**Hyperostosis.**—In chronic otorrhea radical mastoidectomy is indicated where obstructive narrowing of the meatus, due to hyperostosis, is present.

**Optic Neuritis.**—The presence of optic neuritis, accompanied by continued slight headache, during a chronic otorrhea, frequently indicates the onset of serous meningitis, especially if there is no other evidence of intracranial complication, and is a definite indication for immediate radical mastoidectomy.

**Tuberculosis and Diabetes.**—The question of proper drainage is equally important in chronic suppuration accompanying tuberculosis. The radical mastoid operation, in this infection, is beneficial both to the general health and to the local infection.

Since the introduction of insulin and its proper administration and supervision in diabetes, mastoidectomy has proved to be not only wholly safe, but most beneficial to the underlying disease.

**Spinal Puncture.**—For some years I have advocated an examination of the spinal fluid to determine the presence of meningeal involvement before resorting to a radical mastoid operation for the relief of a chronic otorrhea. If a positive result is shown the radical mastoid operation is justified, even in the absence of additional confirmatory symptoms. This procedure has also the advantage of pointing out the presence of a chronic preoperative involvement of the meninges which does not manifest itself otherwise and would naturally be attributed to the operation.

On several occasions where the examination indicated the presence of meningeal irritation patients refused operation, only to succumb later to meningitis. Notwithstanding the fact that other cases, indicating the presence of a chronic meningitis or at least a meningeal irritation, died from meningitis following operative intervention, still this examination is advisable, as it will frequently serve to show the presence of a meningeal involvement that would be unsuspected otherwise. This knowledge will enable the surgeon to explain the situation and will aid him in determining whether he wishes to assume the responsibility of operative interference in such cases. Patients of this type will usually succumb to an acute exacerbation without operation, whereas removal of the focus of infection is our only hopeful means of preventing this issue.

I have seen ambulatory patients suffering from chronic meningitis complicating a chronic otorrhea otherwise almost symptomless, who suddenly became unconscious and died within a few hours, the spinal-fluid tap showing free pus in considerable quantity. This definitely presents one of the grave sequelæ complicating a chronic otorrhea; furthermore, it shows that

one may enjoy even reasonably good health in the presence of a chronic meningitis. Preventive or prophylactic measures for the relief of a chronic suppurative otitis media are, therefore, all the more urgent.

The development of Gradenigo's syndrome is a definite indication for mastoidectomy.

**Labyrinthine Complications.**—In the presence of symptoms indicating labyrinthine irritation or actual involvement, such as vertigo, nystagmus, interference with equilibrium, and perhaps nausea and vomiting, the radical operation is called for. When these symptoms are indicative of an acute suppurative labyrinthitis we should wait until it has subsided. While waiting, however, the consideration of a developing meningitis must be given due weight.

**Intracranial Complications.**—In the presence of a chronic otorrhea, even though there may be an entire absence of all mastoid symptoms, if indications point to the beginning of an endocranial lesion, such as meningitis, sinus thrombosis, or brain abscess, mastoidectomy is immediately called for. Inasmuch as I have never seen an intracranial complication develop after a radical mastoid operation, it would seem that this procedure establishes an effectual barrier against the advance of pathogenic micro-organisms to the most vulnerable parts of the brain and sinus, and unless an intracranial lesion already exists this operation may be regarded as reasonably preventive.

**Roentgen Ray.**—The assistance to be obtained from study of a case by Roentgen ray has been indicated in an earlier part of this chapter, in the consideration of the indications for the simple mastoid operation.

**Modified Radical Mastoid Operation.**—It not infrequently happens that young children suffer from a bilateral chronic otorrhea which resists persistently all non-operative measures for relief. Under such circumstances the question of resorting to the radical mastoid operation should have our most careful thought. If for any reason, operative or otherwise, these patients up to five or six years of age, or even older, lose their hearing, many will forget how to talk and eventually become deaf-mutes, producing a serious economic question, to say nothing of the tragedy to the individual.

It has been my custom with these children to perform first a simple mastoid operation on the ear in which the hearing is more impaired, on the theory that the principal pathology is located in the mastoid antrum and this procedure corrects the otorrhea in a fair number of instances. When the ossicles are intact and the perforation fairly small, regeneration of the membrana tympani occasionally results, together with a considerable improvement in audition. If the disease is not eradicated by this means, then a modified radical mastoid operation, which completely exposes the antrum, should be tried. This failing, we should seriously consider the advisability of performing the radical mastoid operation, selecting the worse ear first and noting the effect on the hearing, and should still further postpone additional operative measures if audition has been seriously impaired unless further complications threaten. Although I do not consider the modified radical mastoid operation a thoroughly good surgical procedure, I believe it has a place in aural surgery, especially in the case of children as above discussed, particularly from the fact that if properly performed any effect it may have on audition is for its betterment. Finally, the surgeon must determine which type of mastoid operation, if any, is required for the

relief of the individual case, his judgment, of course, being based on the exigency of his findings,

The indications above enumerated should make the necessity for operation comparatively clear. It is quite obvious, however, that no single case could possibly present all of these symptoms, but there should be no difficulty in arriving at a diagnosis when a reasonable number are present at the same time. Furthermore, a general rule is that when symptoms present themselves indicating the development of graver affections, such as lateral sinus thrombosis, facial palsy, intracranial lesions, or general septicemia, mastoidectomy is in order.

In the absence of urgent symptoms a safe and sane rule to follow is that the radical mastoid operation is indicated when conservative treatment will not produce the desired result. In other words if palliative measures do not bring improvement and more especially if the offensive odor and discharge continue, we should not hesitate to perform a complete mastoidectomy.

In the last analysis it is at times impossible to determine, even with the aid of roentgenology, the limitations of the gross pathology of the temporal bone, hence the fundamental principles of surgery must be applied and one of the most successful operations of modern surgery instituted for the relief of a condition of great potential danger.

S. MACCUEEN SMITH.

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## SURGERY OF THE MIDDLE EAR

Intratympanic operations are carried out in a field which is never visible as a whole to the surgeon. Under brilliant reflected light he must examine each part of the drum through a narrow canal, carrying in his mind the piecemeal picture as he operates. The surgeon must cultivate extreme delicacy of touch, and must be familiar with the various depths and elevations of the tympanic walls. Study of dry preparations, models, and diagrams avails him little unless he can grasp quickly the infinitesimal gradations of monocular depth perception necessary to proper interpretation of intratympanic changes. The surgeon who is highly astigmatic or presbyopic may well have an accurate glass correction set in the eye hole of his head mirror. The so-called daylight blue gas-filled tungsten electric globes afford a less tiring source of illumination than do the standard yellow lights. The Klaar mirror, adapted for tungsten globes, is also a most comfortable apparatus when used with a portable battery.

Dangers of intratympanic surgery are slight at the hands of the careful operator. Roughness, force, haste, and carelessness are never so disastrous, however, as in this field. It must be recalled that the facial nerve, scarcely covered by its shell-like ridge, swings just above the stapes and oval window; the chorda tympani traverses the anterior and posterior folds crossing the neck of the malleus; the exposed promontory may resemble a reddened and bulging tympanic membrane; loosened ossicles may be lost in the hypotympanum or the aditus; polypi may cover bony fistulæ leading to the brain or the labyrinth; cholesteatomatous pressure may have caused bony erosion over the dura or the jugular. Accurate identification of all

structures, especially when extensive destructive changes have occurred, must be made by cleansing, delicate probing, pneumatic otoscopy, and tubal inflation, before any operation is projected.

Instruments for intratympanic work may be set in straight or angled handles at the operator's preference. Many of wide experience like straight instruments which permit turning and lateral excursion to a greater degree; the handle may well be flattened so as to identify the direction of the blade or tip when in use. Razor-like sharpness of the little knives employed is essential. Against the intratympanic structures, so intimately connected with the perceptive mechanism for sound and equilibrium, there should be as little dragging, sawing, and slashing as possible. With smooth, sharp, polished instruments there is a minimum of bleeding and trauma, and healing is singularly rapid.

From the imposing list devised by Sexton, Delstanche, Ludewig, Politzer, Lucae, Dench, Blake, and many others, may be selected a limited variety

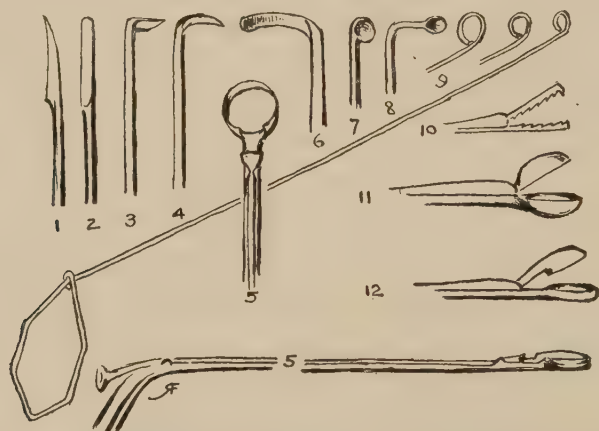


Fig. 307.—Types of middle-ear instruments: 1, 2, Knives; 3, tenotome; 4, synechotome; 5, fixation curet for malleus extraction; 6, incus hook; 7, 8, spoon curets; 9, ring curets (piano-wire); 10, alligator; 11, 12, biting forceps (magnified about 4 diameters).

of the following (Fig. 307): little bistouries, scalpels, and synechotomes; right and left angular tenotomes; straight and elbowed sharp-spoon curets; right and left incus hooks; tiny probe-hooks for the stapes; dull ring curets, straight and right-angled; sharp ring curets, of which one may be Brunschwig's clever modification of Delstanche's upward-cutting ring for extracting the malleus—a little sliding grip which holds the ossicle firmly after tenotomy; delicate forceps opening near the end; and the ordinary ear forceps, specula, and fine metal applicators and probes. Stout piano-wire may be bent into all manner of ring curets by the surgeon to suit special cases. A delicate ear snare with flexible silver cannula and very fine brass or steel wire is needed. The excellent chisel-forceps of Kerrison is essential for removal of the outer attic wall. Eustachian burrs may be used in smoothing the margins of the tympanomeatal junction.

As for myringotomy, the external canal must be freed from obstructive débris, notably by instillation of blood-warm peroxide solution, washed out freely with 1 : 3000 bichloride of mercury solution. The canal is carefully swabbed dry, and all crusts, adherent scales, or ceruminous fragments

and hairs taken out. A final swabbing with bichloride solution or bichloride-alcohol, 1 : 5000, and drying, leaves the field ready for anesthesia.

General anesthesia is best in children and neurotic adults; unless pain is very severe, the manipulations of preparation can be carried out before the patient goes to sleep. Gas-ether sequence is preferable to gas-oxygen because of the possibility of sudden reflex movements at certain stages of the latter.

In most grown persons, however, some form of local anesthetic may be employed with entire satisfaction. It is well to instil a few drops of 1 : 1000 adrenaline first of all, and to wipe the parts dry. Next, the mixture of Bonain (equal parts of phenol, menthol, and cocaine crystals rubbed together; a liquid at room temperatures) is very lightly swabbed over the field of operation. Anesthesia from this liquid is adequate in five to ten minutes, the parts touched are blanched. Instillation of aqueous cocaine solutions 10 to 20 per cent. is effective, but may cause syncope from drainage down the shrunken eustachian tube. Swabbing on is in general less dangerous. Butyn 5 per cent. is less toxic than twice the strength of cocaine, but reddens the mucosa somewhat. If the attic is to be entered, novocaine 1 per cent. with 1 drop of 1 : 1000 adrenaline to each cubic centimeter should be injected with a fine needle into the superior meatal wall near the tympanic margin. The resultant bleb will spread to the rivinian notch, and further injection may be made very close to the notch, through which subperiosteal infiltration will occur. Deep anesthesia of the membrana flaccida and of most of the attic is complete in eight to fifteen minutes. During the operation butyn or cocaine solutions may be packed into the tympanic recesses on thin cotton pledgets, but close watch must be kept for absorption symptoms, and restorative measures must be readily available. A comfortable operating chair and head-rest are highly important in avoiding shock during these trying operative procedures.

**Procedures for the Relief of Hearing Defects.**—For exploratory tympanotomy a C-shaped incision through the membrana tensa is carried up along the posterior wall, *forward*, hugging the posterior fold, *downward* along the handle of the malleus. This flap formed by the superior posterior portion of the membrana tensa is turned outward and down, disclosing the incudostapedial articulation and the oval window (Fig. 308). Examination of the ossicular chain can be made, and mobility of the stapes in the oval window may be tested by a delicate probe-hook. Great care is necessary to avoid traumatic dislocation, fracture of the crura, or injury to the stapedius tendon, and the incudostapedial joint. The mobility of the latter may be tested by pressure on the malleus, while observing through the tympanotomy flap. Adhesions not previously visible may be observed and attacked by this route, and tenotomy of the tensor tympani may also be effected. Hemorrhage is very slight. After such manipulation and comparative hearing tests as may be required, a disk of thin glazed paper, moistened with normal saline solution, is applied to the flap and permitted to adhere. The flap is then turned up into place and held to the meatal skin and membrana flaccida by the adhesive sizing of the paper splint. Inflation must not be permitted, and the flap is left in place for three or four days, when healing is ordinarily complete.

Synechotomy, the release of adhesions disclosed by probing, inflation

or inspection, is effected by division with little sickle-shaped bistouries, which may be curved to the right or left (Fig. 309). The procedure will vary with location; obviously as broad an interval as possible must be gained between the separated tissues, and occasionally little ribbons of gutta-percha, celluloid, gold or platinum foil, must be interposed. Subject to this operation are the false membranes, tympanic scars, or canopy-like superior webs of membranous tissue which divide the middle ear into closed pouches.

Closure of perforations after healing of a suppurative process may occasionally be assisted by freshening the scarred edges, then splinting the membrane upon a moistened disk of thin sized paper, as noted above. This process may need to be repeated several times to secure a firm fibrous growth with epithelial covering. The disk is best applied with a narrow swab of moistened cotton, rather than by forceps.

**Procedures for Securing Free Drainage and Removal of Septic Foci.**—Removal of granulation tissue holds first place in the surgical relief of



Fig. 308.—The flap (A) in exploratory tympanotomy. The incudostapedial joint and stapedius muscle are disclosed. Invasion of the outer attic wall by a Shrapnell perforation shown at B.



Fig. 309.—Right ear from in front. Tenotome in position to section tensor tympani (A). Adherent cicatrix over promontory, synechotome in place to cut (B).

purulent discharges. Adrenaline must precede any instrumentation, after such ears are cleansed and anesthetized. Small granular masses may now be curetted or bitten away with very little bleeding. If large polypoid growths occlude the canal, anesthetics should reach the drum on long narrow cotton pledgets before the snare loop is used. A smooth snare-cut is made without any traction. After hemostasis the pedicle or remaining portion of the polyp may be curetted or snared more closely (Fig. 295, C, p. 513). Under no circumstances should an aural polyp be dragged away by snare or forceps. Curettement of granulations should reach any underlying roughened bone, and further outgrowth should be impeded by sparing local application of 20 per cent. to full strength silver nitrate, trichloroacetic acid, or chromic acid.

Ossiculectomy may be done under local anesthesia, but is quite painful. In many cases the surgeon will steer a much easier course if general anesthesia be used, even at the cost of slightly increased hemorrhage and an unfavorable position of the recumbent head.

The membrana tympani is circumscribed from its ring, then cut away from the malleus and removed. Some operators now separate the incudostapedial joint, cutting from behind forward with an angled tenotome. The majority proceed at once to free and extract the malleus. Two methods are in general use. Many American operators now insert the angled

tenotome in front of or behind the neck of the malleus, above the attachment of the tensor tympani, and scrape it downward along the bone, thus freeing the ossicle from its internal ligament and from the muscle (Fig. 309, *A*). The malleus now hangs down from the superior ligament, and should be cut free from the anterior and posterior folds inserting about the short process. The body of the bone is now seized by Sexton's upward-grasping forceps, remaining adhesions are cut by a synchotome, and the ossicle is "delivered" by forceps traction downward, then outward (Fig. 310, *B*). Seizure by the loop of a snare, while convenient, has the disadvantage that it may cut through the bone during efforts at removal. Traction on the handle of the malleus alone is usually disastrous, resulting in fracture.

The alternative, or French method, consists in the severance of the tensor tendon by slipping the sharp upward-cutting ring curet of Delstanché-Brunschwig up along the manubrium as far as possible, cutting again the short-process fibers and adhesions. Brunschwig's sliding lock is now slipped down, firmly seizing the neck of the ossicle and facilitating its delivery as soon as the upper attachments are sectioned (Fig. 310, *D*). Section of the chorda tympani, with taste disturbances of the side of the tongue for some time, is usually consequent on removal of the malleus.<sup>9</sup>

Removal of the incus may occur with the malleus when adhesive inflammatory processes have webbed the bodies of the bones together. Here too a choice of methods obtains. Since the body of the incus lies hidden behind the superoposterior rim of the annulus, it may disappear altogether unless preliminary disarticulation with the stapes has been done. The right and left incus hooks of Ludewig (mounted at opposite ends of one handle by Vacher) are thus used by the Continental school: The concavity of the right-angled tip of the hook being turned backward toward the body of the incus, the tip is entered well up into the attic, hugging the outer wall. The hook is now rotated a quarter turn downward and backward, while the handle of the instrument outside is pressed backward a little. The concave surface of the instrument will be felt to engage a yielding bony surface, which slips back and down, and under proper conditions the incus appears in the superoposterior quadrant as a white, smooth, irregularly rounded object which should be picked up by forceps and removed at once.

Many American operators prefer opposite rotation of the incus hook (Fig. 310, *A*). With concavity forward and upward, the hook hugging the attic wall as before, it is inserted below and behind the incus, rotating the ossicle forward and downward into the visible tympanic cavity. This method obviates preliminary section of the incudostapedial joint.

Accidents in removal of the incus are not infrequent. Sustained pressure

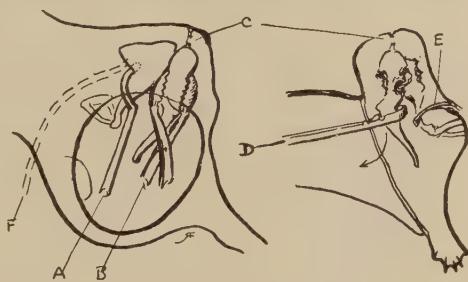


Fig. 310.—Right ear, from without, and in front. Upward-grasping forceps (*B*) holding malleus. Ruptured superior ligament (*C*). Sharp ring-curet (*D*) holding malleus; arrow shows direction of traction. Sectioned tensor tympani (*E*). Incus hook (*A*) in place for anterior rotation. Course of facial nerve (*F*).

must never be made by the hook upon unyielding bone in the posterior epitympanum or aditus, lest damage to the facial nerve occur. Too strong backward pressure may lose the ossicle in the aditus or mastoid antrum, or it may drop down into the hypotympanum, or forward into the anterior attic pouch, or slip into the eustachian tube mouth. These accidents are facilitated by hemorrhage; unless picked up or washed out, the lost ossicle becomes a sequestrum and may precipitate radical measures later. Ossicles whose processes have been lost during the course of long-continued supuration are not infrequently hard to identify and may be cut away in the midst of polypoid debris or be washed out with the attic cannula.

Stapedectomy is never permissible through an infected ear, and its value in clean cases is questionable. Tinnitus, vertigo, and purulent labyrinthitis have been observed following this procedure. The ossicle may be mobilized by delicate lateral and up-and-down probe pressure, but the foot-plate should remain in place after adhesions are removed. Thick grafts or membranous webs over this region should not be permitted to form if hearing is to be retained and assisted.

Removal of the outer attic wall demands great care in employing the powerful Kerrison or Dench forceps to cut away the heavy bone. No pressure can be permitted from the nose of the instrument against the inner wall of the attic, lest the facial nerve be crushed. The attic must be as well cleaned out as possible by right-angle curets and syringing before the heavy forceps is entered. The instrument should be pulled into place against the outer wall and held with a firm outward pull, against which the bite of the forceps should be made. The roughened surface should be rasped or curetted, as also any exposed roughened or carious spots.

After operation the cavity may be gently syringed again with warm saline or bichloride, then packed, after complete hemostasis, lightly with very narrow selvedged gauze tape. Dichloramine, 2 per cent., in chlorococaine is the best deodorant, and because of its bland oily solvent, also the best antiseptic addition to plain gauze packing. Iodoform tape, while unpleasant in odor, and sticking to granulations, is most frequently used. This first dressing is covered by an ordinary mastoid pad of gauze and cotton and left alone for twenty-four to forty-eight hours. Subsequently, light packing may be used for a week. If dichloramine be used, packing will prove an irritant on account of the free chlorine, and must be stopped in three or four days.

Discharge following ossiculectomy should gradually disappear in two to three weeks. If it continues profuse and foul, radical procedures must be undertaken.

Closure of the eustachian orifice, commonly done during the radical mastoid operation, may be attempted through the tympanum, by curettage, or reaming out by special burrs, followed by implantation of skin-grafts or infoldings of flaps from the tympanic membrane. Pierce has recently used an ingenious method depending upon the plugging of the isthmus and tubal lumen by a double-knotted catgut strand, threaded through to the throat and closing the tube by inflammatory adhesions.

Ossicular surgery, primarily valuable because it assists in the drainage of tympanic suppurations, is successful as to cure of discharge in 40 to 60 per cent. of cases. It is a conservative measure, since the hearing is retained in almost 85 per cent. of cases reported. It merits wider study and eventual

employment by younger surgeons of special deftness, who are too often trained only in the performance of the more spectacular and remunerative radical operations. As in ophthalmic surgery, a timely iridectomy may often forestall blindness and enucleation; so the intratympanic procedures may avoid deafness and radical mastoids. Furthermore, the same lightness of touch and accurate depth perception which are vouchsafed to successful cataract and glaucoma operators are needed for surgery of the ossicular chain and the other tympanic contents.

RALPH A. FENTON.

## TECHNIC OF SIMPLE AND RADICAL MASTOID OPERATIONS

### THE SIMPLE MASTOID OPERATION

The simple mastoid operation is undertaken for the relief of suppuration in the mastoid process, secondary for the most part to acute suppuration in the middle ear, and calls for the systematic investigation of the entire

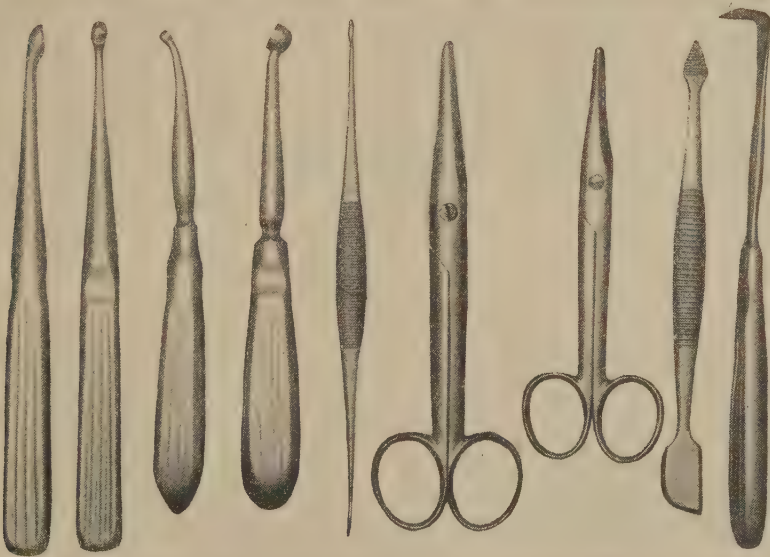


Fig. 311.—From left to right shows: Two straight curets of the type known as Spratt's; two angled curets known as Richard's; Jansen's double-ended curet for use in the tube in the radical operation; scissors straight and curved on the flat; a convenient elevator; and the Langenbeck raspatory.

cellular structure of the mastoid process and for the removal of all infected material.

For clinical operative purposes, one thinks of the mastoid process as being one of three types: First, the pneumatic; second, the diploic; or, third, the sclerotic. In the pneumatic type there is complete development of the cell structure and this type might be known as the "cellular" mastoid. In the diploic type the mastoid process conforms to the structure of the

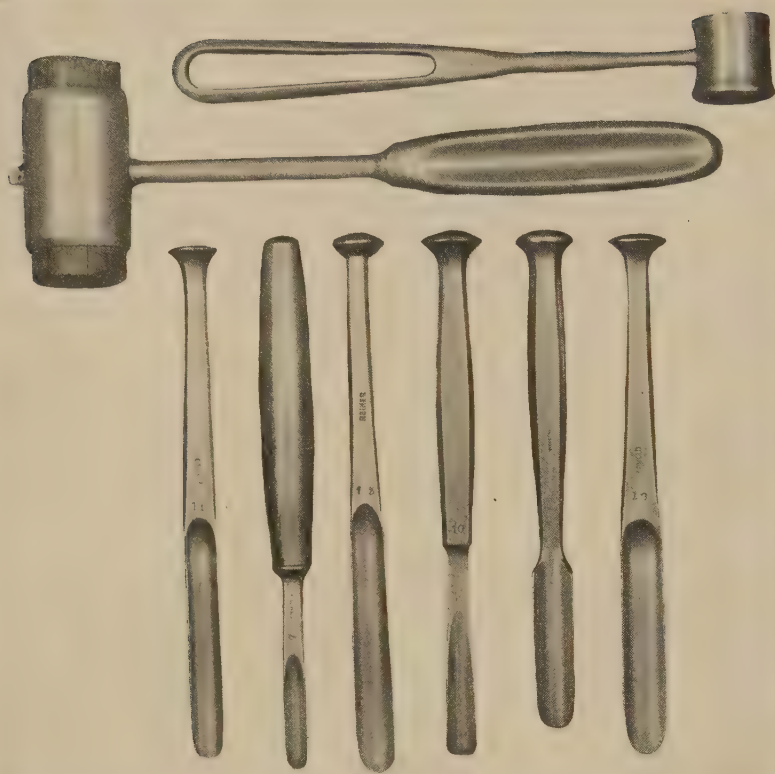


Fig. 312.—Shows different types of modern gouges all of which come in convenient sizes. Above are two mallets—one of lignum vitæ and the other of lead.

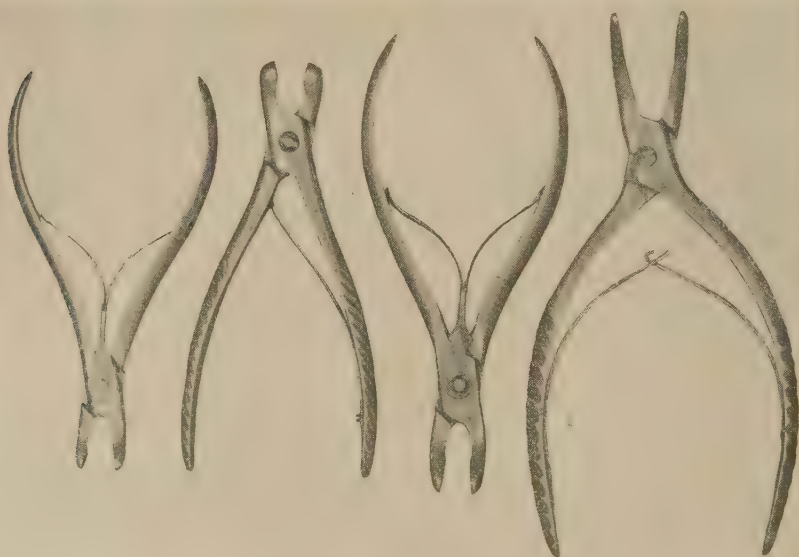


Fig. 313.—Shows types of rongeur forceps.

other cranial bones, and shows an outer and an inner table with diploë between. The antrum is usually the only cell present. The sclerotic may frequently be looked upon as a residual mastoid. As a result of chronic or recurring acute middle-ear suppuration, usually with extension to the developing cellular system, a condensing osteitis has brought about the non-development or complete obliteration of the cell system. The antrum remains, although frequently reduced in size. The remaining bulk of the mastoid process shows little or no trace of cell structure and is usually of ivory consistence. Roentgenological examination enables one to type the particular mastoid upon which operation is proposed.

**Cell Groups.**—All cells lie between the tables so that the operation may be said to be completed when one has exposed inner table in all directions and has in addition explored the region of the zygoma and has followed backward to the limit of cell structure the area between the floor of the middle fossa and the upper part of the sigmoid sinus. It must be remembered, however, that the operation is not an anatomical dissection. A

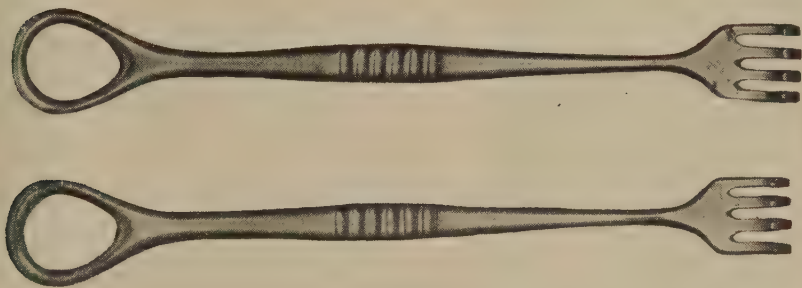


Fig. 314.—Shows long-handled sharp retractors.

highly polished inner table is the least favorable basis for granulation, and healing may very well be retarded in those cases which look best from the anatomical standpoint on completion of the operation.

**Instruments.**—Many of the instruments used in the mastoid operation, such as scalpels, clamps, thumb forceps—plain and toothed—do not differ from those used in other surgical operations and will vary with the choice of the operator.

The types of instruments peculiar or specially adapted to the mastoid operation are shown in the accompanying photographs.

**Preparation of the Patient.**—General preparation for the mastoid operation is similar to that required in any major surgical procedure. It will include attention to catharsis, restriction of diet, and examination of urine. It is also wise to know beforehand by culture the nature of the bacteriological infection, and such other laboratory investigations should be made as the needs of the case call for.

Local preparation will include shaving the side of the head over an area extending 2 inches from the middle of the external meatus. The hair edge next to the shaved area is held down by a narrow strip of gauze kept in position by flexible collodion.

The following is the method taught the nurses in the Manhattan Eye, Ear, and Throat Hospital in New York. The canal is cleansed first with hydrogen peroxide, then with 1:5000 solution of bichloride of mercury. A strip of gauze is inserted in the canal. The auricle and field of operation, the cheek and neck are next scrubbed with green soap and water, alcohol and ether, and washed off with 1:5000 bichloride solution. A moist dressing of 1:8000 bichloride solution is then applied and held in place by a bandage.

It is now customary upon the table to paint the operative field with tincture of iodine, washed off immediately by alcohol. When the iodine-alcohol method is used, the wet bichloride dressing should be omitted.



Fig. 315.—Shows a young woman prepared for operation. Note ample shaved area, hair margin held by strip of gauze fixed with flexible collodion. Note also the "forelock" retained but braided and kept out of the way. (Courtesy of the Superintendent of Nurses, Manhattan Eye, Ear, and Throat Hospital, New York City.)



Fig. 316.—Shows the incision. The auricle is not pulled, so that the postauricular fold remains distinct. Both incisions are to the bone, except at extreme upper end of the first. (Drawing from author's dissection.)

On the table the head of the patient is supported by a suitable block, and the operative field isolated by means of sterile sheets and towels. A sand-bag under the shoulder is often useful in securing a comfortable position of the patient's head with relation to the operator.

**Steps in Operation.**—In the instruction of post-graduate students, the writer has found that arranging the operation in definite steps has been a distinct help. The plan which follows is not, of course, intended as an arbitrary guide, but is to be modified according to the circumstances of the individual case. The description will be that of operation upon an uncomplicated case and reference will be made later to the conditions which lead one to modify the procedure.

*Incision*—The operator stands at the head of the table. The auricle

is folded forward on itself, but not pulled upon, so as to leave the post-auricular fold as sharply defined as possible. The incision begins over the mastoid tip and is continued upward parallel to the posterior fold and about  $\frac{1}{4}$  inch behind it, as high as the upper insertion of the auricle. The incision is to the bone except at its upper extremity where it is advisable to avoid cutting the temporal muscle. The distance between the post-auricular fold and the incision should be such as to permit easy retraction of the flaps and yet allow comfortable introduction of sutures on completion of the operation.

This single incision is sufficient in the average case, but where additional room is needed, it is obtained by splitting the posterior flap at right

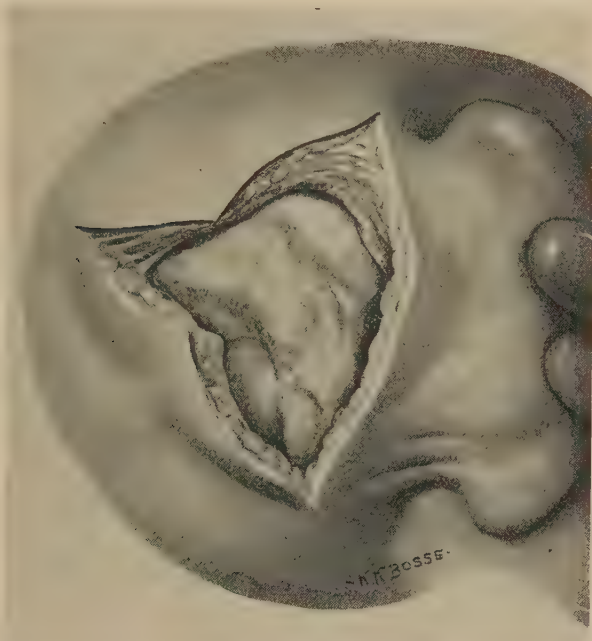


Fig. 317.—The periosteum has been elevated and the tip freed. The surface of the mastoid process is unusually irregular. (Drawing from author's dissection.)

angles, beginning at the level of the middle of the external meatus and continuing backward for an inch or more.

With the single incision one is dealing with two flaps, an anterior and a posterior. With the extra incision one has three flaps, an anterior, an upper posterior, and a lower posterior.

*Elevation of Periosteum.*—This is best done by the Langenbeck raspatory, and is easy when the incision has been carried to the bone. The anterior flap is pushed forward until the posterosuperior curve of the external bony meatus is seen. The cartilaginous canal is not disturbed. The anterior surface of the tip is carefully cleared.

The posterior flap is pulled backward by the raspatory and the posterior surface of the tip dealt with at the same time. Where an extra posterior incision has been made it is wise to elevate the lower posterior flap last, as one is liable to meet hemorrhage from the mastoid emis-

sary vein. All bleeding points in the flap should be clamped to secure a dry field.

*Freeing the Tip.*—The attachment of the sternomastoid muscle should be freed until the point of the finger can be passed around the tip. This is best done by scissors curved on the flat. The scissors is closely applied to the bone and by a series of short snips, the detachment is made. If the scissors be held in the hand with the palm upward and their curve made to conform to the curve of the mastoid tip, the separation can be made close to the bone. A little time spent in securing clean elevation of flaps and a clear time means time saved later, as bony fragments that remain



Fig. 318.—The cortex has been removed in successive grooves, and disclosed a very cellular process. Note large superficial cells at tip. (Drawing from author's dissection.)

attached to periosteum or aponeurosis do not come away, but must be freed individually by scissors.

*Retraction.*—Where an extra nurse is available long handled sharp retractors are best, as retraction can be made exactly where and when needed. If circumstances prevent the employment of this extra assistant some form of self-retaining retractor, such as the Allport is used.

*Removal of Cortex.*—In former years the next step was the opening of the mastoid antrum. This was attempted through as small an area of cortical bone as possible and its successful accomplishment was demonstrated by the passage of a bent probe through the cortical opening and the aditus ad antrum into the middle ear. This procedure has rightly been abandoned and it is now customary to remove the whole cortex in successive grooves, the first of which runs from its starting point to the tip parallel to the posterior bony canal wall. The apex of the suprameatal triangle is

the point of election for the application of the wide gouge which is recommended for the removal of this first groove of cortex. The original supra-meatal triangle was described by Macewen. It has for one side the posterior root of the zygoma, for a second side the posterosuperior bony canal wall, and is completed by joining these two sides. It is not always easy to get a definitely marked posterior root of zygoma in the operative wound.

Dench describes a suprameatal triangle formed by drawing tangents to the superior bony canal wall and to the posterior bony canal wall. This triangle, while practical, seems to call for quite a little dissection if it is to be determined with accuracy. Fortunately, the margin of the posterosuperior canal wall can always be readily demonstrated, and if with that as a base one constructs in imagination an equilateral triangle no other landmark is necessary.

The cortex of the first groove having been removed, the procedure is continued backward in successive grooves until the whole cortex has been taken off. The nature of the bony structure can be determined on this exposure, and the exact method of continuing planned. The usual mode of procedure is to deepen the first groove with a gouge not so wide as that originally employed. If softened cell structure has been exposed the operation may be continued by using curets. One must look carefully for inner table over the sigmoid sinus and satisfy himself when the antrum has been opened. The appearance of the aditus ad antrum and its location form sufficient evidence that the antrum has been reached. It is not advisable to use a probe to demonstrate the fact. The reason for this is apparent if one remembers that the short process of the incus lies in the aditus and the forcible introduction of a probe is quite liable to dislocate the ossicle.

From the region of the antrum one finds a series of cells running down toward the tip and encroaching upon the posterior canal wall. At the tip, itself, it is not unusual to find quite large cells immediately under the cortex or deeper. This series of cells, often at quite a deep level, encroaches upon the inner table covering the sinus and curving backward from the sinus, is continuous with the cells posterior to the sinus and between that vessel and the posterior surface of the tip. Wherever one can demonstrate overhang of outer table by clearing away infected cell material between the tables, this overhang is most easily removed by one of the heavier rongeur forceps.

Passing backward from the antrum and between inner table forming the floor of the middle fossa and inner table covering the sinus, one frequently finds well-developed cells. These must be cleaned out as far as they extend backward. Overhang posterior to the sinus must be removed downward toward the tip until one is satisfied that all infected material has been removed in this location. There need be no definite step in the operation calling for the removal of the tip as the systematic exploration of the cells in the posterior wall, in the group running from antrum to tip, and in the group running posterior to the sinus will have guaranteed its removal.

The region of the zygoma calls for exploration. Neglect of this is one of the causes of delayed healing and secondary operation. One must be careful not to expose dura and in this region one must think of the facial nerve. Working upward, outward, and forward from the aditus, there is no danger as the back of the curet will be directed toward the horizontal semicircular canal and the cutting edge away from the facial nerve.

*Lowering of Posterior Bony Canal Wall.*—With heavy forceps the posterior bony canal wall is lowered in its whole length from  $\frac{1}{8}$  to  $\frac{1}{4}$  inch and smoothed off with a large curet. This is done for two reasons: first, that the auricle may sit back better on cicatrization, and second, to lessen the depth of the operative wound. The latter reason seems to be the more important as so much less space is left to be filled by granulation.

*Suture and Dressing.*—The wound should be flushed with hot saline solution and the canal irrigated. The practice of forcibly injecting solution with a piston syringe through the aditus and middle ear into the canal is

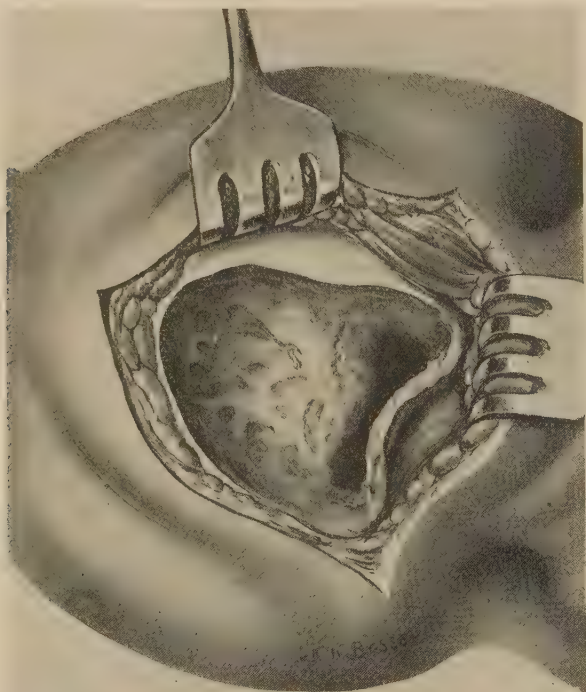


Fig. 319.—Shows completed simple mastoid operation. The bulge of inner table over the sigmoid sinus is distinct. The irregularities represent the floors of cells lying on inner table and purposely left a little rough. The posterior canal wall has been lowered and smoothed off. (Drawing from author's dissection.)

condemned. Hemostats having been removed and bleeding points ligated when necessary, the wound itself should be lightly packed and sutured so as to leave only a slight opening at its lower end. A strip of gauze should be inserted into the cartilaginous canal and the usual mastoid dressing and bandage applied.

**Mastoiditis with Subperiosteal Abscess.**—Where subperiosteal pus is present the usual incision should be made. Before proceeding to elevation of the periosteum, the abscess cavity in both flaps should be carefully curetted, and the operation carried out as already described. In adults it is the rule to find a cortical perforation in cases with subperiosteal abscess. This is usually in the region of the antrum and one may be able to begin the operation at the site of perforation, enlarging the opening with rongeur forceps, and omitting the use of the gouge.

Occasionally, the cortical perforation is so far back as to make it advisable to begin in the region of the suprameatal triangle and modify the procedure in accordance with the findings. In infants and young children one may find a subperiosteal abscess without cortical perforation. Such a case calls for complete exploration of the mastoid.

**Mastoiditis in Infants and Young Children.**—Upon completion of the mastoid operation in infants and young children, one is struck by the fact that, although the antrum may be the only cell present, the operative wound resembles a miniature adult mastoid. Edema or abscess is frequently present and the periosteum is bound down at the squamomastoid suture. One should proceed with caution in the elevation of the periosteum and dis-



Fig. 320.—Shows beginning of application of bandage. The short piece placed over head and face is afterward used as tie to secure bandage. (Courtesy of Superintendent of Nurses, Manhattan Eye, Ear, and Throat Hospital, New York City.)

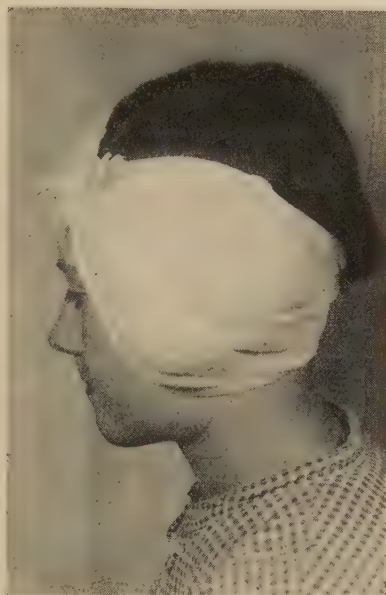


Fig. 321.—Shows completed mastoid dressing.

card the use of scissors entirely. The antrum is relatively higher and may usually be opened without difficulty with a curet. Indeed, the operation may be carried to completion with the use of one or two instruments. Where edema or subperiosteal abscess is present a wet dressing is indicated, and the writer has found boric acid and alcohol to make a satisfactory application.

**Accidents During Operation.**—Injury to the facial nerve in the performance of the simple mastoid operation is extremely rare if one is careful in working around the region of the aditus ad antrum and of the posterior canal wall at the depth of the aditus or even deeper.

While working in the latter situation it is advisable to keep close to the sinus rather than run the risk of injury to the nerve. One should work as a general rule in the line of the course of the nerve, and the anesthetist should be asked to watch carefully for twitching of the orbicularis muscle.

Occasionally, in long neglected cases a large part of the tip will separate as a sequestrum. The writer has seen such a case in which removal of the sequestrum resulted in complete and permanent facial paralysis.

**Exposure of dura** should be avoided when possible, but one should never hesitate to expose dura rather than leave carious bone in contact with it. Experience teaches that granulation takes place more readily from exposed dura than from smooth thin inner table, and it is extremely rare for any complication to arise as a consequence of this necessary exposure.

**Injury of Dura.**—Injury to the dura is quite uncommon. If the dura has been accidentally torn, free exposure in the neighborhood of the tear should be made and the injured part packed off separately by a fold of iodoform gauze. The same remarks apply to exposure of the sigmoid sinus. It is the preference of the writer to expose sinus when called for with a wide gouge. There is little chance of injuring the sinus wall as long as the gouge can comfortably straddle the opening in the bone. The lower lip of the gouge acts as an elevator and keeps the sinus wall itself out of danger while the bone is being removed.

Accidental opening of the sinus results in copious hemorrhage. This, however, can be controlled easily, first, with a large piece of gauze. Then the operator at his leisure can replace this large piece with a tiny piece applied at the exact location of the bleeding. Gentle pressure controls the hemorrhage and no step in the usual course of the operation need be omitted on account of the accident to the sinus wall.

#### THE RADICAL MASTOID OPERATION

The radical mastoid operation differs from the simple mastoid operation in that the operator, upon completion of the simple operation, takes down the posterior canal wall and proceeds to deal with the pathological contents of the middle ear and its bony walls.

The operation is undertaken for the relief of mastoiditis in the presence of chronic middle-ear suppuration with caries of the walls and ossicles. It is also undertaken for the relief of chronic middle-ear suppuration without mastoiditis and may constitute a preliminary step in other operative procedures, such as the labyrinthine operation. Where undertaken for relief of chronic middle-ear suppuration without mastoiditis, it is possible that it will be sufficient to open and clean out the antrum, remove the posterior canal wall, clean out all diseased material from the middle ear, and thus convert antrum, middle ear, and canal into one cavity with its most favorable point for drainage at the external meatus. This might be very well described as the antrotympanic operation. In most cases, however, a complete mastoid operation is called for.

**Steps in the Operation.**—*Incision.*—It is the custom of the writer to use the same incision for the radical operation as for the simple. The incision usually described is one with a generous curve backward, reaching from the mastoid tip to the upper insertion of the auricle. This leaves an anterior flap very wide at its middle part, thus making it difficult to retract and obtain the best exposure. The original idea about this flap was that on complete closure of the posterior wound, the line of skin suture would be behind the opening in the bone and thus be less liable to infect the wound and less apparent on cicatrization. The writer has not found these advantages sufficient to balance the benefit of easy retraction and good exposure.

*Elevation of periosteum* is carried out exactly as described for the simple operation.

*Retraction of Cartilaginous Canal.*—One may leave this step until completion of the mastoid part of the operation, but there seems to be some advantage in doing it early. With a suitable elevator the cartilaginous canal wall is separated first on its posterior aspect, and later, above and below. On reaching the depth of the drum a tape is inserted through the external meatus. With a toothed forceps between the posterior cartilaginous and bony canal walls the deep end of the tape can readily be picked up and pulled out. Separation of the cartilaginous wall can now be completed and the tape, having by see-saw motions been spread evenly, is used to retract both auricle and canal. The mastoid part of the operation is completed as described above.

*Removal of Posterior Bony Canal Wall.*—There is no danger of doing harm in removing posterior wall until one gets to the depth of the aditus. It is best to begin with a heavy forceps, straddling the wall and removing successive bites as far as possible. The wall can be thinned by the use of a sharp curet. The back of the curet lies in the aditus and the bulk of the bridge can be diminished by side-to-side motions of the curet, always working outward and upward. There is no reason why the probe should not be used in this operation so that one can be informed as to the identity and course of the aditus whenever desired. The careful use of the chisel at the upper or lower extremity of the bridge may be of assistance at any stage. Working in this manner one finally reaches the point at which a small part of the bridge remains, and this is broken through with a curet. A probe can now be passed through the aditus into the middle ear and lifted out through the gap in the posterior canal wall. Additional room is now obtained by the use of the chisel or curet at the upper end of the bridge.

*Removal of the Inner End of Superior Canal Wall.*—The inner end of the superior canal wall is the floor of the attic and makes a distinct projection into the middle-ear space. This is best removed under inspection from the region of the aditus by side-to-side movements of a sharp curet which should be continued until the ledge is completely gone so that the roof of the tympanum will make one smooth surface to the inner end of the upper bony meatus.

*Removal of Débris from Middle Ear.*—The horizontal semicircular canal has been identified long before now, and the next step is to remove remains of ossicles and granulation from the middle ear. It may be possible to see remains of incus or malleus, when their extraction can be effected by forceps. In the absence of such identification débris may be removed with a sharp curet, care being taken to keep as close to the floor of the tympanum as possible.

It is sometimes a good plan to make the first rough cleansing with a piece of gauze passed into the middle ear and swept out along the floor with thumb forceps. Remains of ossicles should be identified. Frequently a small part of the malleus consisting of carious head and short process will represent all that is left of ossicles. It is often convenient at this stage to pack the middle-ear space with a small pledget soaked in adrenaline.

*Removal of Lower Part of Posterior Wall.*—The lower pillar of the bridge can be safely removed until one reaches the level of the face of the horizontal

canal. This may be done with sharp curet or chisel, according to the choice of the operator.

*Cleaning the Walls of the Middle Ear in Turn.*—1. The internal wall (paries labyrinthica): The outstanding landmark on the internal wall is the promontory covering the first turn of the cochlea. Above and posteriorly is the oval window occupied or not by the stapes. Immediately above the oval window lies the facial nerve inclosed in the aqueduct and above the nerve, the prominence of the horizontal canal. Behind and below the promontory is the region of the round window. One should carefully avoid all manipulation of instruments in the neighborhood of the oval window; first, because of the danger of opening up the labyrinth to infection; secondly, because of the danger of injury to the nerve in case erosion of bone has caused its exposure.

2. Anterior wall (paries carotica): The important relations on the anterior wall are the tympanic opening of the tube and the processus coch-



Fig. 322.—Shows completed radical cavity. The bulge of the sinus, the prominence of the horizontal semicircular canal, the promontory, the oval window, and the fossa of the round window (the latter somewhat diagrammatic) can be distinguished. (Drawing from author's dissection.)

leariformis which can be seen as a distinct prominence passing from the upper aspect of the tube across the promontory almost to the anterior edge of the oval window. This contains the body of the tensor tympani muscle. The relation from which the wall takes its name is the carotid artery. Closure of the tube is considered essential to success in this operation. One therefore proceeds to curet the tube and the instrument devised by Jansen is satisfactory. One should endeavor to remove the lining of the tube from its opening in the tympanum as far down as the isthmus. The lining should be everted. It is also considered advisable to break down the processus and evert the muscle.

3. The floor (paries jugularis): The important relation on the floor is the jugular bulb and one must not forget that cases are on record in which the bony floor was lacking and the bulb projected into the middle ear. This

is an extremely rare condition. It is more common to find some cell structures in the floor and these should be carefully cleaned out.

4. The roof (*paries tegmentalis*): The relation here is middle fossa. In breaking down the bridge and removing the inner end of the superior wall one has already attended to this part of the operation.

5. Posterior wall (*paries mastoidea*): The posterior wall has for its relation the *aditus ad antrum*, and this has also been already dealt with.

*Cutting the Flap.*—Before dealing with the membranous canal the whole operative cavity should be thoroughly cleansed with hot saline and all bony fragments removed. The purposes of the flap are, first, to enlarge the meatus so as to facilitate dressing and to aerate the cavity and, second, so to place the ends of the flaps as to favor the ingrowth of epithelium with which we expect the operative cavity to be lined.

Many types of flap have been described, from one calling for simple splitting of the posterior wall to quite elaborate flaps in which the skin of the concha is used after dissection and removal of its cartilage. The writer has discarded all other types of flap in favor of the Siebenmann. This flap enlarges the meatus without deformity and does not call for dissection of cartilage or special suture. With the forefinger of the left hand in the meatus pointing backward, a straight scissors is inserted into the canal from its deep end. The point of the scissors will be felt by the finger in the meatus. With one snip the posterior wall is divided in the middle line to the tip of the finger, but not quite to the external meatus. From the deep end of this incision another is made with a single cut upward and forward to the middle of the superior wall of the meatus. Still keeping the left forefinger in position, a third incision is made from the end of the original incision forward and downward to the middle of the floor of the meatus. If care is taken, the flap may be made in three successive cuts without removing the scissors. Inspection will now show one that he has three flaps—an upper, a lower, and an anterior. One can prove that the meatus is enlarged by his ability to pass the tip of the little finger of the left hand readily through the external meatus. It will be apparent that all three flaps will be held in position by the dressing without suture.

*Suture and Dressing.*—It is expected that subsequent dressings be made through the external meatus, but it is not always wise to close the posterior wound completely at the time of operation. According to the judgment of the operator, it may be better to leave the extreme lower end of the incision with a small drain projecting until at least the first complete dressing. The

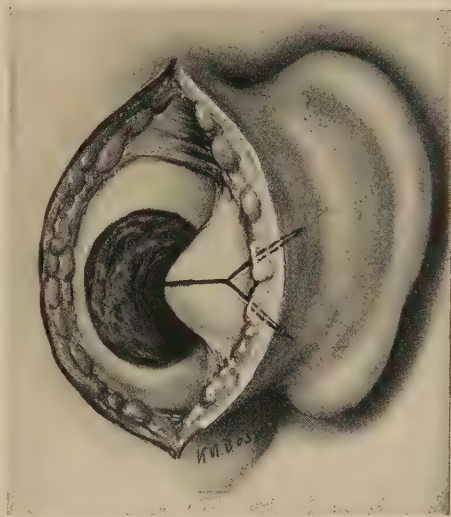


Fig. 323.—Shows the Siebenmann flap. Dotted portions indicate the cuts to the superior and inferior meatus. These incisions do not include the skin parts.

remainder of the posterior wound is sutured and the packing is inserted through the external meatus. The usual outer dressing and bandage are then applied.

**Accidents in the Course of the Radical Operation.**—Exposure of and injury to the dura or sinus are not more likely in the course of this operation than in the simple and should be handled as already described.

The possibility of injury to the facial nerve is much greater than in the simple operation, and the surgeon should not only be constantly on guard to avoid it, but the patient should invariably be warned beforehand of the possibility of this mishap. It must never be forgotten that the surgeon is dealing with a pathological bone, that the amount of destruction cannot be accurately forecast, and that due to disease the nerve itself may be exposed.

Instrumental manipulation in the neighborhood of the oval window, particularly above it, and in the posterior canal wall when one has reached the depth of the face of the horizontal canal, should be undertaken with the greatest care and the anesthetist should be especially warned to look for twitching of the facial muscles.

When facial paralysis is observed immediately on recovery from anesthesia, it is probable that direct trauma of the nerve is the cause. When observed twenty-four to forty-eight hours after the operation, it is probable that one is dealing with a pressure paralysis, the result of edema within the sheath and not of direct injury. Prognosis in the latter case is as a rule good.

Where division of the nerve is the cause of paralysis the question of anastomosis either with the spinal accessory or hypoglossal must be considered later.

#### CARE OF THE PATIENT FOLLOWING SIMPLE MASTOID OPERATION

The twenty-four hours immediately following the mastoid operation are liable to be stormy. Pain and restlessness are controlled by the use of codeine or morphine, by mouth or hypodermically, sufficient in amount to insure sleep. The patient is kept on liquid diet, and the bowels should be moved on the day following operation.

The outer dressing is made daily and the sutures inspected for any appearance of suppuration. Needless to say, the sutures should be removed on the first suspicion of suppuration. At each daily outer dressing the drain is removed from the canal, the latter carefully cleansed, and the drain replaced.

The first full dressing is undertaken on the fourth or fifth day. In little children and in many adults it is wise to do this full dressing under nitrous-oxide anesthesia. The packing is removed from the posterior wound and the wound itself cleansed, if necessary. A small drain replaces the packing, and when this is completed the sutures are removed. The custom of doing the first full dressing under anesthesia is recommended. In little children the dressing is done leisurely and without struggle. Most adult patients dread the mastoid dressings, and as subsequent dressings are free from pain, the advantage is obvious. After the first full dressing, there being no indications to the contrary, the patient may be put on full diet and permitted to get up and walk around.

After a few dressings it will usually be found practicable to dispense with the bulky postauricular dressing and bandage, and a smaller and less

noticeable dressing can be employed. Various pads fitting behind the ear and held in position by tapes have been devised for this purpose. These will readily suggest themselves to the individual operator or nurse. Later a small strip of adhesive plaster which should not cover the entire dressing will be found sufficient.

In the majority of cases in which the trouble in the mastoid has been secondary to an acute otitis media the middle ear will be found to be dry, with the opening in the drum entirely closed after a few days. It is wise in these cases to think of the hearing, and there is no reason why early inflation with the catheter should not be practiced if any defect in hearing is discovered.

The uncomplicated case generally makes a rapid recovery and it should be the object of the otologist to encourage healthy granulation. This is best accomplished by restriction of packing and the average case does better if only a small drain or no packing at all be used. Exuberant granulation, as it contains no nerves, can be painlessly removed with the scissors if large in amount or by the application of a solution of silver nitrate, up to 480 grains to the ounce, if small in amount.

Occasionally, for no very apparent reason, granulation is delayed and the otologist must face the situation of the wound skinning over with a large postauricular depression. Various medicinal agents are employed locally to stimulate granulation, the best known and most frequently employed being balsam of Peru. Internal medication, such as cod-liver oil, should not be forgotten.

In delayed granulation, if the case is one in which the wound is healthy, it may be wise to make a secondary plastic operation. The edges of the wound are freshened and elevated. The wound cavity is permitted to fill with blood, and when this has clotted the edges are brought together throughout the entire length of the wound. The writer has employed this method successfully on several occasions; not only is considerable time saved, but a much more sightly scar results.

Healing is sometimes delayed by the presence of carious bone. This can be detected by the probe. It is not surprising when one considers the structure of the mastoid and the nature of the operation that this condition sometimes results. Indeed, it is more surprising that it does not occur more frequently. Occasionally a small sequestrum embedded in granulation may be detected, and on removal of this sequestrum healing promptly follows. On rarer occasions secondary operation must be considered to secure healing.

#### TREATMENT AFTER RADICAL OPERATION

The immediate treatment after the radical operation is quite similar to that after the simple mastoid operation. Outer dressings are done daily and the sutures inspected. If a drain has been left in the lower angle of the posterior wound and the case is progressing favorably, this drain can be removed at one of the earliest dressings. On the fourth or fifth day packing is removed by way of the external auditory meatus and the sutures taken out.

As soon as is practicable, one dispenses with the bulky posterior dressing entirely. The two main purposes of the radical operation are to secure freedom from discharge and to save as much hearing as is possible. The patient should be seen daily and the wound kept clean with little disturbance. It is a good general rule to keep the middle ear and antrum as

dry as may be, but one should not hesitate to irrigate when such treatment is indicated. Boric acid powder blown into the middle ear and antrum makes a good dressing in many cases. Profuse granulation may call for continued packing. Much granulation is not to be desired as its later change into fibrous tissue will constitute a mechanical obstruction to hearing. Various antiseptic drops may be required and those do best in the presence of granulation which have alcohol up to full strength as the fluid constituent.

If the flap has been successfully made, there will be free aëration of the cavity and this should not be hindered by any dressing.

JOHN B. RAE.

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### SIMPLE MASTOIDECTOMY BY THE "BLOOD-CLOT METHOD"

The simple mastoid operation, mastoidectomy or mastoid exenteration, is performed ordinarily for the relief of acute or subacute purulent inflammation of the mastoid portion of the temporal bone; occasionally it may be found applicable to chronic mastoiditis, especially in children, but to avoid possible confusion with the more extensive procedure of tympanomastoid exenteration—the so-called "radical mastoidectomy"—we shall here limit consideration to its employment in the treatment of acute conditions.

Every surgical procedure should rest upon a clear understanding of the pathology of the condition to be treated; which is to say, upon a comprehension of the essential nature of the disease and of the structural and functional changes possibly induced by the disease. It will not, therefore, be amiss to review briefly the pathology of acute mastoiditis.

Primary inflammation of the cellular mastoid process is very rare. Ordinarily mastoiditis is secondary to otitis media, but in just what proportion of all cases of acute purulent inflammation of the tympanum infective secretions actually invade the antrum we cannot say. Some eminent authorities believe that such invasion is an invariable accompaniment of suppurative otitis media. That the opportunity for such trespass is always present may be granted, but there are also certain restrictive factors to be considered. The aditus ad antrum is not always a large, widely open canal; it is frequently small in diameter. Furthermore, it is conceivable that advancing inflammatory swelling of the tympanic mucous membrane, preceding the formation of pus, may occlude this channel and tend to prevent overflow or extension into the antrum. On the other hand, it is to be remembered that the physics of this process may sometimes work toward the opposite effect, disastrously, through penning up pus which reached the antrum early and remained to cause trouble there after protective closure of the passageway took place and after inflammation in the tympanum had subsided.

The canaliculi uniting the antral space with the other cells of the mastoid process are usually very small, and if it be true that the antrum is so frequently concerned in the inflammatory affections of the tympanum, the swelling which accompanies inflammation of the lining tissue of this cavity must generally serve to wall off the abscess and limit its progress. Under

such circumstances any excretions that may be produced find their exit into the tympanic cavity by the existing channel, after the swollen condition of the tissues has subsided, or become an isolated focus of suppuration. The pathological process then is not different from that occurring under similar circumstances in any other part of the body. When the mucous membrane becomes inflamed, the underlying bone is very apt to be affected, and to participate in the degeneration if the irritation persists. The speed with which an inflammation advances depends upon the virulence of the invading micro-organism, the density of the structures, and the powers of bodily resistance. By a process of molecular disintegration or ulceration the abscess cavity grows as the necrosis advances. Naturally, the more dense the bone, the slower will the pernicious change proceed; the more cellular its composition and the larger the vacuoles, the more rapid and extensive will be the destruction. The normal histological structure of the mastoid portion of the temporal bone is subject to wide variations, the antrum mastoideum and the apical cell near the tip of the process being the only fairly constant cellular spaces existing therein. The location of other pneumatic areas may play an important part in determining the course which a destructive suppuration shall pursue. Thus, in a purely pneumatic type of mastoid the entire internal framework may be rapidly broken down and the cortex perforated. In the diploëtic or the partially eburnated form the necrosis, advancing in the direction of least resistance, may descend toward the apex of the mastoid, ascend toward the cerebral cavity, or proceed anteriorly, posteriorly, internally or externally, toward the digastric fossa, the lateral sinus, the internal ear, or the outer surface, respectively, according to the resistance encountered. When an active inflammation has once become established in the mastoid bone, even when limited to the antrum, the escape of its products through the tympanum and external auditory canal is difficult, the floor of the antral cell being decidedly lower than the antrotympanic canal, and retention of the deleterious substances is favored by physical conditions.

Fortunately, not every case of mastoiditis goes on to complete destruction of the mastoid process or to invasion of surrounding structures. In many instances the antrum alone is involved, or only a limited area of bone disintegrates, and the purulent mass is discharged through the tympanum and external auditory canal. Occasionally the necrosis may cause segmentation of a small area of bone and the formation of a sequestrum. When the inflammation is checked in its incipency, or is of the milder type, nature endeavors to remove the irritant through increased vascularization, and to repair any injury by new tissue formation.

The portions of the mastoid bone which appear to be most frequently concerned in circumscribed abscess formation are, in the order named, those involving the group of cells immediately below and in front of the antrum, including that wedge-shaped area between the antrum, tympanum, and external canal; the cell or cells situated in the tip of the process; the vertical line of cells along the posterior border of the process, encroaching upon the sigmoid sinus groove; and the supra-antral cells located in the tegmen mastoideum.

As might be inferred from this description of the pathological process, acute inflammation of the mastoid, occurring as a sequel to acute suppurative otitis media, has a favorable prognosis. The treatment to be

employed, as well as the gravity of the affection, depends upon the stage to which the disease has progressed, *i. e.*, whether it is simply in a state of congestion or has reached the condition of empyema. If it can be determined that pus is present in the cells, the prognosis can be kept favorable only by prompt institution of measures for its evacuation. On the other hand, if it is not clear that pus has formed, tentative measures may be employed with a reasonable hope of aborting the process. The danger of mastoiditis lies not in the disease itself, so long as it is confined to the cellular structures of the mastoid process, but in the possibility of early and rapid invasion of nearby vital structures, and the strong probability that such extension will take place if the proper treatment be delayed or neglected. When a reasonable doubt exists as to the advisability of instituting operative measures, an exploratory opening of the bone is not only justifiable but affords the patient the greatest possible assurance of safety.

The indications for mastoidectomy need not be considered here, since they are dealt with fully elsewhere in this book, but there is one symptom of mastoid disease that is much relied upon as a guide to operative intervention and of which we should like to speak. Next to localized pain and the external evidences of underlying disease in this region, *tenderness* in response to pressure over certain areas is the predominant factor in determining the existence of mastoiditis and in deciding upon the progress the disease is making; an increasing tenderness from day to day being considered as positive evidence that the ulcerative process is advancing. What we would wish to emphasize here is the importance of measuring that tenderness with some degree of accuracy. The method most commonly employed is to make pressure with the thumb or forefinger over the site of the antrum. Time and again we have seen consultants differ in opinion as to the existence of tenderness, or as to the degree of tenderness elicited, and this would seem to be natural when we stop to think that no two persons have fingers of the same size or delicacy of touch and no one can possibly tell whether both examiners are exerting the same amount of pressure; in fact, no one person can be absolutely sure that he always exerts the same amount of pressure when testing the same patient on different days. The very small superficial area that corresponds to the location of the antrum is slightly depressed below the general surface of the mastoid bone and if the examiner possesses a very large thumb or bulbous forefinger he may well cover a large area of the mastoid surface and make considerable pressure on the ring of bone surrounding the depression and very slight pressure on that part directly over the antrum; whereas the examiner with a small fingertip will elicit tenderness in a given case with considerably less pressure because he more easily touches the spot of vital importance.

Some years ago an instrument was devised for the accurate measurement of mastoidal tenderness and in clinical use it proved of incalculable service to the author. The *algometer* (see Blake and Reik's *Surgical Pathology and Treatment of Diseases of the Ear*, p. 322) consists of a fenestrated cylinder in which a slender piston-rod moves against a resistance spring. The outer end of the rod is capped by a smooth, spherical button, and the inner end is attached to an indicator that slides in the fenestrum. The margin of the slit in the cylinder is marked to show the amount of pressure being exerted upon the piston, being graduated in hundreds from zero to 2000 gm. With such a device it is possible to determine accurately

the amount of pressure required to induce tenderness at any given spot. If pressure be made with this instrument over the antrum or mastoid tip of a normal healthy temporal bone, the full amount of power it registers can be borne without material discomfort. If less than 700 gm. of pressure elicits a sense of pain, one can be reasonably sure of the presence of inflammation and, if the amount of pressure required to induce tenderness varies from day to day, one can readily determine whether the condition is improving or is becoming worse.

We confess to having felt some disappointment that this instrument was never widely adopted by the profession, for its clinical value can be so positively proved. We insist upon accurate measurement of the temperature, upon an accurate count of the leukocytes, and upon recording such facts daily in a case under observation, and yet the majority of us seem to be satisfied with the most inaccurate and careless method of testing for tenderness while admitting that this is the most important of all the symptoms to be considered when trying to decide whether or not the mastoiditis requires surgical treatment. Once more we enter a plea for the employment of this instrument, or of some more accurate means, in the estimations of mastoidal tenderness.

Having decided that a mastoid operation is necessary, the next point for serious consideration is, naturally, the character of operation to be performed, and in that respect we lean heavily in favor of the general applicability of the technic devised by the late Dr. Clarence John Blake, for many years Professor of Otology at Harvard University Medical School, that operation which has come to be known best under the title of the "blood-clot" procedure. There are doubtless some cases of mastoiditis to which one would from the start consider this operation unsuitable, and there are others where one would find it impossible or inadvisable to carry through the plan because of conditions disclosed during the actual operation, but in the vast majority of instances, in our opinion, the effort to procure primary healing is justifiable and the operation itself is worthy of being designated as the ideal form of mastoidectomy. We have used this term "ideal" in relation to this operation because it submits the patient to the least risk, additional to the disease, offers the maximum assurance of cure in the shortest space of time, and leaves the minimum of scar as a sequel. However well devised an operation might be, if its performance added materially to the risks and dangers the patient already faced in the disease itself, it would not be an ideal procedure, and unnecessary prolongation of the period of recovery or an unnecessarily large or unsightly postoperative scar would prevent a skilfully performed and practically dangerless operation from being classed as truly ideal. The experiences of a steadily growing number of surgeons, through a long number of years now, enable us to say quite positively that this method of operating is certainly no more dangerous than any other form of mastoidectomy. The blood-clot method is admittedly more difficult for the surgeon than is the ordinary operation of simply opening the bony process, draining the abscess, and waiting for the cavity to heal up by granulation from the bottom. But that is no argument against its performance; we owe every patient all of the care, all of the skill, and all of the labor that correction of his condition calls for. To attain success with this method we must be skilful, patient, and thorough in our work, and it is this very thoroughness that gives the greater

assurance of a perfect cure, a lessening of the period of convalescence, and the reconstruction of the parts without undue amount of scar tissue.

Preoperative care of the patient is the same as is deemed necessary to all major surgical operations, plus the fact that we believe it advisable whenever possible to have the field of operation specially prepared several hours in advance. The head should be shaved over an area approximately 1 inch above and 2 inches posterior to the auricle, and this area should then be scrubbed with green soap, using a stiff brush, washed with sterile water, then with alcohol or ether, and covered with a protective pad of moist bichloride gauze. The external auditory canal should be carefully syringed to remove all foreign matter and then closed with a pledget of cotton or gauze. These measures for cleansing the canal and the operative field should be repeated just before the operation is started. Preliminary to the operation the field may be painted with iodine as an extra antiseptic precaution, but such painting should not be used as a substitute for thorough mechanical cleansing, and if either one is to be done away with let it be the iodine.

Having secured a clean field upon which to work, every precaution must be taken to preserve it in that state. A sterile rubber cap, or sterile towels, can be bound around the head in such manner as to prevent any possibility of hair working into contact with the region of the wound. This is a point that can scarcely be too strongly emphasized, for we have not infrequently seen hair creep into the field and get pushed into the wound by the sponging assistant. With the hair properly held back, and the entire field protected by sterile towels, place over the whole a sterile sheet having a circular perforation just large enough to expose to clear view the auricle and mastoid region. This sheet should cover the head and shoulders of the patient and be fastened to the towels beneath so as to prevent slipping, and by carrying the distal side of the sheet over a suitable metal frame attached to the table it can be made to completely shut out the anesthetist and obviate any chance of his touching anything pertaining to the operation; it must be rendered impossible for anybody but the surgeon and his single assistant to come into contact with the operation field.

With reference to personal preparation of the surgeon and assistants, one must insist upon the strictest asepsis; this means thorough aseptic preparation of the hands and forearms by prolonged scrubbing and immersion in chemical antiseptic solutions, the use of sterile rubber gloves, and the wearing of sterile gowns, caps, and face-masks. Throughout the whole course of events in the operating room, the surgeon and his assistants should watch each other to see that no one becomes contaminated and carries that contamination carelessly to the patient. It is advisable to have but one assistant at the table and one clean nurse, both of whom should be as careful in their preparatory technic as the surgeon himself. The nurse should have charge of the instrument table and should see to it that every used instrument is washed, first in carbolic solution and then in sterile water, before being replaced on the instrument table or again presented to the operator.

Preparation of the instruments is in accordance with the rules followed in all first-class hospital operating rooms today. About the choice of instruments, a matter which is largely personal, we have nothing special to say except that we have come to earnestly advocate the Doyen burr or some similar bone drill, for perforating the mastoid cortex. Formerly using the

chisel solely, we never had any accident with it nor, so far as we know, did we ever cause any cerebral complication from concussion, but we became convinced that the burr has decided advantages; there is certainly with it no danger of producing concussion, there is far less danger of injuring the dura, and the necessary amount of cortex can be removed and the antrum exposed in less time and with greater safety than is possible with the chisel.

After etherization, which should be carried to full narcosis, the ear should be examined, and if the opening in the membrana tympani is small and apparently insufficient for drainage, it should be enlarged or a crescentic peripheral incision should be made to assure free escape of any fluids or secretions from the tympanic cavity. Carefully cleansing this part of the field and closing the canal with a fresh sterile plug, the auricle and mastoid region may be finally prepared for operative attack. Incision through the soft spots should be, preferably, curvilinear, extending from a point above the superior border of the concha to or below the tip of the mastoid. After the periosteum has been completely divided, either by the first incision or by repeated cuts, it may be retracted along with the soft tissues and held widely spread by a self-retaining speculum. Bleeding will, of course, be controlled by application of artery forceps as occasion requires. Careful drying of the exposed bony surface with sterile pledgets of gauze permits inspection of the structures to determine the location of the external auditory canal and the meatal spine, thus localizing the most direct approach to the antrum, and study of the mastoid surface for detection of any points of congestion or perforation resulting from disease. In a fair proportion of cases attention will be attracted to evidences of disease over the tip-cell or in the direction of the digastric fossa or of the sigmoid sinus, but even in the face of such perforations or apparent weak spots, we believe it is best to proceed direct to invasion of the antrum. The antrum is the one fixed central port of importance in every mastoidectomy and the sooner and more directly the surgeon enters it and uses it as the base from which to carry on further explorations the quicker and more satisfactorily will he conclude his operation.

The opening in the cortex, whether made with drill or chisel, must in all cases be large enough to permit free visual as well as tactile examination of the antrum and of all the cellular structure. Never be satisfied to probe around blindly in such a cavity; use all the illumination you can get and make the external opening large enough to afford direct and satisfactory vision. With spoon curets, plus the occasional aid of the chisel where the bone is very hard, the cellular bone can easily be broken down and removed together with all the débris of curettage or of disease. From the antrum work toward the tip-cell and when that has been exposed and investigated, as it should be in every case, the exploration may be carried in whatsoever direction and to whatever extent the conditions disclosed may demand. Complete obliteration of all evidences of pathological tissue must be the point aimed at—this is the most important point about this operation, and we believe that most of the failures of those who have attempted the blood-clot method have hinged upon failure to observe this rule. One may well ask, when is a mastoidectomy satisfactorily concluded? How much bone shall be removed and how can one determine when to stop? Our answer is that most men do too little rather than too much. Not only should all pus and débris of necrosis be removed, but every particle of visibly abnor-

mal bone must come away. The mastoid process should be cleaned out as completely as a peanut shell from which the nut has been taken. Perhaps the inner wall of such a shell shows infection; very well, continue to curet away that portion and it matters not if the dura, even over the sinus, be exposed. It is a good guide to continue curetting as long as there is spontaneous bleeding from the bone. Exposed dura above the antrum or over the sigmoid portion of the lateral sinus is no bar to later efforts at primary healing and one had far better have exposure of healthy dura in this way than to leave it covered by infected bone. In this connection we may express the opinion that sinus thrombosis secondary to mastoid operation is not the result of the operation itself, but arises because the primary operation was not sufficiently thorough. Practically all such cases of thrombosis make their appearance several days after the operation and have their origin in a condition that might have been obliterated by thorough curettage at the time of the mastoidectomy; infection spreads from a bit of undiscovered or overlooked diseased bone to the dural covering of the vessel, phlebitis is set up, and in due time thrombosis results. The operator is possibly to blame, but it is a sin of omission not of commission. Of course one would not needlessly expose the dura or sinus, but if there is any doubt about the character of the bone covering these structures the suspicious-looking bone should be removed. If you desire to obtain primary healing under blood-clot dressing, you must secure an absolutely clean wound cavity. A minute spot of infected bone or small infective granulations left *in situ* will soon infect the clot and cause it to break down, thus upsetting the effect of all the previous good work.

Shall this mechanical cleansing be followed by chemical sterilization of the cavity? We have not found it necessary and we believe that any marked efforts in that direction are prone to do more harm than good; most of the solutions that have been proposed for use in that manner have a tendency to prevent organization of the blood-clot. Washing of the cavity with sterile plain water or normal saline effects all the good that can be expected from any solution and does no harm. After such washing, followed by careful final inspection of the field, the cavity is permitted to fill up with blood, and usually there is quite sufficient of this obtainable by oozing from the soft tissues as the artery clamps are removed. Then the wound is to be closed. It probably matters very little what form of suturing is employed so long as strict asepsis is maintained. The Michel metal clamps have proved efficacious, if the rule be observed to remove them within forty-eight hours. Our preference has always been for the subcutaneous silver wire suture, entering at the upper end of the wound, zig-zagging from side to side, emerging at the lower end, and closing the gap by pulling like a drawing string. By this method we avoid the possibility of reinfecting the wound through piercing the skin with suture needles. Over the closed wound, care having been taken to nicely coapt the margins, we place a sterile silver-foil dressing and cover the whole with sterile gauze and bandage.

We are quite conscious of the fact that many operators have suggested modifications of this technic, but we are not aware of their having obtained any better results than we have published as a result of employing the simple method described. Through fear of absolutely closing the wound many have tried the partial closing—the insertion of wicks of gauze or of strands of suture material—or have recommended drainage for twenty-four hours

and then secondarily closing the wound. We see no reason for such make-shifts. Primary union of the surgical wound and speedy cure of the patient are possible in the majority of instances and we see no good reason why any competent surgeon should not accomplish satisfactory results with this method. There are reasons, we know, for some failures, but they are not *good* reasons, such as the operator would care to submit to a jury of surgeons.

In what percentage of cases may we expect primary healing to result? In our personal experience a record of 75 per cent. was made in dealing with cases of mastoiditis that had occurred in connection with acute suppurative otitis media. We believe, however, that the procedure would be justifiable if no more than 10 per cent. of such perfect results could be obtained, because when failure to heal at once results and the clot breaks down, no harm has been done and the ordinary long period of healing by granulation has not been at all prolonged by the *attempt* to shorten the duration of healing. Assuming that aseptic technic can be controlled and the skill and judgment of surgeons be always the same and always good, can we hope ever to secure 100 per cent. of primary unions by this method? We trust that day may arrive but we do not expect to witness it. There are not a few extraneous elements to be considered over which we have little or no control. Virulence of the attacking micro-organism and lack of resistance on the part of the patient are variable factors which may cause failure despite the most perfect work on the part of the surgeon. Again, having thoroughly eradicated the disease and overcome its toxemia, the patient's recuperative powers may have been so completely depleted by the illness that he cannot at once rally his forces for reconstruction work and, hence, fails in organization of the clot. Failure to succeed is not necessarily the fault of the operator; he can be criticized, however, for failing to try.

We have intimated that, in the main, success with this method depends upon strict asepsis and thoroughness of operative technic. There is not a surgeon in the world who will not subscribe to the propriety of applying those principles to mastoid surgery. But, while doing this theoretically, does the average surgeon, general or special, really apply them in daily work? We are inclined to question the belief that he does. We know, for it has been demonstrated now by many different surgeons, that success by this method is possible, and we believe that the chief reasons for failure on the part of others to adopt the method, or to succeed with it, is unwillingness to give the amount of time and attention required by its rather strict detailed technic, or inexcusable carelessness in the application of well recognized surgical principles. The majority would succeed if they performed their work properly; which means carefully, aseptically, and thoroughly. To them we might paraphrase the words of the old Cardinal who said, "Had I but served my God as faithfully as I have my King," and say, if you will but serve the surgical god of asepsis as faithfully as you labor to excuse yourselves for imperfect technic, you will secure more gratifying results with your mastoidectomies.

HENRY O. REIK.

## GENERAL CONSIDERATIONS REGARDING DIAGNOSIS OF INTRACRANIAL COMPLICATIONS FROM AURAL SUPPURATION

**Introduction.**—In this article the author has attempted to analyze (by the use of numbers and letters) the different pathological and chemical considerations, and to depict the mental processes which he has found of value in arriving at a differential diagnosis in suppurative lesions of the brain of otitic origin.

**Relative Frequency.**—Intracranial complications are much more frequently associated with purulent otitis than with nasal sinus suppuration, because the mucous membrane of the accessory sinus contains a protective mechanism which does not exist in the middle ear or mastoid cells.

Infection is constantly entering the nose, but is controlled by the nasal membrane, as this is its function; on the other hand, the middle ear and the mastoid cells are normally sterile, and infection which ascends through the eustachian tubes must be combated, not by the tissues themselves as in the nose and accessory nasal sinuses, but by phagocytes supplied from the blood-stream.

**Hospital and Staff Preparation.**—If there is to be adequate preparation for the highly technical diagnostic and surgical work frequently demanded by suppurative intracranial diseases, there should be established in every general and aural hospital a department of head surgery. This service should not be compelled to rely upon the general operating room, but should have at its command a special room, because an intracranial operation, such as for cerebellar abscess, may require that the patient be not removed from the table for several hours after evacuation.

The personnel of the cranial surgery department should consist of at least one surgeon, one assistant, one trained nurse, and a technician, while a bacteriologist and serologist should be in attendance. The latter should be accorded the privilege of visiting the patients, and empowered to conduct without delay such examinations as he may regard necessary, such as blood-cultures, and to advise the administration of transfusions, sera, etc.

**Diagnosis Based on Examination and Analysis of History.**—*Neurological and Physical Examination.*—Given a suspected intracranial complication it is of prime importance to determine the gross pathological process present. Is it a blood-stream infection (a sinus thrombophlebitis), a meningitis, or brain abscess?

This having been decided, the surgeon should then analyze the symptoms, trying, in a large way, to distinguish those due: (a) To *general toxemia*; (b) to *local toxemia*; (c) to *pressure*, and (d) to *death of tissue*.

For as no intradural operation should be purely explorative, and the operative procedure will largely depend on how the infection entered the skull, it is only by a combination of analysis of history and examinations that the probable path of advance of the lesion through the dura can be tentatively determined.

To accomplish this, a systematic neurological examination must be conducted by routine, and this by a member of the staff who is to perform the operative work. For, while every case should have the benefit of the opinion of a consulting neurologist, the routine neurological survey should not be delegated to an outsider, but should be made by one on whom the responsibility for the operation rests. This because neurologists as a class

are apt to acquire (by their training and constant treatment of functional and degenerative nervous lesions) an expectant point of view, in opposition to the surgeon's habitual mental reaction of, "Is this a septic surgical lesion? For, if so, it demands prompt interference."

*Importance in Diagnosis of a Complete Chronological History and a Knowledge of the Previous Operative Findings.*—The chief requisite to a positive diagnosis in any aural suppurative complication is a careful study of the history of the case from the inception of the infection in the middle ear. In this study the operative findings of any previous mastoid operation may be of the greatest assistance, as certain lesions in the mastoid are especially liable to give rise to specific intracranial affections. Consequently, it is desirable that during all mastoid operations pathological changes should be accurately and minutely recorded; which data should be carefully reviewed whenever a suspected complication develops.

Thus the finding during a *previous* operation of: (a) Involvement of the deep cells within the petrous pyramid; (b) a perisinuous abscess; (c) an extradural abscess over the tegmen or deep in on the posterior surface of the petrous pyramid, may be of diagnostic assistance.

*Diagnostic Suggestions from Lesions Found at a Previous Operation.*—(a) The finding of an extradural abscess during a previous operation, in a case which later presents vague cerebral symptoms, should awaken the suspicion either of a localized meningitis or brain abscess, as experience shows that an associated intradural suppuration is present in a considerable proportion of cases of extradural abscess. (b) The presence of an osteomyelitis involving the squamous portion of the temporal is always suggestive of an underlying localized meningitis. (c) Mastoiditis with labyrinthitis is a frequent forerunner of meningitis or cerebellar abscess; and (d) mastoiditis with labyrinthitis and an association of thrombophlebitis is especially apt to be followed by cerebellar abscess.

**Surgical Groups of Complications.**—Clinically there are two groups of cases of complication from aural suppuration which present diagnostic difficulties: (1) Cases in which cerebral symptoms are present prior to an operation on the mastoid, and (2) those in which these symptoms develop or are only recognized subsequently to an operation upon the mastoid.

*Temporary Disappearance of Symptoms after Mastoidectomy.*—In cases of an intradural affection which is present prior to an operation upon the mastoid, it is a noteworthy fact that immediately following the operation the cerebral symptoms are apt to temporarily subside; this because the activity of the intracranial process is apparently checked by the elimination of the primary focus of infection. During the period of apparent quiescence time is allowed for the readjustment of the brain's protective reactions.

**Visualization of Pathological Sequences.**—In diagnosing a complication of aural suppuration it is useful to visualize the pathological sequences in the order in which infection usually invades the different tissues.

Infection spreads into the dura either (1) by *direct extension of suppuration*; more frequently (2) by *retrograde thrombophlebitis*, and lastly (3) by the *perivascular spaces of the cerebrospinal system*.

An otitis media may cause, first, an osteitis—congestion, infiltration, caries, and granulation (the usual mastoid disease)—which of necessity is accompanied by a thrombosis of the small veins of the bone, many of

which pass from the middle ear into the large venous sinuses of the cranium. This osteothrombosis is frequently a reactive process, the thrombus forming to obstruct the vein; in which case there may be no infection within the clot itself. However, when the infective process is not limited to the bone there develops either (a) a perisinuous abscess or (b) a thrombophlebitis of one of the large sinuses; the phlebitis occurring either independently of, or largely as the result of, the abscess.

Instead of attacking a large venous trunk (generally the descending portions of the lateral sinus), a retrograde thrombophlebitis of a small venous radicle may carry the infective process from the bone into (c) the *meninges*; in which case there is developed at first a *localized meningitis*, which may remain confined to the original area, or may spread throughout all the cerebrospinal fluid spaces, or a *general septic leptomeningitis*.

Again, the retrograde thrombophlebitis of a small vein may pass through the meninges and enter the brain tissue itself, giving rise to (d) a brain abscess by causing nutritional death of the cerebral tissue.

**Interrelationship of All Infective Cerebral Complications.**—Consequently, there is an interrelationship of the three chief complications—sinus-phlebitis, meningitis, and brain abscess—for usually *underlying all intracranial suppurative affections of aural origin is a thrombophlebitis of a small or a large venous radicle*. However, at any stage the infection may advance by a direct extension of the suppuration, as when the sinus wall is perforated, or the dura ulcerates.

**General Diagnosis of an Intracranial Complication.**—In the presence of suppuration in the ear, the development of cerebral or toxic symptoms, such as headache, vomiting, chills or irregular temperature, is presumptive evidence that an intracranial complication has arisen. Too much importance should not be attached to any one symptom, but the clinical picture as a whole should be considered; as in the early stages of intracranial suppuration all cerebral symptoms may be very vague.

**Importance of Known Focus of Infection.**—Evidence of a present or recently suppurating ear is of great assistance, for, although otitic adjacent brain abscess and localized septic meningitis may be present without aural discharge, they are of such rare occurrence that a normal drum-membrane with normal hearing practically eliminates a complicating cerebral suppuration. In capsulated bacillus cases (*Streptococcus mucosus*, pneumococcus, Friedländer's bacillus), while discharge may be absent and the drum-membrane only slightly thickened and lusterless, there is always considerable deafness, a "full feeling" in the ear, associated with tinnitus.

**Diagnostic Importance of an Acute Exacerbation in Chronic Otitis.**—An intradural complication from a chronic otitic suppuration originates only during an acute exacerbation. This is especially important in bilateral chronic suppuration, as the offending ear can usually be distinguished by having been the site of pain at the beginning or just preceding the symptoms of the cerebral complication, although this pain may have been slight and of transient duration.

**Diagnostic Value of Amount of Aural Discharge.**—The amount of aural discharge is an indication of surgical importance. (a) A discharge greater than can be held in the middle ear is diagnostic of an involvement of the bone. (b) The cessation of a chronic discharge with the continuation of pain is indicative either of a closure of the perforation in the drum or an

intracerebral complication. In capsulated bacillus cases there is frequently little or no discharge.

**Symptoms Common to All Suppurative Complications.**—1. *Pain*—*differentiation between that of middle ear, mastoid, and cerebral affections:* The pain accompanying suppuration of the middle ear is deep in the ear; when the mastoid is involved, the pain may be behind the ear. In a cerebral affection the pain is in the head itself and is apt to be much more severe during the night. During the early stages of the process the pain is usually situated in the temporal region directly above the ear, and is caused by irritation of the dura.

When the patient has previously been subject to cephalalgia the pain of the intracranial complication is frequently attributed to this predisposition; but careful questioning will always demonstrate that it is of a somewhat different type from that previously complained of.

2. *General malaise* is frequently the only outstanding symptom during the early period of suppuration within the cranium.

3. Any *vomiting* should be regarded with the gravest suspicion in the presence of an aural suppuration. It is seldom of the projectile type; and it is apt to be attributed to an indiscretion of diet.

4. *Disproportion*, viz., severity of pain or an elevation of temperature out of proportion to the local suppurative condition present, should play a large part in diagnosis.

A combination of severe headache, vomiting, and general malaise not warranted by the manifest process in the ear or mastoid is an indication of intracranial involvement, especially if preceded by a vague chill and associated with an increased cell count of the fluid from the lumbar region.

5. *Chill*.—The chills of blood-stream infection are generally severe and outstanding. The single "initial rigor" of brain abscess is frequently so "vague" that it is often regarded by the patient as "a nervous chill," and is apt to be forgotten, its presence being only elicited on careful inquiry.

6. *Temperature*.—In thrombophlebitis the temperature is apt to be remittent; in septic meningitis, continuously high; and in brain abscess, slightly elevated, with irregular remissions below normal.

**Positive Signs of the Existence of a Suppurative Complication.**—

1. *Protective Meningitis*.—(a) A high cell count in the lumbar fluid is a definite indication of meningitic irritation and signifies either sinus thrombophlebitis, local meningitis, or brain abscess. The character of the cells is of importance. In a general way a high percentage of polynuclears indicates an active process, while a large proportion of mononuclears suggests regression.

(b) Increased pressure of the cerebrospinal fluid is indicative of irritation of the cerebrospinal fluid system. However, during the development of brain abscess (if it is localized entirely within the cerebral tissue), there may not be a high cell count or an increased pressure of fluid in the lumbar region because the meningeal system is only locally involved.

(c) Disappearance of Fehling's reaction (sugar content) in the fluid is indicative of suppurative disease, the bacteria probably assimilating the normal small amount of glucose from the fluid.

(d) Diminution of the normal chloride content is suggestive of the breaking down of the barrier between the blood and the cerebrospinal

fluid systems. Greenfield<sup>1</sup> thinks that, in suppurative meningitis, a fall below the normal content of 680 mg. is of grave prognostic import, while a continuation of the normal content (725 to 750 mg.) above that of the blood's is of good augury, demonstrating that the hemolytic cerebrospinal barrier is still functioning.

**ACCIDENTS FROM LUMBAR PUNCTURE.**—Spinal puncture for diagnosis in meningitis and degenerative lesions may be performed with safety, but lumbar puncture in cases of cerebral displacement (from localized suppurative diseases of the brain) or in blood-stream infection is attended with some danger.

The pons and medulla, normally containing the vital centers, are held almost immovable by the vessels and nerves in their exit from the posterior fossa. In abscess of the cerebellum the brain stem may be displaced beyond the median line, while the blood-vessels are stretched. This stretching is tolerated because the displacement is gradual. However, if the cerebrospinal fluid is suddenly withdrawn, the displaced blood-vessels are apt to rupture, especially those of the pons.

Small pontine hemorrhages may not give rise to sudden death (as when the medulla is forced downward through the foramen magnum), but may occasion Cheyne-Stokes respiration for a considerable period.

2. *Convulsion.*—A convulsion during suppuration of the ear is a positive sign of intracranial involvement. At the beginning a convulsion may only signify an aseptic meningitis.

Convulsions are much more common in infants than in children or adults, in whom they take the place of chills.

In brain abscess, convulsions are much more apt to occur when associated with a temporosphenoidal lobe lesion, probably because of disturbances of the circulation of the middle central area. Convulsions subsequent to evacuation of an abscess or in the course of meningitis are of cortical origin.

3. *Positive Blood-culture.*—A positive blood-culture is pathognomonic evidence of blood-stream infection, but there may be repeated negative blood-cultures in spite of an extensive subacute thrombophlebitis of a large venous radicle.

#### SINUS THROMBOPHLEBITIS

**Preoperative Visualization of the Septic Process in Chronic Sinus Thrombophlebitis.**—In the protracted types of lateral sinus thrombophlebitis—which frequently extend over several weeks—it is often possible to visualize preoperatively the pathological process, providing there are available (a) a detailed history of the case prior to the original mastoid operation, (b) the pathological findings of the operation, and (c) a trained nurse's postoperative temperature charts and clinical notes.

The record of a chill (frequently mistaken as due to mastoid suppuration or sapremia from retention) registers the date of the entrance of the infection; the operative finding of a small discolored area of granulation in a particular part of the sinus marks the point of entrance; while the postoperative record of low sepsis (chills and irregular pyemic temperature) tells of the extension of the clot and its breaking down into purulent areas.

<sup>1</sup> Greenfield, J. G.: Discussion on Non-tuberculous Meningitis, Proc. Roy. Soc. Med., 18: 10, February, 1925.

**Frequency.**—Sinus thrombophlebitis is the most frequent of the aural complications. It is of such common occurrence that a few surgeons advocate the exposure and even the incision of the sigmoid portion of the lateral sinus during all mastoid operations in which the temperature elevation is unusually high or there are symptoms of general toxemia. Many aural surgeons regard one chill as justifying sinus puncture or aspiration.

**Surgical Pathology.**—Two processes must be recognized: 1. An *acute septic phlebitis*, characterized by an inflammation of the wall of a large cerebral sinus, the infection originating from an adjacent area in the mastoid or middle ear and giving rise to general toxemia. The phlebitis is not sharply limited; it may travel in the wall of the vein without causing the formation of a clot. Phlebitis is accompanied by high temperature, which but seldom has remissions to normal. Blood-culture reveals micro-organisms in a considerable proportion of the cases.

In this type of acute toxemia the surgical indications are to remove thoroughly the primary focus of infection and to limit the spread of the infection in the vein itself.

2. In chronic thrombophlebitis the septic process is more or less localized. The clot formation may be very largely protective, with areas of suppuration in it. Blood-cultures are apt to be negative; there are sharp elevations of temperature associated with chills, followed by sweats and remissions to normal (pyemia). The presence of remissions of temperature is a favorable prognostic sign, indicative of a tendency toward the localization of the septic process and clot formation. An elevated temperature with a feeling of well-being signifies a good reaction of the patient.

**Surgical Indications.**—The surgical indications in chronic thrombophlebitis are to drain the sinus, removing the protecting clot with as little trauma as possible, and thus to obtain free bleeding from both ends (posteriorly toward the torcular and inferiorly toward the jugular bulb). When bleeding cannot be induced from below, it is evidence of obliteration of the jugular bulb and, possibly, of the internal jugular vein. Between the extremes of infection all grades of virulence occur.

**Invasion.**—The possibility of involvement of the sinus may present itself to the surgeon either (1) at the time of an acute primary mastoid operation, (2) a few days or weeks following the operation, or (3) in the course of a chronic suppurative otitis.

*Clinical Types of Invasion.*—(1) When the sinus is attacked during the early stage of an acute otitis and prior to operation upon the mastoid, the temperature is persistently high and there is apt to be one or more severe chills which cannot be distinguished clinically from the rigors which now and then accompany an acute suppurative otitis.

The sinus on exposure may not contain a clot—the process being chiefly a phlebitis. The early diagnosis must rest on (a) a disproportion between the temperature and the otitis present, (b) an unusually high leukocyte count, and (c) the severity of the toxemia.

(2) When thrombophlebitis occurs a few days or weeks after a mastoid operation, it is generally ushered in by a chill and a sudden elevation of temperature following a period of irregular, slightly elevated temperature. The patient during the quiescent period may have been regarded as doing well, although complaining irregularly of vague pain in the region of the ear, general weakness, and tendency to sweating at night. It is during

the apparently quiescent period that a clot is formed. It is believed that the discharge of a septic nidus into the circulation is the cause of the chill.

(3) Sinus thrombophlebitis complicating chronic purulent otitis occurs only during an acute exacerbation of the chronic suppuration, although the exacerbation may be so insignificant as to be forgotten by the patient; consequently, there can always be obtained a history of slight pain in the ear and possibly sudden cessation or augmentation of the aural discharge, followed by elevation of temperature, which may be accompanied by chills and remissions; the latter depending upon whether the process is phlebitic or thrombophlebitic.

#### DIAGNOSTIC IMPORTANCE OF KNOWN FOCUS OF INFECTION

In the presence of a suspected intracranial suppuration and a known focus of infection, no other possibility should distract the surgeon's attention from the ear as the probable cause of the cerebral symptoms—this, of course, in the absence of positive evidence of another disease, such as rapid respiration or physical signs in the chest, etc.

For while an independent infection, such as erysipelas or pneumonia, frequently develops in the course of chronic suppurative otitis, the slightest acute exacerbation of the chronic process—as transient earache—points to the aural suppuration as the origin of the complication, although the otalgia may have been so slight as to be disregarded by the patient.

**General Blood-stream Infection.**—With a suspected cerebral lesion the presence or absence of a blood-stream infection should be determined at as early a date as possible, because in the early stage of the disease such information enables a differential diagnosis between sinus thrombophlebitis, meningitis, and suppuration of the cerebral tissue itself. Later, however, differentiation may be impossible because two or all three lesions may co-exist.

A blood-culture should be made daily as long as a complication is suspected, for in the chronic types of thrombophlebitis even repeated negative cultures are the rule, although a septic clot may be present and extend a considerable distance.

**Importance of Positive Blood-cultures as a Localizing Symptom of Involvement of a Large Venous Sinus.**—One positive blood-culture is a definite indication for surgical interference.

Thrombophlebitis of small venous radicles within the cranium is not associated with positive blood-cultures; and, although cases of positive streptococcic blood-cultures from thrombophlebitis of small venous radicles in other parts of the body are reported, in infections from the ear a positive blood-culture should be regarded as diagnostic of phlebitis of a large venous sinus.

**Differential Cell Count.**—Similarly, a differential leukocyte count should be performed daily, as it is an indication of the degree of the infection and of the resistance of the patient.

**Importance of Bacterial Examination of Secretion from Mastoid.**—In certain instances of double otitis media in which both mastoids had been opened, diagnosis of the side of the involved sinus was later made by obtaining a cultural growth of the same micro-organisms from the blood which had previously been isolated from but one of the mastoid cavities, that of the opposite side having contained a different micro-organism.

**A. Symptoms of General Intoxication.**—(a) *Temperature.*—Preconceived ideas as to the necessity for chills and sudden elevations of temperature followed by perspiration and remission—symptoms frequently present in lateral sinus thrombosis—must be abandoned in view of the present knowledge regarding intoxication and bacteremia, as there is no hard-and-fast line between sapremia, septicemia, and pyemia, each being a manifestation of general sepsis.

(b) *Patient's Appearance and Resistance in Blood-stream Infection.*—Until the terminal stage of blood-stream infection the patient does not look sick, although recording high temperatures with or without rapid excursions. He is apt to present an appearance of well-being—apparently too well in proportion to the temperature recorded. This “favorable impression” is a good prognostic sign. As long as the pulse-rate is proportional to the temperature, the prognosis is favorable. An ascending disproportionate pulse is often the earliest sign of complicating meningitis.

(c) *Headache*, except the usual “confused feeling” of high temperature, is not present when the infection is confined to the sinus itself. Headache severe enough to be complained of signifies meningeal irritation from the phlebitis or meningitis.

(d) *Delirium.*—As long as the infection is limited to the sinus the sensorium is clear. The appearance of delirium is of the gravest significance, for, while muttering during sleep occurs in cases of lateral sinus thrombosis from the elevated temperature, true delirium is indicative of a meningeal involvement—the usual terminal process.

However, the meningitis need not necessarily be either diffuse or suppurative, but may be protective in type; in which case, if the existing cause of the meningeal irritation be removed, the inflammation within the dura may subside.

(e) *Albumin in urine* from a mild grade of acute parenchymatous nephritis is the rule in blood-stream infections.

*Effect of Ether Administration in Sinus Thrombophlebitis.*—Patients with pyogenic blood-stream infections stand ether anesthesia remarkably well, at least until the time of myocardial degeneration. The usual bacteremia apparently causes changes within the blood which give the individual greater resistance as far as surgical manipulation is concerned. As a class they are good surgical risks, although suffering from severe toxemia.

**B. Local Manifestations of Sinus Involvement.**—(a) *Griesinger's sign*—pain behind the mastoid—is generally significant of perisinuous abscess, but occasionally is due to phlebitis of the mastoid emissary vein.

(b) *Infiltration of the jugular vein* is impossible to demonstrate clinically; the “core-like feel” of the vein which has been described is uniformly a chain of enlarged glands. It consequently can be of but slight diagnostic value, as in acute otitis the lymphatics accompanying the jugular are always more or less involved in Nature's effort to limit the infection.

(c) *Abscess of the Soft Parts, in the Neighborhood of a Venous Radicle, Which Terminates in a Basal Sinus.*—In the presence of a positive blood-culture, a local abscess of the soft parts (if situated in an area drained by one of the large venous sinuses of the head, neck, occiput, pharynx, or orbit) should suggest the possibility of the abscess being secondary to a sinus thrombophlebitis, the result of a direct extension of the infected clot from the infected large sinuses. The position of the abscess may locate the

particular venous trunk affected. Consequently a *neighborhood abscess* should not be regarded as the primary source of the blood-stream infection, or dismissed as of metastatic origin. A deep abscess of the occipital region may originate from a lateral sinus thrombophlebitis, or a retropharyngeal abscess from an infected cavernous sinus.

**C. Symptoms of Venous Stasis.**—(a) *Papilledema* is more frequent with sinus thrombophlebitis than with either cerebellar or temporosphenoidal lobe abscess. As a rule it is of a mild degree and without hemorrhages. Theoretically, blurring of the disk margins and dilatation of the ophthalmic veins should accompany the formation of a clot by a thrombophlebitis of the lateral sinuses and, exceptionally, a high degree of papilledema with dilated tortuous veins and hemorrhages has followed the *sudden* obliteration of a long venous tract, such as the longitudinal sinus, following the ligation of the main jugular and facial veins.<sup>1</sup>

But in the vast majority of cases of sinus thrombophlebitis the venous obliteration is so slow that the return venous flow from the head is not appreciably disturbed; this because other venous channels promptly establish compensation.

The mild papilledema so frequently seen in lateral sinus thrombophlebitis is usually a manifestation of a protective meningitis, which later may accompany disturbances in the communications between the venous return and the cerebrospinal fluid system into which it empties. The protective meningitis is probably largely the result of an inflammation of the intima of the mesial wall of the sinus.

(b) *Slight transient dilatation of the retinal veins* may be an early manifestation of involvement of the cavernous sinus by the thrombophlebitis.

(c) *Crowe-Beck sign*—dilatation of the retinal veins on pressure of the unaffected jugular (in the carotid triangle), thus obstructing the remaining large venous channel from the head—requires a great deal of practice and patience. It is but exceptionally applicable, because it necessitates an acute obstruction of a large sinus and this in the early stage of the thrombus formation before the collateral circulation by way of vertebrals, condyloids, and other veins has been established.

#### **D. Operative Findings Suggestive of Venous Stasis in the Lateral Sinus.**—

(a) *Excessive hemorrhage* from the operative area, the blood filling the diploic spaces of the bone in its effort to leave the skull, or (b) *dilatation of the mastoid accessory* from the same cause, both suggest that the sinus is not pervious. Exceptionally, the uncovering of (c) a *thrombosed mastoid emissary* will demonstrate that the sinus itself is obliterated. (d) *The loss of the normal bluish luster* of the sinus, and the slightly sluggish return of contour after indentation by a probe, may permit of the diagnosis of an obstructing clot. (e) *Perisinuous abscess or thickening of the sinus wall* by granulations at the point of entrance of the infection is frequent in cases following chronic aural suppuration. (f) *The collapse of the sinus* from aspiration of blood into the chest during inspiration shows that it is pervious. This sign is especially valuable subsequent to an operation when the question of the possibility of an occlusion of the sinus by a septic clot has arisen. In these cases, if the sinus is well exposed, the placing of the

<sup>1</sup> Eagleton, Wells P.: Circulatory Disturbances Following Ligation of the Internal Jugular Vein in Sinus Thrombosis, Arch. Otol., 1906, vol. xxv, No. 2, p. 91, Brain Abscess; Case XXIII, p. 129.

patient in the sitting position, and requesting him to take a long breath, may graphically demonstrate the sinus permeability by its collapse during each aspiration.

**E. Symptoms Suggestive of the Area of the Sinus Involved.**—(a) *Pain behind the eye and in the teeth* in the presence of a chronic blood-stream infection suggests that the venous obliteration has extended through the petrosals and involved the cavernous sinus. The fifth nerve pain is apt to be associated with (b) *external ophthalmoplegia* from involvement by edema and round-cell infiltration of the homolateral sixth, third, and fourth nerves in their course through the cavernous sinus.

(b) *A primary jugular bulb involvement*, although secondary to an otitis, may occur; the infection going through the floor of the middle ear. It occurs almost exclusively in infants, because of the presence of dehiscences in the bone. There is no way of positively diagnosing the condition symptomatologically except by inference.

(c) *Jugular bulb or Avellis' syndrome*—anesthesia of the pharynx and the posterior one-third of the tongue, from pressure on the ninth; with paralysis of the laryngeal muscles, from involvement of the tenth; and prominence of the median border of the scapula from paresis of the trapezius, by pressure on the eleventh—has been recorded in chronic jugular bulb thrombophlebitis.

(d) *Recent manometric measurement*—Ayer-Tobey sign—of the cerebrospinal fluid pressure in the lumbar region has been utilized. Pressure on the jugular of the unaffected side (in the carotid triangle) may show the existence of an obliterating clot by a sharp rise in the height of the fluid; the release of the pressure on the jugular being followed by an immediate fall to the original manometric reading.<sup>1</sup> It is most graphically present in the early stages of sinus obstruction, before venous compensation has become fully established.

**Symptoms of Extension of the Phlebitis.**—These are generally confined to (a) *a continuation of the temperature*, irregular chills, and the appearance of (b) *metastatic abscesses*, or (c) *infective arthritis* in different joints. The sternoclavicular articulation is most frequently attacked in lateral sinus thrombophlebitis. An isolated swelling of the sternoclavicular joint (toxic arthritis) calls for an examination of the ear, as it is seldom involved in any other pathological condition. The sternoclavicular joint rarely suppurates, but the hip-joint frequently does, in which case spontaneous dislocation may occur if the purulent nature of the arthritis is not earlier treated. The knee and the shoulder are also frequently involved.

**Differential Diagnosis Between Sinus Phlebitis and Meningitis.**—With continuously high temperature and headache, especially if irregular attacks of delirium are present, meningitis is to be suspected. It may be excluded relatively by examination of the cerebrospinal fluid, as meningitis secondary to sinus phlebitis is apt to be confined to the basal cisterna for some time before becoming general; during this period the cerebrospinal fluid from the lumbar region may or may not give evidence of the invasion of the leptomeninges, depending on the extent that the protective process has been able to localize the meningeal infections.

<sup>1</sup> Tobey, G. L., and Ayer, J. B., *Dynamic Studies of Cerebrospinal Fluid in the Differential Diagnosis of Lateral Sinus Thrombosis*, Arch. of Otol., vol. 2, No. 1, p. 50, July, 1925.

### DEEP EXTRADURAL ABSCESS WITH MENINGEAL INVOLVEMENT

*Deep extradural abscess*—median to the plane of the inner wall of the antrum—with meningeal irritation or involvement is associated with fever, pain deep in head (usually localized above the ear), low cell count in the cerebrospinal fluid, and at times with paralysis of the external rectus of the homolateral side (Gradenigo's syndrome). The sixth nerve paralysis is supposed to result from (a) a serous or protective meningitis attacking the basal cisterna (cisterna intrapeduncularis) of which the sixth nerve traverses, (b) but anatomico-developmental studies show that the nerve is generally paralyzed from compression of the nerve by swelling of the periosteal aponeurotic dural constituents as it passes through Donellos' canal, or (c) under the petrosphenoidal ligament at the apex. The pathological lesion of the bone may be congestion, inflammation, or caries of the apical cells of the petrous pyramid. Deep extradural abscess may be accompanied by a mild papilledema, which is due to the serous meningitis when it has subsided after the evacuation of the abscess. (*Suppurative inflammation of the petrous apex with localized pontine cistern meningitis.*)

Gradenigo's syndrome, if accompanied by signs of sepsis, however slight, should always awaken suspicion that the meninges are involved, as nearly one-fifth of the cases diagnosed as Gradenigo's syndrome have ended fatally from meningitis or brain abscess.

Pain in the fifth nerve region, even when accompanied by abducens paralysis, generally subsides after removal of the infection of the mastoid, but *first branch pain* (*pain behind the eye*), associated with a low-grade of sepsis which continues or appears after the mastoid perilabyrinth cells have been completely extenuated, is diagnostic of a localized meningitis of the pontine cistern the result of an inflammation of the cells at the apex of the petrous pyramid.

*Roentgen-ray examination* may be of some differential value in deciding whether Gradenigo's syndrome requires a direct surgical attack or not; as infection in the presence of a pneumatic petrous may give rise to abductor paralysis from congestion of the apical cells (which congestion will be relieved by drainage of the mastoid cells, with which the apical cells are continuous), while a diploic type of mastoid suggests an intracranial or suppurative apical process. The latter is especially apt to be present if the pain behind the eye and the sixth nerve paralysis are associated with signs of sepsis (no matter how slight), and appear subsequent to a well-conducted mastoid operation.

### TEMPOROSPHENOIDAL LOBE ABSCESS

During the early stage of brain abscess the clinical manifestations are frequently so vague as not to warrant a positive diagnosis of intracranial suppuration, much less its definite localization.

**Operative Findings Suggestive of the Presence of Brain Abscess.**—Brain abscess, being a circumscribed inflammation, results much more frequently from a chronic than from an acute otitis. During a radical operation the exposure of granulations on the dura, or an extradural abscess directly above the tegmen, suggests the possibility of an associated abscess of the middle fossa. Very exceptionally, a teat-like protrusion of dura, from which a drop of pus exudes (the remains of the causative thrombophlebitis), furnishes positive evidence of an adjacent temporosphenoidal abscess.

**Diagnosis of the Presence of Brain Abscess.**—Of paramount importance is a detailed chronological history of the case, which in the early stages is apt to be indefinite and confused. A proper history can only be obtained by the surgeon enlisting the co-operation of the patient, and of such of his associates as may have been with him from the first appearance of the suspected complication. Only by careful questioning, with a calendar in hand (so that each day can be accounted for, its happenings reviewed), can exact time of the appearance of each symptom be ascertained, and a proper history be elicited.

By such a method it is generally possible to determine, first, *whether cerebral suppuration is present*; and second, *its probable duration*. A chronological history associated with a complete neurological examination (see Appendix) allows of an analysis of the symptoms into those (1) from cerebral suppuration and (2) from cerebral pressure; during this analysis symptoms allowing of (3) a localizing diagnosis will usually present itself. Clinically, adjacent abscess of the brain can be divided into three stages:

**Clinical Stages of Brain Abscess.**—(1) *Acute stage without capsule formation*, with progressing symptoms—"manifest stage." (2) A more or less "*quiescent stage*" following capsule formation, which, if it becomes complete, may be followed by a period of disappearance of all symptoms; (3) "*latent stage*."

Cases in the latter stages are but rarely encountered, as the vast majority of patients succumb during the period of capsule formation when the suppuration is not completely walled off from the surrounding cerebral tissues.

*Acute brain abscess* is characterized by (a) death of cerebral tissue with (b) an associated surrounding edema and (c) an increase of the cerebrospinal fluid; all three processes contributing to the clinical manifestations of cerebral suppuration.

#### SYMPTOMS OF BRAIN ABSCESS

(A) **Manifestations of Cerebral Suppuration.**—(1) An initial "*vague chill*" usually ushers in the cerebral suppuration, because adjacent brain abscess of otitic origin most frequently results from a retrograde thrombophlebitis of a small cerebral vein. In the much rarer abscess from direct extension of the suppuration through the dura, the initial slight rigor may be absent.

(2) *Vomiting* and *headache* are the two most constant symptoms of cerebral disturbance. Usually both have little or no localizing value.

The *vomiting* may be projectile, but is more frequently regurgitant. It is generally attributed by the patient to indiscretions in diet.

(3) *Headache* during the acute stage is apt to be severe and limited to the side of the head affected. If the abscess becomes quiescent, the severity of the headache may largely subside; in which case, especially in brain abscess the result of a chronic otitis, the remaining headache, like the vomiting, may be attributed by the patient to a digestive disturbance. This is especially apt to be the case, as the headache is uniformly relieved by dehydration with magnesium sulphate, the result of a reduction of the increased intracranial pressure. Careful questioning, however, will demonstrate that the type of pain is somewhat different from "sick headaches" to which the patient "was previously disposed."

(4) There may be a *characteristic appearance of the patient* which is suggestive in the same way that the "drawn, anxious look" is diagnostic of an acute abdomen. The facies of a patient with acute cerebral suppuration is the result of a combination of apathetic depression with toxemia. It resembles the physical, mental, and psychic state which follows a prolonged debauch. The general malaise, loss of appetite, coated tongue, and feeling of ill-being are the result of chronic sepsis; accompanied by a disproportional degree of dry skin, anxious expression, sordes covered teeth, and general carelessness of appearance. These symptoms, in the presence of a slightly elevated temperature, alternating with intervals of subnormality, headache, and irregular vomiting, furnish the clinical picture of cerebral suppuration in the early "manifest" stage, before firm encapsulation has completely walled off the suppuration. The characteristic appearance when associated with a high cell count and increased globulin, without micro-organisms in the cerebrospinal fluid, allows of no other interpretation.

(5) *Slight dizziness* is the rule, but it lacks the severity or persistency and has not the qualities characteristic of vestibular vertigo. No turning sensation is described by the patient.

(6) *Subnormal temperature*, interspersed with slight elevations, is irregularly present; the rectum (the only reliable temperature), usually recording around 100° F. with occasional drops to 97° F., or slightly lower.

(B) **Compression.**—The symptoms of cerebral compression should be differentiated from those of cerebral suppuration. The extreme intracranial pressure which so frequently accompanies brain tumor is never present in suppurative lesions, as in abscess the patient succumbs from bulbar paralysis long before an excessive intracranial pressure develops. Even a moderately high degree of compression is only present in the terminal stage of brain abscess. However, slow pulse, slight papilledema, and varying degrees of stupor are frequent symptoms. A papilledema sufficient to cause reduction of vision signifies an additional factor, such as acute venous stasis. Greatly increased intracranial pressure is a definite indication for surgical intervention as, uncontrolled, it as a rule causes a sudden fatal termination.

(C) **Localization of Adjacent Temporosphenoidal Lobe Abscess.**—Adjacent abscess of the temporosphenoidal lobe without cortical involvement (the most frequent variety of brain abscess) is usually situated from  $\frac{1}{2}$  to  $1\frac{1}{2}$  inches above the tegmen, in or near the second temporosphenoidal convolution, which, being a so-called "silent area," fails to produce outstanding localizing symptoms, especially when the abscess is on the right side. However, the diagnosis of a probable adjacent temporosphenoidal lobe abscess can usually be made from (a) the presence of an adjacent suppuration within the mastoid, (b) the general symptomatology of intracranial suppuration, and (c) the probable exclusion of the cerebellum as the site of the abscess.

The diagnosis becomes positive when manifestations of central suppuration or compression are associated with:

(D) **Pathognomonic symptoms of temporal lobe involvement**, viz.: (a) nominal or "naming" aphasia; (b) hemianopic indentation of the visual field; (c) contralateral paresis or paralysis; (d) absence of the temporal horn of lateral ventricle as demonstrated by ventricular puncture or ventriculograph, or (e) Roentgen evidence of air in the abscess (from gas-producing

micro-organisms); and (f) a differential diagnosis by the probable exclusion of the cerebellum as the site of the suppuration.

(a) *Aphasia*.—Marie and Head<sup>1</sup> contend that all attempts to explain aphasia as an interference with specific areas and tracts are to be abandoned, and each case of aphasia must become a study by itself. This is especially true in temporosphenoidal lobe abscess.

Life's greatest accomplishment—speech—was not acquired by the simple addition of certain cells in the cortex of man of specific areas, as formerly postulated, although many architectural changes occur.

Speech is the supreme achievement of the association of ganglionic tissue, the most recent parts of the associations occurring in the neighborhood of the suprameatal osseous sutures, the complete bony union of which is delayed until old age in man, thus allowing the longest period of development unhampered by bony union.<sup>2</sup>

Head<sup>1</sup> has called attention to the fact that naming aphasias, like all other forms of aphasia, are alike, and that the area of the cortex involved in the development of a naming aphasia is in relation with the squamous suture, which does not completely unite until extreme old age. Thus man's speech area has been permitted development and growth unhampered by compression from an early closure of the overlying suture.

The suprameatal sutures can thus be used as a surgical landmark in cases of aphasia, for from the pterion in front (which is directly over the inferior extremity of the lateral central fissure—the latter dividing the inferior frontal gyrus from the superior and middle temporal gyri covering the island of Reil—the so-called Broca's area) to the parietosquamomasto-occipital junction—the asterion—behind, they lie over the areas of associations in which speech developed; the region directly above the tegmen and internal to the suprameatal suture being the most frequent site of temporosphenoidal abscess.<sup>3</sup>

*Subcortical and Cortical Lesions in Aphasia*.—The majority of temporosphenoidal abscesses are situated in the white substance of the temporal lobe, directly above the tegmen, but even when a patient is right-handed, with the abscess on the left side and the pathognomonic nominal aphasia present, the diagnosis of the exact location of a temporal lobe brain abscess, whether cortical or subcortical, is always more or less provisional. The aphasia, although usually subcortical, may be of cortical origin from pressure of a localized pia-arachnoid abscess.

In the pathognomonic naming aphasia the speech disturbance is frequently unrecognized by either the patient or his friends, or if noticed, is apt to be attributed by them to a transient loss of memory because of his headache; this because the patient, while apparently speaking fluently and precisely, only fails now and then when attempting to find the proper substantive necessary to name an object that he may desire in connection with his wants. Moreover, the patient soon learns to get around the defect to hide, as it were, by substituting the verb denoting the action of the article for the name of the article itself, thus hiding his nominal naming gaps by employing his unaffected verbal ability.

<sup>1</sup> Head, Henry: "Aphasia and Kindred Disorders of Speech," Macmillan Co., 1927.

<sup>2</sup> Harris, H. O.: Closure of the Cranial Sutures in Relation to the Evolution of the Central Cortex and Aphasia, Bull. of Mayo Clinic, August 31, 1927.

<sup>3</sup> Smith, Elliot: "Evolution of Man," Oxford Press, 1927.

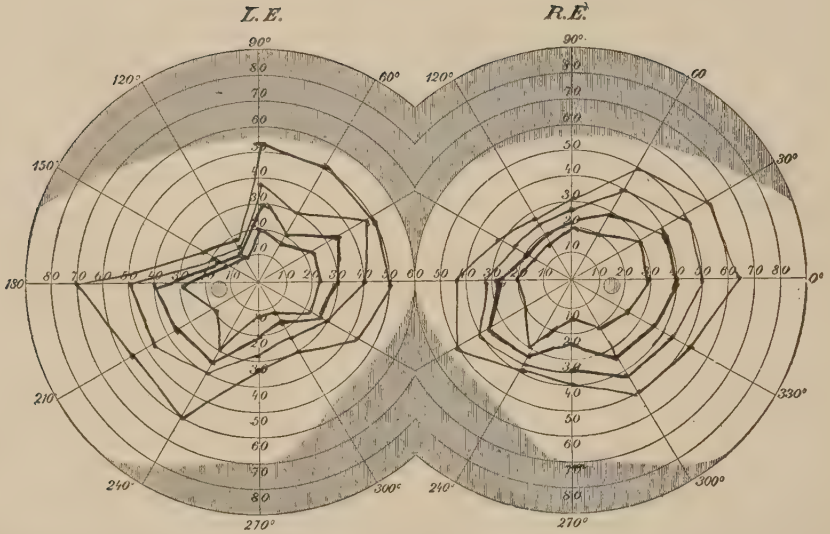
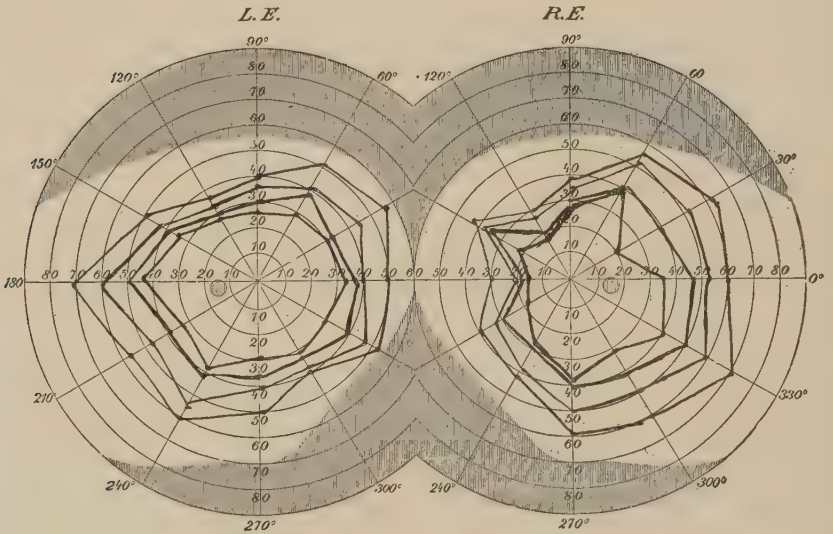


Fig. 324.—Upper left sided hemianopic indentations, two days prior to operation, in a case of adjacent abscess situated low down in the temporosphenoidal lobe just above the tegmen.



Published by E.B. Meyrowitz, Inc., New York.

Fig. 325.—Same case on following day with hemianopic indentations greatly reduced, probably because of subsidence of edema.

Figs. 324, 325.—Variations in the hemianopic indentations in inflammatory cerebral edema on successive days; demonstrating the necessity for the daily routine plotting of the visual fields in all cases of suspected brain abscess.

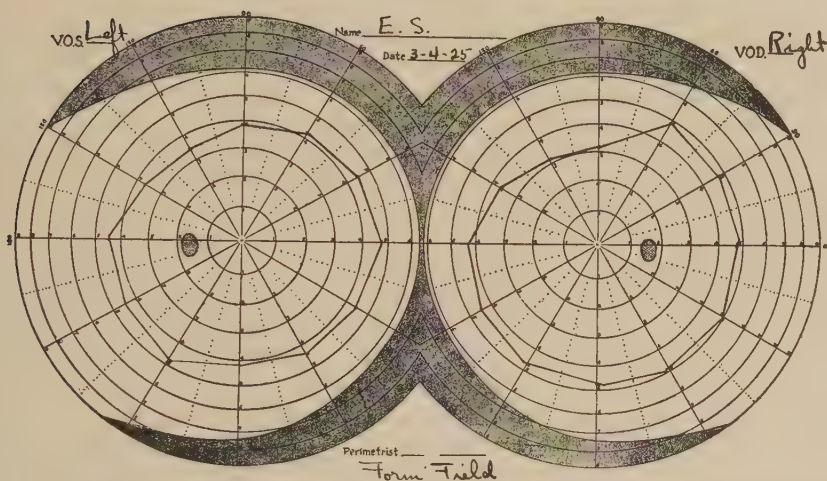
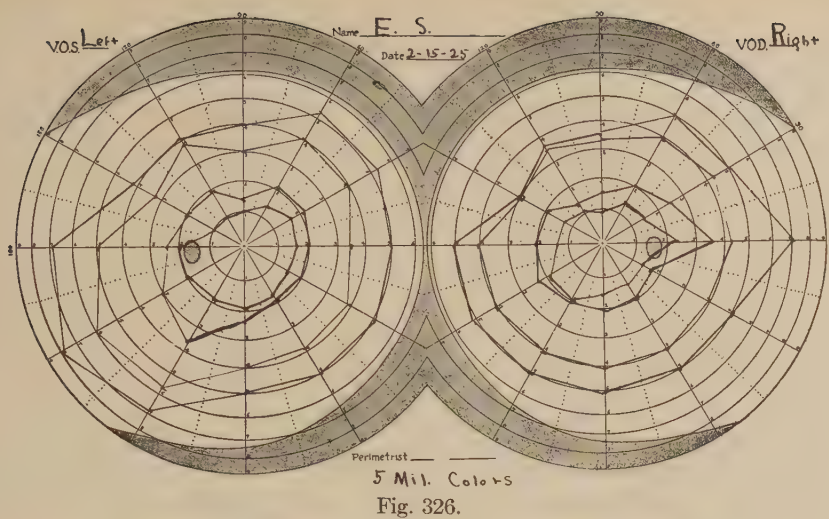


Fig. 327.—Sixteen days after second operation (ventricles tapped) twelve days after third operation.

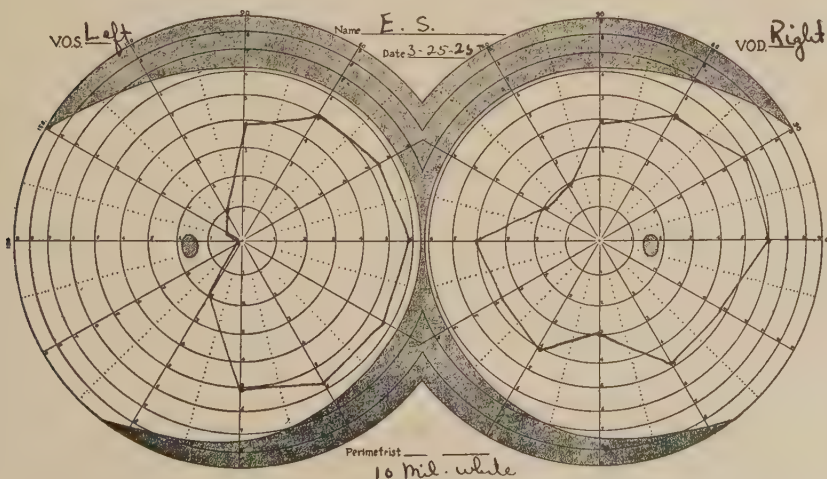


Fig. 328.—Sudden increase of hemianopsia nineteen days after fourth operation.

Figs. 326, 327, 328.—Tendency to left-sided upper hemianopic indentations in case of temporosphenoidal lobe abscess—prior to operation.

Thus in one case of a left temporosphenoidal lobe abscess the patient, on desiring a drink of lemonade, said "Give me—; give me—," and then becoming annoyed at his inability to say the right word, arose from his bed, went to the closet and picked out a lemon.

The speech difficulty can easily be elicited by showing a succession of objects and asking the patient to name them. Thus if shown a watch, the patient will say, "Um, um—that is what you tell the time by," or, shown a match, he will say, "Um, um—to light a," signifying its use by imaginatively striking the match and lighting a cigarette.

Subcortical nominal aphasia may be only partial. It is frequently characterized by the carrying over of one word; thus, if shown a key, the patient may answer correctly "key," but will repeat the same word, "key," to successive articles such as a watch or a match, although correcting himself by describing the use of the object, "to tell the time" or "to light a."

The subcortical type of "naming aphasia" may entirely disappear after being present, when due to cerebral edema from a neighboring abscess. The formerly so-called motor aphasia from involvement of the area of the island of Reil does not occur in adjacent otitic brain abscess. Either a verbal, syntactical, or somatic type of aphasia may, however, be present as the result of a metastatic thrombotic vascular occlusion originating from suppuration in the ear; in which case it is ushered in as part of a sudden apoplectic seizure.

(b) *Word deafness from cortical suppuration of the temporosphenoidal lobe* is seldom present when the collection of pus has been in the left side in a right-handed person. Theoretically there should be an associated meningitis attacking or compressing the opposite temporal region as word deafness should not occur from abscess alone—the center for hearing having a bilateral representation in the great mass of individuals.

(c) *Hemianopic indentation of the visual fields*, from edematous infiltration or pressure on the optic tract between the abscess and the lateral horn of the ventricle as it passes from the primary optic center to the cuneus (cuneopulvinar—Meyer's tract), is a frequent accompaniment of temporal lobe abscess. It is seldom complete either for form or colors. It is frequently transient because due to edema from the adjacent suppuration. Its existence is not recognized by the patient; consequently it should be carefully and repeatedly sought for by a trained technician; as, once definitely elicited, it is pathognomonic. The indentation is always bilateral and is usually situated in the contralateral upper quadrant of the fields, because of the usual position of the abscess being low down, just above the tegmen where it compresses the lower subcortical fibers of *Gratiolet* (Figs. 324, 325). However, the indentations may be in the lower quadrants, in which case the abscess is higher up and deeper in.

Exceptionally, there may suddenly appear a complete contralateral hemianopsia from the pressure of a large abscess (Figs. 326–328). If the abscess is evacuated early, the indentations completely disappear; if late, they are apt to be permanent.

(a) **Diagnostic Value of Ventricular Puncture.**—A temporosphenoidal abscess may be suspected when the posterior horn of the lateral ventricle on the side of the aural suppuration *cannot be located by puncture*, while fluid from the opposite side is obtainable.

(b) **Protective meningitis**—increased amount of fluid in the basal cisterna—always accompanies an abscess. When the suppurative process is located near or approaches the meninges, there is an associated increased cell count, chiefly polynuclear, which may differentiate it from tuberculous meningitis, in which the cells are chiefly mononuclear.

(c) **Roentgen Ray**.—There has been reported one case in which development of gas in an abscess by gas-producing bacilli made a diagnosis possible.<sup>1</sup> The injection of air into the ventricles has not been performed a sufficient number of times to determine its danger in suppurative lesions. It should be a valuable aid in the early diagnosis of temporal lobe abscess.

(d) **Motor symptoms**—contralateral paresis of face, arm, leg—are characterized in adjacent brain abscess by their slow but progressive development, in contradistinction to the apoplectic (sudden) onset of the vascular disease.

(e) **Facial weakness** of the lower portion of the opposite side, *elicited only on emotion*, is generally the earliest motor manifestation. It is of the supranuclear type, never complete; it does not include the eyelid, as is the case in peripheral facial nerve paralysis from disease of the temporal bone. The facial weakness always precedes, by an appreciable interval, the paresis of the arm and later of the leg, which is apt to follow.

In the presence of suppuration of the ear or a mastoid wound, a history of vague chill and headache followed by a supranuclear paralysis of the lower face of the opposite side to the otitis, followed later by paresis, first of the arm and then the leg, makes a diagnosis of cerebral abscess positive.

Bilateral "Greek-mask" paralysis of the face has occurred from a large abscess pressing on the deep basal ganglia.

(E) **Non-localizing Paralysis**.—(a) *Sixth nerve paralysis* from involvement of the nerve in its long course through the basal cisterna by meningeal inflammation is relatively frequent. It is usually of the homolateral side, but may be contralateral. It has little localizing significance in temporal lobe abscess.

When homolateral it may direct attention to the apical cells of the petrous pyramid as the possible origin of the intradural infection. If preceded by a thrombophlebitis of the lateral sinus it suggests involvement of the inferior petrosal sinus, the inner third of which may be under the nerve as the latter ascends in the basal dura in the neighborhood of the sphenopetrosal articulation.

(b) **Third Nerve Paralysis**—*Ophthalmoplegia Interna*.—The central communications that supply the plain muscular fibers of the sphincter of the iris are apparently more susceptible to pressure or irritation than those to the striated external muscles, thus accounting for the greater frequency of pupillary changes.

An inequality of the pupils is of little localizing value. It is evidence of cerebral involvement, but a contracted pupil of the homolateral side from irritation may be of assistance.

(c) **Ophthalmoplegia Externa**.—Paralysis of the external portion of the oculomotorius is very unusual in brain abscess; however, it is a frequent symptom of suppurative meningitis when the latter is the terminal process of brain abscess.

(d) **Fifth Nerve (Sensory Disturbances)**.—Pain in the teeth from

<sup>1</sup> See References at end of article.

pressure on the trifacial by an intracerebral abscess of otitic origin exceptionally occurs in the encapsulated variety. A dental origin is usually attributed to the pain. The absence of signs of blood-stream infection will distinguish it from fifth nerve neuralgia of cavernous sinus thrombosis.

The pain behind the eye from irritation of the first branch of the fifth from congestion or caries of the apex cells may precede the signs of a deep temporosphenoidal lobe abscess.

(F) **Psychic disturbances**—dreamy state and toxic delirium—are frequent manifestations but are apt to be mistaken for the delirium of meningitis. There is no recorded case of uncinat symptoms from a cerebral abscess.

(G) **Vestibular Manifestations.**—Spontaneous nystagmus is not present in temporosphenoidal lobe abscess, but lateral deviation of the eyes on the application of the cold caloric may be elucidated.

The vestibular manifestation of increased intracranial pressure—absence of caloric reactivity of the opposite labyrinth in the upright position—also has been observed in the early stages of brain abscess. It is of great diagnostic value as showing the presence of pressure before the appearance of the general symptoms of compression. It should always be sought for.<sup>1</sup>

(H) **Secondary Subdural Abscess.**—After evacuation and the partial disappearance of hemiparesis, the reappearance of a progressive paralysis points to extension of the suppuration in the subdural space.

#### CEREBELLAR ABSCESS

Otitic cerebellar abscess originates from an infection which has reached the brain: (1) *By direct extension* either through the labyrinth or from the perilyabyrinthine cells of the petrous pyramid, especially those in Trautman's triangle; (2) *secondary to a sinus thrombosis of the lateral sinus*; or (3) by way of a *retrograde thrombophlebitis of a small vein* originating in the area of a bony caries.

Cases arising from disease of the perilyabyrinthine cells frequently present labyrinthine symptoms; the surrounding inflammation causing a circumscribed (non-purulent) labyrinthitis.

**Factors to be Considered in the Diagnosis of the Abscess as Being Situated in the Posterior Fossa.**—Even with definite symptoms of cerebral suppuration, the localization of the abscess to the cerebellum is frequently extremely difficult; as the symptoms that accompany abscess formation in the posterior fossa are but rarely due to pressure on or destruction of an area of cerebellar tissue having a function specific enough to enable the clinician to say that the cerebellum is diseased.

For although relatively slight increase in the bulk of the contents of the posterior fossa may cause sufficient bulbar involvement to cause death, nature in its efforts to protect these vital centers allows considerable destruction of a cerebellar hemisphere with but slight impairment of its functions.

In many of the cases of cerebellar abscess of otitic origin all the symptoms present in the early stages are due to transmitted pressure (which may

<sup>1</sup> Eagleton, W. P.: Decompression for the Relief of Disturbances of the Auditory Apparatus of Intracranial Origin; Report of Three Cases with a Previously Undescribed Aural Condition. Transactions of the American Otological Society, June 10-11, 1912, vol. xiii, Part III, p. 564; also, Laryngoscope, 1913, vol. xxiii, p. 592.

continue until the suppurating area invades or presses on the brain-stem). The reason why symptoms of general pressure so frequently overshadow all others in cerebellar abscess is because of the small size of the posterior fossa, as any increase in the bulk of its contents may cause cerebral displacement, which later may obliterate the small lumen of the iter and thus produce an obstruction in the ventricles—acute internal hydrocephalus—which, in its turn, gives rise to an increase of the entire intracranial pressure. A vicious circle is thus created.

#### **Principles Usually Underlying the Early Diagnosis of Cerebellar Abscess.**

—Clinically, the *early diagnosis of cerebellar abscess* from chronic otitis is usually made by (1) the probable presence of cerebral suppuration; (2) pressure symptoms, possibly from the posterior fossa; and (3) the probable exclusion of the temporosphenoidal lobe.

The *localization of the suppuration in the cerebellum* rests on the clinical or operative findings of (a) a previous labyrinthitis, (b) caries of the perilyabyrinthine cells (generally in Trautman's triangle), or (c) a lateral sinus thrombophlebitis, more than on the pressure of any symptom of true cerebellar origin.

On the other hand, when the abscess is associated with (4) a labyrinth long known to have been functionless, as evidenced by total deafness and absence of vestibular reactivity; or (5) when the cerebellar abscess results from a sinus thrombosis or caries of the cells in Trautman's triangle with the labyrinth still intact, the patient hearing and the vestibular apparatus reactable, the symptoms (6) of cerebellar or brain-stem involvement—viz., (a) nystagmus, lateral deviation of the eyes; dilated pupils, abductor or adductor paralysis; (b) incoördination of arm, leg, or both on the side of aural suppuration, and (c) homolateral hemiparesis of arm, leg, or face—may be outstanding and pathognomonic.

**Importance of Operative Findings in the Diagnosis of Cerebellar Abscess.**—(a) Labyrinth disease is so frequently followed by cerebellar abscess that a dead labyrinth associated with symptoms of brain abscess presumably locates the suppuration in the posterior fossa. Again, the operative discovery of (b) a labyrinthine fistula or caries in the cells of Trautman's triangle should occasion apprehension that the cerebellum may become involved either from abscess or meningitis. The appearance of symptoms of localized cerebral suppuration after (c) sinus thrombosis or perisinuous abscess should suggest the cerebellum, rather than the temporosphenoidal lobe, as the site of the abscess. For, although cerebral abscess in the temporal or occipital lobe may follow transverse sinus blood-stream infection, the majority of brain abscesses which are secondary to a sinus thrombophlebitis are located in the cerebellum.

**Position of Abscess in Relation to Path of Infection.**—In a general way the position of an abscess of the cerebellum is dictated by the route through which the infection extended into the cerebellum, whether (a) through the labyrinth, (b) from a thrombophlebitis of the lateral sinus; or (c) by way of the cells surrounding the petrous pyramid. Consequently, the larger portion of the cerebellar abscesses are situated (1) near the posterior surface of the petrous pyramid; (2) in relation with the internal auditory meatus, or (3) behind or near the descending portion of the lateral sinus.

**Operative Policy in Exploration for Abscess.**—A useful operative policy in exploration of the cerebellum for suspected abscess, is (1) to try to find

the *tract of infection through the dura*, by inspection for granulations, adhesions, or a fistulous opening in the dura of the area internal to the sinus and external to the lip of the internal auditory meatus. This area includes (a) the region of the ductus endolymphaticus; (b) the perilabyrinthine cells between the horizontal and superior semicircular canals; (c) the dural prolongation into the bone; and (d) the convexity of the posterior semicircular canals—all of which may be the path of infection from the bone in the order of frequency above named.

This area contains the "stalk" leading to the cerebellar abscess in the small proportion of cases in which such a fistula is present.

(2) Failing to find such a lead the dura should be opened and the cerebellum searched (a) in the neighborhood of the internal auditory meatus when the labyrinth is dead; and (b) behind and above the knee of the sinus when the lateral sinus is the cause of the abscess. A considerable proportion of cerebellar abscesses without a stalk occupy these sites, because of the route by which the infection extends.

**A. Localization of Cerebellar Abscess.**—Symptoms of labyrinthine suppuration that warrant the suspicion that the cerebellum is affected—spontaneous nystagmus, vestibular dizziness and falling—if associated with either headache or fever, point to an involvement of the posterior fossa either by meningitis or abscess, or both.

*Vestibular Mechanism in Relation to Cerebellar Abscess.*—Vestibular manifestations are frequently present in cerebellar abscess. The nystagmus, vertigo, and falling of acute labyrinthine suppuration resemble, although they are not exactly the same as, those from pressure on the brain-stem; but in cerebellar abscess it may be impossible to distinguish between them, especially as the two processes are, in the majority of cases, co-existent, there being an associated labyrinthine suppuration with brain-stem irritation.

*Development of the Vestibular and Cerebellar Mechanisms.*—The semicircular canal systems are primarily for the reflex control of the relative position of the eyes to the head—orientation.

Nystagmus is a pure reflex, presided over by the vestibular mechanism with peripheral portions in the static labyrinth, the eye, and the ocular muscles and central connections and nuclei in the brain-stem—corpora quadrigemina, medulla, and pons. Although the vestibular apparatus has a decided influence on the muscular control (tonus of the head and other muscles) in animals, in man this tonus control is so slight as to be of little clinical value.

The cerebellum, on the other hand, is for the reception, co-ordination, and regulation of motor and sensory stimuli; so that from the cerebellum are transmitted the nervous impulses which occasion co-ordinated muscular movements and sensory acts—synergia. Orientation of the head, like all muscular movements and sensory stimuli, is regulated and co-ordinated in the cerebellum. Thus, the cerebellum cannot produce nystagmus, but it influences its intensity.

**Differential Diagnosis Between the Vestibular Manifestations—Nystagmus, Dizziness, and Falling—Seen in Cerebellar Abscess.**—(1) *Nystagmus.*—The nystagmus seen in cerebellar abscess may be either of labyrinthine (peripheral) or of brain-stem (central) origin. It is but rare that a positive diagnosis of the site of the lesion can be made from the direction

of the spontaneous or induced nystagmus alone, although the former may furnish valuable confirmatory evidence.

*Characteristics of Peripheral and Central Nystagmus.*—Theoretically, pure spontaneous vestibular nystagmus of peripheral origin, from labyrinthine disease, is more marked in one or other of the extreme corners, depending upon (a) the side affected, (b) whether due to irritation or

Acute destruction of  
right labyrinth  
(Non-functioning)



Left labyrinth  
irritated



Direction of nystagmus.

Fig. 329.—Acute destruction of labyrinth gives horizontal rotatory nystagmus at first toward the labyrinth still functioning—that is, *away from the side of the lesion*.

Non-destructive labyrinthitis (right). Right labyrinth irritated.



Direction of nystagmus

Fig. 330.—Irritation of labyrinth without destruction (cholesteatoma erosion) gives horizontal rotatory nystagmus *toward the side of the lesion*.

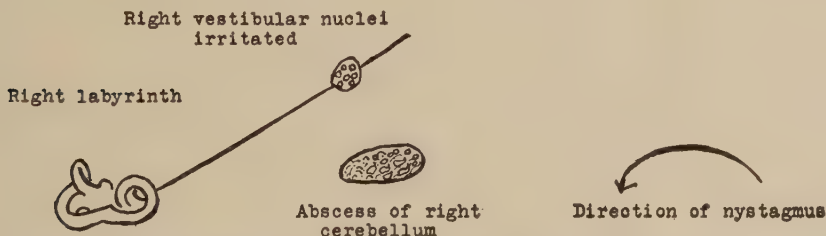


Fig. 331.—Cerebellar abscess without labyrinthitis during the early stage may cause irritation of vestibular nuclei in the brain-stem, and then *should* cause nystagmus toward the side of the lesion.

Figs. 329, 330, 331.—*Theoretical differences in the direction of the spontaneous nystagmus in different types of labyrinthine disease and cerebellar abscess.* The direction of the nystagmus should be toward the labyrinth or nucleus irritated at least during the period prior to the establishment of compensation.

destruction, and (c) the duration of the lesion; that is, how far compensation has progressed.

The nystagmus following cerebellar destruction from gunshot wounds, which is most marked on looking away from a "rest point of Holmes," 10 to 30 degrees from the midline away from the side of the lesion has not been observed in cerebellar abscess. (This "rest point" is thus a forced muscular position of cerebellar involvement.)

Theoretically the nystagmus of peripheral vestibular origin is toward the labyrinth furnishing the irritation. Thus, when the right labyrinth is suddenly destroyed (Figs. 329, 330), the opposite one causes nystagmus at first away from the side destroyed on account of imbalance. With the readjustment which rapidly supervenes (in which the cerebellum plays a large part) the nystagmus disappears after first undergoing modifications.

The nystagmus associated with cerebellar abscess of the early stage, before coma supervenes, is usually toward the side of the suppurating ear and results from irritation of the vestibular nuclei in the brain-stem (Fig. 331). For theoretically and practically the direction of the *brain-stem nystagmus* should be toward the side of the lesion. Clinically, however, the brain-stem nystagmus in cerebellar abscess frequently is (a) entirely absent, especially in the early stages; and if present is usually characterized by (b) its transient duration and (c) changing character. In fact, a sudden change in the direction with an increase in the intensity of the nystagmus is frequently the first positive sign of involvement of the cerebellum secondary to a labyrinthitis. The changing character of cerebellar nystagmus—one day being toward the one side, and the next day more marked to the other—is partially explained by the edematous nature of the inflammatory process.

Clinically the spontaneous nystagmus associated with cerebellar abscess, while frequently absent entirely or changing in character, in many cases has a persistence in direction and degree not present in labyrinthine nystagmus.

*Conclusions Out of the Diversity of the Clinical Findings in the Spontaneous Nystagmus in Cerebellar Abscess.*—It may be stated that in the imperfect knowledge of the cerebellar functions now at our disposal the following clinical conclusions are of practical value:

(a) The presence of spontaneous nystagmus is important only in relation to other symptoms. Its absence does not exclude cerebellar abscess—its direction being, as a rule, of much less importance than the theoretically confused statements would lead the student to believe.

(b) The presence of spontaneous nystagmus in a suspected case of cerebellar abscess *with* a functioning labyrinth is suggestive of cerebellar involvement. If there are symptoms of brain suppuration in addition to the presence of a spontaneous nystagmus, it is diagnostic of posterior fossa involvement.

(c) In the presence of a labyrinth non-reactable at the time of the examination, its reactability, however, having but recently been lost, the presence of a spontaneous nystagmus against the rule of labyrinth disturbance (Fig. 329), that is, *toward* the side of the now functioning labyrinth, is diagnostic of cerebellar involvement, *when* associated with other symptoms, headache, vomiting, and temperature.

(d) The direction of the nystagmus (while occasionally of value in the diagnosis of cerebellar abscess), when it changes suddenly, should not play a prominent part in the diagnosis of cerebellar abscess in general.

(e) Vestibular nystagmus, both peripheral and brain-stem, is generally of the horizontal rotatory type.

(f) Vertical nystagmus is always of central origin, usually from the brain-stem and probably from involvement of the vestibular central mechanism.

(g) *Lateral* deviation of the eyes away from the side of the lesion—the

result of the elimination of the quick component—and the assuming of the eyes in the “rest position,” away from the side of the lesion, is a frequent localizing symptom in cerebellar abscess, especially during the stage of semicoma. With spontaneous lateral deviation of the eyes the voluntary or involuntary movement away from the “rest point” causes a nystagmus.

(h) *Abnormalities in induced nystagmus*—such as the absence of the quick component (the production of lateral deviation)—are of little localizing value.

(2) *Vertigo and Falling*.—Both labyrinth and cerebellar disease may cause vertigo and falling. (a) The vertigo of vestibular origin is either of the turning variety or an error in sensation. (b) The vertigo in cerebellar abscess is chiefly the turning dizziness from vestibular brain-stem irritation; the distinctive cerebellar part being incoördination of the muscles of locomotion—ataxia. (c) The theoretical alterations in the direction of the falling, which should occur with a change in the position of the head, have proved of little clinical differential value; for, while in labyrinth disease, in the early stage, the patient falls in the direction of the slow component of the nystagmus, and the direction is *often* dependent upon the position of the head; in central disease experience shows that, although the patient should fall in the direction of the quick component, the falling is, as a rule, independent of its directions, as well as of the position of the head.

(d) When the worm of the cerebellum is the site of the disease, the falling is generally persistently backward—a condition seldom present in abscess.

(e) The attacks of sudden forced “lateral propulsion,” which occur in lateral sclerosis and cerebral syphilis, have not been observed in cerebellar abscess.

(3) *Forced positions of the head or body* may be cerebellar, but are never vestibular.

(4) *Past-pointing* is of corticocerebrocerebellar origin. It should not, in any sense, be regarded as a reflex; and thus comparable with nystagmus or lateral deviation of the eyes. Abnormalities of past-pointing are not diagnostic of cerebellar disease, but when present, may be regarded as further evidence of central involvement; only, however, when the clinical picture warrants such an interpretation. The specific direction of the abnormality is of no clinical value. The theoretical contentions on which they were formulated have not been substantiated anatomically or clinically.

**B. Posterior fossa symptoms in brain abscess** may be analyzed as those due to:

(a) Cerebellar tissue suppuration;

(b) Those from brain-stem displacement with an associated pressure;

(c) Internal hydrocephalus; and

(d) Those from secondary meningitis.

(1) *Constitutional symptoms suggestive of cerebellar suppuration*: (a) *Suboccipital tenderness*, (b) rigidity of neck, (c) yawning, (d) rapid loss of flesh, (e) alteration in knee-jerks, (f) vomiting, and (g) sugar in the urine without an excess in the blood.

Of these, perhaps the most distinctive is the rapid loss of flesh which occurs in all cases of manifest cerebral suppuration, but especially in cerebellar abscess.

The reduction of the increased intracranial pressure following the administration of magnesium sulphate by rectum—thus reducing the brain's bulk—helps to demonstrate the intracranial nature of the vomiting.

(2) *Physiological impairment of cerebellar function* constitutes asynergia, which condition is a combination of homolateral muscular (a) asthenia, or lack of strength; (b) astasia, or muscular unsteadiness; and (c) hypermetria, or lack of muscular inhibition.

The asynergia of cerebellar abscess takes on the clinical appearance of one or other of these and can be so analyzed.

*Clinical Manifestations of Impaired Cerebellar Function Which Occur in Abscess.*—(1) *Forced cerebellar attitudes:* (a) The patient always lying curled-up on one side. It is generally elicited only after inquiry as to whether the sleeping position was formerly on either side, but since the advent of the present illness has inclined to one side. (b) Lateral conjugate deviation of eyes (the primary component of nystagmus). (c) Conjugate ocular palsies, both eyes looking toward the right or left, the patient being unable to move the eyes together in different directions and hold them there. This condition is often overlooked because unaccompanied by diplopia, and may only be discovered when the consciousness is removed, as in coma. In acute destructive lesions, such as gunshot wounds, the eyes spontaneously tend to assume the rest position of about 30 degrees away from the side of the lesion. It may be associated with lateral deviation or ocular paralysis.

(2) *Homolateral "cerebellar paresis,"* generally of both arm and leg. It is not a complete paralysis, but a paresis. It is made up of asthenia—all the muscles being flabby—lack of tone and, while the limb can move, it does so uncertainly.

(3) *Incoördination*, generally of one arm or hand, manifested by:

(a) Cerebellar ataxia—Romberg;

(b) Adiadokokinesis;

(c) Dysarthria—the scanning speech of cerebellar abscess.

(4) *Hypermetria.*—Easily demonstrated in a conscious patient by a failure of inhibition on testing the spontaneous past-pointing, the patient bringing the arm too far backward.

(5) *Cerebellar Convulsions.*—Largely oscillatory in type; rarely occur.

**Compensation in Cerebellar Abscess.**—As the cerebellum is primarily for co-operation, destruction of any of its parts is rapidly compensated for; thus in cerebellar abscess the pure cerebellar manifestations are apt to be very transitory.

**C. Symptoms from Direct Pressure in the Posterior Fossa in Cerebellar Abscess.**—1. *Sixth nerve paralysis* of either side is the most frequent ocular motor symptom in cerebellar abscess. It may result from direct or transmitted pressure on the nerve as it passes in its long course around the crus, over the apex of the petrous portion of the temporal bone, and under the sphenopetrous ligament. Abductor paralysis is of little localizing value in cerebellar abscess.

2. *A fourth (trochlearis) nerve paralysis* may be present in cerebellar abscess when the suppuration is situated high up toward the mesencephalon. The resulting diplopia may be disregarded by the patient, being overcome by an assumed position; in which case the ocular paralysis may be elicited by the application of the cold caloric in the recumbent position; the induced

nystagmus is horizontal, its rotary element being largely eliminated. The rotary element in a normal nystagmus is chiefly furnished by the trochlearis (the more commanding of the two obliques).

3. *Fifth nerve involvement*—hyperesthesia of the face and diminution of the corneal reflex on the homolateral side—may be present.

*Oppenheimer's symptom*—induced loss of corneal reflex by utilizing the increased weight of the diseased hemisphere for pressure on the fifth (after bringing the head forward and the side of the lesion downward)—should be carefully investigated in all cases of cerebellar abscess, for, although it has not been observed, its presence would be of great localizing value.

**D. Ocular Symptoms from Transmitted Pressure.**—1. *Mydriasis* and *inequalities* in the pupils are frequent manifestations.

2. *Third nerve paralysis*, possibly from pressure on the nerve between the abscess and the inferior anterior cerebellar artery, might occur.

**E. Bulbar Symptoms.**—The increase in the size of the cerebellum from the abscess, the induced internal hydrocephalus, and the displacement of the midline of the brain-stem may give rise to bulbar symptoms from pressure on the ninth, tenth, eleventh, and twelfth cranial nerves. Sudden respiratory paralysis is a frequent termination either from (a) cerebellar herniation through the medulla, or (b) punctate hemorrhages into the pons—especially when the cerebral displacement is suddenly altered following the withdrawal of fluid by a lumbar puncture or a disturbance of the circulation during the administration of an anesthetic.

Avellis' syndrome of paralysis of the ninth, tenth, and eleventh has not been observed in cerebellar abscess.

Deviation of the tongue from twelfth nerve involvement is sometimes present.

**F. Symptoms of internal hydrocephalus** are especially apt to be present in cerebellar abscess, from obliteration of the iter by the abscess. An acutely produced internal hydrocephalus causes severe papilledema. A moderate degree of papilledema is much more frequent in cerebellar than in temporosphenoidal lobe abscess.

Severe papilledema in cerebellar abscess suggests the immediate reduction of the intracranial pressure, by ventricular puncture or the administration of magnesium sulphate, before subjecting the patient to the further cerebral strain of an operation.

**Localizing Value of Ventricular Estimation.**—The demonstration of a dilatation of both lateral ventricles by ventricular puncture may be of the greatest assistance in localizing the abscess in the cerebellum; as whenever the temporosphenoidal lobe is the site of the abscess, the homolateral ventricle is compressed. Dilatation of both lateral ventricles in a case of cerebral suppuration is presumptive evidence that the abscess is in the cerebellum.

The injection of air into a ventricle dilated from compression of the iter by a cerebellar abscess is attended with considerable danger, the injection of air apparently being followed by an overproduction of the cerebrospinal fluid. Any interference of the ventricular fluid by aspiration or air injection in a ventricle distended by pressure of a cerebellar abscess should immediately be followed at least by an extensive decompression of the cerebellum.

**Vestibular Manifestations of Increased Intracranial Pressure.**—Absence

of reactivity of the opposite labyrinth to the cold caloric in the upright position<sup>1</sup> is of some localizing assistance. It may be present in temporosphenoidal lobe abscess, but in cerebellar abscess it occurs at a comparatively early stage, frequently long preceding the development of a papilledema.

**Secondary Meningitis.**—In all cases of cerebellar abscess there is an associated increase of the cerebrospinal fluid in the (a) cisterna pontolateralis and (b) cisterna cerebellomedullaris, both of which greatly increase the general pressure symptoms.

**Localizing Value of Eliminations of Hemianopic Indentations of the Visual Fields.**—Routine and daily repeated examinations of the visual fields are important aids in localizing the cerebral suppuration in the posterior fossa; as the persistent absence of hemianopic indentations assists in excluding a temporosphenoidal lobe abscess. There is clinical and pathological evidence, however, that the general central edema that accompanies cerebellar abscess may cause hemianopic visual indentations accompanied by a general field contraction.

**Combined temporosphenoidal and cerebellar abscesses** occur in about 2.5 per cent. of all cases of brain abscess. A localized temporosphenoidal suppuration occurs in about 6 per cent. of all cerebellar abscesses. Up to the present time combined abscesses of the cerebellum and temporosphenoidal lobe have been diagnosed and successfully operated upon in but one case.<sup>2</sup>

#### OTITIC MENINGITIS

Surgically, otitic meningitis may be defined as a septic inflammation of the cerebrospinal fluid system; the type and nature of the meningeal symptoms depending upon (a) the virulence and extent of the infection within the subarachnoid meshes, and (b) the part played by the protective reaction.

An overpowering infection involving the whole system presents the clinical manifestations of *fulminating septic leptomeningitis*, usually following, or concomitant with, an acute blood-stream infection.

On the other hand, when the pathological process is largely a protective reaction (in nature's effort to limit the infection) and the suppuration is confined to a small area of the dura outside the subarachnoid spaces, there is present a local or general *protective meningitis*, which, however, is frequently accompanied by cerebral and constitutional symptoms.

The *symptoms of exudative septic meningitis*, local or general, acute, subacute, and chronic (the type most frequently encountered with otitic cases), are generally produced by a combination of both processes. For it is the usual course for a protective meningitis to become a localized septic exudative meningitis, which is followed, after a longer or shorter period, by a general exudative leptomeningitis, which may at any moment become fulminating.

**Surgical Classification.**—*Exudative Suppurative Meningitis.*—From a surgical standpoint exudative suppurative meningitis of otitic origin should be divided into (a) an *initial primary* stage in which the infection is limited to the subarachnoid space in the neighborhood of the primary focus of in-

<sup>1</sup> Eagleton, Wells P.: Decompression for the Relief of Disturbances of the Auditory Apparatus of Intracranial Origin: Report of Three Cases with a Previously Undescribed Aural Condition, Tr. Am. Otol. Soc., June 10, 1912.

<sup>2</sup> Eagleton, W. P.: Localizing Value of Ophthalmic Examinations in Suppurative Lesions of the Brain, Tr. Ophthalmological Section, American Medical Association, 1928.

fection, and (b) a *terminal* stage, in which bacteria have invaded the whole cerebrospinal fluid system.

**Relationship Between the Circulatory and the Cerebrospinal Fluid Systems.**—There is an intimate relationship between the cerebrospinal and the circulatory systems. The perivascular prolongations of the subarachnoid spaces surround the small capillaries, while the arteries of the base lie in the basal cisterna. The cerebrospinal fluid is discharged through the villi into the venous system of the dura.

**Diffuse Fulminating Leptomeningitis Concomitant with Blood-stream Infection.**—Normally there is a free interchange of fluid between the cerebrospinal and the hemic systems, and micro-organisms which may have gained entrance into the subarachnoid spaces are discharged into the circulation; but there is a barrier that prevents the bacteria, so frequently present in the blood-stream, from entering the cerebrospinal fluid system in hemic infections, sinus thrombosis, etc.

However, in certain severe types of overpowering blood-stream infections, bacteria within the blood-stream attack the intima of the blood-vessels of the brain—fulminating concomitant meningitis. The disease is characterized pathologically by multiple small hemorrhages into the pia.

Again, it has been experimentally demonstrated that if the normal hemic encephalic barrier is disturbed—such as by massive lumbar puncture (the removal of a large quantity of fluid) while micro-organisms are free in the blood-stream—the bacteria may enter the meninges, and give rise to fulminating exudative leptomeningitis.<sup>1</sup>

**1. Fulminating Meningitis Secondary to Bone Lesions.**—In certain encapsulated bacillus—*Streptococcus mucosus*—the disease in the bone may be largely quiescent for some time, and then suddenly cause a diffuse fulminating meningitis, with death within a few hours or days.

**Symptoms.**—Fulminating meningitis as a rule follows an acute otitis, with a coryza. It is ushered in by sudden agonizing pain in the head, persistent high temperature, associated with one or more chills, rapidly followed by stiff neck, restlessness, convulsions, and delirium, with diffuse terminal motor-paralysis, coma, and death. The cerebrospinal fluid from the lumbar region in the early stage may be clear but under pressure, and although the cell count may be low, it contains numerous micro-organisms—streptococci, diplococci, pneumococci, and *Streptococcus mucosus*—which may also be isolated from the blood-stream.

Postmortem reveals, in addition to exudates in the basal cisterna and along the sulci, that the cortex contains numerous punctate hemorrhages with isolated areas of septic phlebitis and pneumococcic infection, while fluid from the nasal accessory sinuses may be filled with this organism—all evidences of the blood-stream origin of the disease.

**2. Otic Exudative Meningitis.**—Septic exudative leptomeningitis is the usual pathological process in otitic cases. In its terminal stage it presents such outstanding symptoms as to offer little difficulty in diagnosis; but once the pyogenic process has become general, a fatal termination is inevitable, except in very unusual cases.

Surgery, to be effectual, must attack the disease while it is still limited to an area of the cerebrospinal fluid system, and this at a time when the purulent nature of the meningitis is only manifest by vague symptoms.

<sup>1</sup> Case XXV, p. 188, from Eagleton's Brain Abscess.

A sufficiently early diagnosis can, however, be made by attention to (1) *trivial symptomatic manifestations*, in conjunction with (2) the *clinical diagnosis* of the path of infection from the primary focus, by which the suppurative process reached and extended into the meningeal spaces.

The *symptoms* of the type of meningitis (exudative) of otitic origin which is ordinarily encountered may be divided into three stages :

(A) *Stage of protective reaction* of the cerebrospinal fluid system in an area more or less contiguous to the local infection in the bone and dura; (1) *localized protective meningitis*, which may be confined (a) to the subarachnoid prolongation in the internal auditory meatus (when the infection has reached the cerebrospinal spaces by way of the modiolus); (b) to the anterior surface of the cerebellum (when due to caries of the perilabyrinth cells, or to Trautman's triangle, with vestibular suppuration from involvement of the ductus endolymphaticus; or caries of the posterior semicircular canal); (c) to the under surface of the temporal lobe (when the infection follows osteophlebitis, extradural abscess, or pachymeningitis internal over the tegmen); (d) to the external surface of the temporal lobe (when associated with an osteomyelitis of the squama); (e) to the superior surface of the cerebellum, or (f) the under surface of the occipital lobe (when due to transverse sinus thrombophlebitis).

Localized reactive (protective) meningitis when so limited, generally presents few symptoms attributable to the part of the meninges affected, although there may be slight headache with a moderate degree of temperature and accelerated pulse. The lumbar puncture reveals increased fluid, with a cell count above normal.

When, however, the protective reaction is limited (g) to the basal cisterna (from infection of the deep cells of the petrous pyramid through the labyrinth or by way of the peritubal, the perilabyrinthine cells, or the canalis subarcuatus) it is manifested by Gradenigo's syndrome—pains in the homolateral temporal or trigeminus region (pains around or behind the eye or in the teeth), associated with sixth nerve paralysis.

Between the mild symptoms of local protective meningitis and the severe constitutional and cerebral symptoms of (2) *general protective meningitis* (*meningismus* of infants)—high temperature, delirium, convulsions, and coma with excessive cloudy fluid and increased globulin, but without microorganisms in the fluid—every degree of severity is possible.

In both localized or general protective meningitis, removal of the primary focus of infection is usually followed by rapid subsidence of all symptoms.

**3. Protective Meningitis in Brain Abscess.**—Intracerebral abscess in the manifest stage is always accompanied by an increased production of cerebrospinal fluid. The protective reaction of the meningitis contributes toward the symptoms of compression and cerebral irritation—irregular sudden rise of temperature, vomiting, pain behind the eyes and, when the posterior fossa is involved, stiff neck.

With the extension of the infection from failure of the protective reaction to limit the process, there is apt to follow (B) a *stage of localized septic meningitis*—the infected cerebrospinal fluid being confined to the area adjacent to the primary focus or to the neighboring basal cisterna. This stage may continue several days, or in exceptional cases even for weeks. It is characterized by moderate headache, slight sleeplessness, irritability,

irregular vomiting, temperature, slight stiff neck, and Kernig. The general symptoms may be with or without localizing symptoms—such as nystagmus and vertigo when the posterior fossa is involved or, when confined to the middle fossa, pain behind the eyes, in the face or teeth, or ocular paralysis. Lumbar puncture demonstrates excessive fluid under pressure with very high cell count, and an increased globulin without micro-organisms, although culture may isolate bacteria from the fluid of the area adjacent to the primary focus of infection.

Eradication of the focus with or without the evacuation of the infected fluid, depending on the degree of the infection, will stop the process in a limited number of cases. If unrelieved, localized septic meningitis passes into (C) a stage of general septic leptomeningitis with continuously high temperature, rapid pulse, severe headache, excessive irritability, great restlessness, sleeplessness, screaming, delirium, alternating with coma, opisthotonos, marked Kernig and Bondi signs, ocular paralysis, nystagmus, and death usually in eight to fourteen days from the beginning of the disease.

**Paths of Infection in Otitic Meningitis.**—Labyrinthitis causes 30 per cent. of the cases of otitic meningitis. Thrombophlebitis of a large sinus, 27 per cent. with an additional 10 per cent. from thrombophlebitis of a small vein of the bone, thus making a total of 37 per cent. of phlebotic origin. Brain abscess causes 13 per cent. In 27 per cent. of all cases of meningitis associated with otitis, the path of infection is unknown. This applies especially to the fulminating type. Many of the latter probably result from labyrinthine disease or from small vein phlebitis.

Consequently, labyrinthine suppuration and thrombophlebitis of a basal sinus comprise the majority of cases of otitic meningitis.

**Clinical Subdivisions of Otitic Meningitis According to the Route of Infection.**—Clinically, otitic meningitis may be divided into cases following (1) labyrinth suppuration, (2) during a thrombophlebitis of a large venous sinus or of a small vein; and (3) secondary to brain abscess.

*Surgically and anatomically, otitic meningitis* may be divided into cases originating from (1) disease of the bone; (2) along preformed ways; or (3) through the blood-vessels. Any one of these routes may reach the meninges (a) external to the labyrinth; (b) through the labyrinth; or (c) from the cancellous tissues of the apex of the petrous pyramid.

**Etiological Importance of "Preformed Ways."**—Infection through a "preformed way," by an acute inflammation or an acute exacerbation of a chronic otitis, furnishes the largest number of cases of otitic meningitis, the infection entering the dura either as (1) the result of labyrinthine suppuration, or (2) through a dehiscence in the bone.

1. *Meningeal infection by way of the labyrinth* is the more important, as the cochlear part of the labyrinth should be regarded surgically as an outpost of the cerebrospinal fluid system. Once the cochlea has been involved, a direct path is opened into the subarachnoid space through the modiolus. The semicircular canals (although in continuity with the cochlea through the vestibule) are much less often the cause of meningitis, because of their small size and more external position; nature limiting the infection in the canals before the vestibule becomes the site of a purulent inflammation. Even after invasion of the vestibule the disease is frequently controlled by an exudate into the cornucopia-like prolongation of the cisterna pontis of the internal auditory canal.

2. *Meningitis in Infants with Acute Otitis*.—The route of infection is frequently by a dehiscence in the bony wall, which thus allows a direct extension of the inflammatory process through the dura.

**Importance of General Cerebral Symptoms When Accompanying Specific Types of Aural Suppuration in the Early Recognition of Meningitis.**—(a) Labyrinthine suppuration associated with temperature and headache is evidence that the meninges are attacked; especially if the fluid from the lumbar region has a high cell count and increased globulin. However, the meningitis may be chiefly of the protective type. The fluid obtained from the subarachnoid prolongations of the cisterna pontis into the internal auditory meatus often contains micro-organisms, while that of the lumbar region still remains sterile.

Meningitis is to be suspected in (b) cases of sinus thrombophlebitis with headache, irritability or delirium out of proportion to the degree of temperature; in (c) cases of deep cell involvement when associated with ocular paralysis, headache and temperature; in (d) all cases of aural suppuration associated with high cell count and increased globulin.

*Importance of Non-functioning Labyrinth in Diagnosis of Meningitis.*—Under no circumstances should a functioning labyrinth be opened in the presence of a suppurative otitis, because of the danger of precipitating a meningitis from extension of the suppurative process through the labyrinth to the meninges. Consequently, a diagnosis of whether the labyrinth is functioning or not is of importance.

A serous labyrinth may give rise to a spontaneous nystagmus (at first to the irritated side) with dizziness and falling, while a little hearing is still retained and the vestibular apparatus reacts. When nystagmus, dizziness, and falling accompany a dead labyrinth—total deafness and vestibular non-reactability—the symptoms may still arise from the labyrinth alone (an acute localized purulent labyrinthitis).

When, however, nystagmus, dizziness, and falling are associated with headache and fever, they signify secondary meningeal involvement and call for the immediate opening of the labyrinth if the infection is to be checked.

4. **Meningitis Secondary to a Subacute Thrombophlebitis of a Basal Venous Sinus.**—In subacute thrombophlebitis of the large venous sinuses the blood-stream infection itself is rarely the cause of death, which is generally due to secondary leptomeningitis. Cases of sinus thrombophlebitis of the cranial base, at an early stage, are frequently associated with symptoms of meningeal involvement—papilledema and slight headache—because of the protective reaction in the meninges (protective meningitis), which is excited by the phlebitis.

The **symptoms of septic exudative leptomeningitis** are chiefly the result of (1) general toxemia and (2) cerebral irritation. They depict the progress of the inflammation as it extends at first through a basal cisterna, then over the cortex, and down the cord, during which it occasions (3) paralysis and inhibition of the nervous tissue, as the exudate plugs the spaces of the subarachnoid meshes and perivascular channels, and gives rise to (4) cerebral toxemia.

5. **Localized Otitic Meningitis.**—The anatomical formation of the subarachnoid in the neighborhood of the spaces of the petrous pyramid, viz., (a) the lateral prolongation of the bulbar cisterna surrounding the structures which pass through the internal auditory meatus; (b) the small caliber of

the ductus endolymphaticus, and (c) the fine mesh of the spaces above the tegmen, all lend themselves to the localization of suppuration. In addition, the physiological function of the cells of the arachnoid is to phagocyte invading bacteria and by adhesion of its surfaces to wall-off invading micro-organisms from the general subarachnoid circulation. Thus, in the early stages of meningeal invasions it is necessary that (1) not only must the exact route by which invading micro-organisms reached the dura be diagnosed, but also (2) the exact anatomical subarachnoid space first invaded. For the opening of this space will frequently evacuate infected fluid containing micro-organisms while the general subarachnoid spaces are still sterile.

**Clinical Subdivisions of Localized Otitic Meningitis.**—Clinically, localized meningitis may be diagnosed as (1) middle fossa cases, the symptoms including those of fifth nerve involvement; (2) posterior fossa cases, with symptoms of cerebellar involvement; and (3) apex of petrous pyramid cases, which give rise to a combination of middle and posterior fossa symptoms, combined with those of invasion of the bulbar cisterna.

(1) *Middle fossa cases*, in which the infection passes through the *tegmen tympani et antri*, are accompanied by: (a) excessive fluid over the infected region and the adjacent squama; (b) headache, especially nocturnal, in the region above the ears; (c) irregular temperature; (d) dilated veins of the optic papillæ, or slight papilledema with little swelling of the nerve head. If the infection extends further inward (e) the fifth nerve may be involved—giving rise to pain in the region of the eye because of congestion of the dura to the first branch of the trifacial ganglion, prior to which the (f) abducens (sixth) of the homolateral side is apt to be paralyzed from swelling of the dura, as the nerve has a long and greatly restricted course through the canal of Donello before it passes under the petrosphenoid ligament.

If the abducens paralysis and fifth nerve irritation are due to congestion of the base alone, they are not accompanied by sepsis. Such cases are apt to be relieved by the subsidence of congestion which follows drainage of the antrum, and complete exenteration of the cells of the mastoid. If, however, Gradenigo's syndrome is associated with sepsis, drainage of the localized infected cerebrospinal fluid is indicated.

(2) *Petrous fossa cases*, which are clinically manifested by involvement of the subarachnoid spaces covering the anterior surface of the cerebellum, may be divided into (a) infection which extends from the cells of Trautman's triangle, in which case the lateral cisterna may be involved when it extends well outward; (b) infection through the ductus endolymphaticus; (c) infection through the labyrinth by way of subarachnoid prolongation of the pontine cisterna; (d) infection through caries and necrosis of the posterior semicircular canal, in which case either the lateral prolongation or the bulbar cisterna may be the seat of a localized collection of fluid. They all give symptoms of acute cerebellar involvement, viz., spontaneous nystagmus, abnormal past-pointing, dizziness, reduction of the duration of the induced nystagmus from turning to at least 50 per cent. of the normal<sup>1</sup> with stiff neck, temperature, and vomiting.

<sup>1</sup> Eagleton, Wells P.: Vestibular Tests in Intracranial Surgery. The importance (1) of the absence of reactivity of the vertical canals of both ears to stimulation by the cold caloric in the diagnosis of increased intracranial pressure of the posterior fossa, and (2) of the reduction of the duration of the nystagmus from turning in diffuse lesions of the cerebrospinal system pathways over the cerebellar cortex (as in protective meningitis and linear fractures of the skull of the posterior fossa). Tr. Am. Laryngol., Rhinol., and Otol. Soc., Inc., 1922.

(3) *Petrous apex cases* due to caries of the cancellous tissue which fills in the space bounded exteriorly by the labyrinth, anteriorly by the internal carotid artery, and interiorly by the petrosphenoid articulation. The resulting meningitis is characterized by a combination of middle fossa symptoms (pain behind the eye) and sixth nerve paralysis, posterior fossa symptoms, central nystagmus, associated with bulbar cisterna irritation—stiff neck and bitemporal hemianopic contraction of the visual fields.<sup>1</sup>

*Symptoms of meningeal involvement* may be analyzed into those of:

1. General toxemia;
2. Cerebral irritation;
3. Symptoms of cerebral compression, which may be either:
  - (a) General, or
  - (b) Localizing.

Compression occurs only in the terminal stage. It is generally due to an associated internal hydrocephalus.

1. **Symptoms of General Toxemia.**—(a) *Temperature.*—The temperature in meningitis is a sign of general sepsis. As long as the process is limited the temperature may be slight; but becomes continuously high as soon as the whole system is involved. A moderate temperature is a favorable prognostic sign.

(b) *Chills* are suggestive of blood-stream origin. In the fulminant type there may be repeated chills with marked remissions of temperature—generally significant of a severe blood-stream infection. Exudative meningitis rarely is ushered in by a chill.

(c) *Pulse.*—The pulse at first may be slow; however, as soon as the process has become diffused it becomes fast, and so continues until the fatal issue. A pulse-rate not out of proportion to the degree of temperature is a favorable prognostic symptom.

(d) *Albumin in the urine* from acute toxemia may be an early symptom of diffuse septic leptomeningitis.

(e) *Sugar in the urine* without an increase in the blood content occasionally occurs, especially with meningitis of the posterior fossa, in patients of metabolic instability.

2. **Symptoms of Cerebral Irritability.**—(a) *Sleeplessness and restlessness* are frequently among the earliest manifestations of general meningeal involvement. A patient who cannot sleep after a mastoid operation should always be regarded with suspicion—provided the wakefulness is not due to actual pain from the wound.

(b) *Excitability and irritability* are very characteristic of diffuse meningitis. In a general way they distinguish meningitis from sinus thrombophlebitis, as in the latter a composed demeanor and optimistic attitude are the rule.

(c) *Delirium* in general suppurative meningitis is apt to be wild and continuous, with intervals of lucidity. It is usually accompanied by a sharp piercing maniacal scream characteristic of the disease. It is easily differentiated from the somnolent dreamy state or mild toxic delirium so frequently present in brain abscess, and from the dreams associated with the high temperature of sinus thrombophlebitis.

<sup>1</sup> Eagleton, W. P.: Bulbar cisterna meningitis in relation to petrous caries, fifth nerve pain, and abductor paralysis, Trans. First International Otolaryngological Congress, Copenhagen, 1928.

(d) *Stiff Neck and Opisthotonos*.—Stiff neck is usually among the early manifestations of meningitis. However, the conception that there must be stiff neck before diagnosing meningitis should be abandoned. Both stiff neck and opisthotonos are due to irritation of the sensory and motor nerves as they leave the skull. They are present only when the base is involved, and meningitis of the base is often of a protective variety.

Meningitis limited to the cortex is not accompanied by stiff neck. The absence of marked stiff neck and opisthotonos is a good prognostic symptom, as it shows that the inflammatory septic process is not severe or diffused through the basal cisterna.

(e) *Suboccipital and general spinal tenderness* is generally present as soon as the process has become diffused. A few cases have been reported in which there was tenderness over the spine and occiput without rigidity of the head. When the exudate extends into the cord, there is generally spinal hyperesthesia and tenderness from involvement of the sensory roots.

(f) *Kernig's sign* is also a symptom of basilar involvement. It is among the earliest manifestations of general exudative meningitis, although the flexor rigidity may at first be slight.

(g) *The pain in the head (frontal or occipital)* of meningitis is agonizing, as a rule. It is usually associated with a peculiar sharp cry not present in brain abscess or sinus thrombophlebitis.

In uncomplicated thrombophlebitis there is no headache, and when complained of it is apt to be due to an associated meningitis, which, however, may only be protective in type.

Pain above the temple or limited to one or other of the branches of the trifacial, especially when limited to "behind the eye" of the homolateral side, is suggestive of a localized meningitis of floor of the middle fossa from disease of the cells at the apex of the petrous pyramid.

3. **Localizing Symptoms.**—*Cranial Nerve Symptoms.*—(a) *Anosmia*, loss of smell, should theoretically be an early sign of basal involvement, although up to the present it has not been observed. It should be routinely sought for, as it passes unnoticed by the patient.

(b) *Papilledema* is rarely present. There may be slight dilatation of the veins of the retina. When papilledema occurs it suggests an obstructive internal hydrocephalus.

(c) *Pupils.*—Unequal pupils are frequent in meningitis, but in the early stages they usually react to both light and accommodation, although they are apt to be "lazy." A widely dilated non-responsive pupil signifies involvement of the intrameningeal portion of the nerve from exudate in the basal cisterna.

(d) *Motor ocular* (third, fourth, and sixth) *paralysis* occurs in the terminal stages. The third nerve is the one most frequently involved early in the disease.

When meningitis originates from deep cell suppuration, Gradenigo's syndrome, viz., sixth nerve paralysis, with pain from irritation of the trigeminus, pain behind the eyes, the teeth, or the forehead, is apt to be among the first symptoms. Nystagmus is one of the usual terminal manifestations.

(e) *Seventh and Eighth Nerves.*—Facial paralysis and total deafness of one or both sides are frequent terminal symptoms, due to effusion in the internal auditory meatus.

**Auditory Nerve Involvement.**—Unilateral total deafness in meningitis is due to a postlabyrinthine neuritis from inflammatory exudate filling the auditory prolongation of the cisterna pontis. The vestibular portion of the eighth (non-acoustic) may or may not be completely functionless.

**Posterior Labyrinthine Vestibular Involvement.**—At times there is a cessation of reactivity of the vestibular nerve to the caloric (in the recumbent position, as well as in an upright position) without loss of hearing, due to effusion into the internal auditory canal.

(f) *Bulbar paralysis*, especially inability to swallow, usually precedes the fatal termination by a few hours.

**Terminal Symptoms.**—Convulsive seizures or sudden localized motor paralysis, when they appear early in fulminating meningitis, are due to hemorrhages in the pia-arachnoid. They are frequent in the terminal stage of exudative meningitis when the spaces of the pia-arachnoid of the cortex have become obliterated.

General cyanosis follows multiple septic infarctions of the lung, the result of a blood-stream infection.

## NEWARK EYE AND EAR INFIRMARY

(Cranial Surgery Department)

### GUIDE FOR DETAILED NEUROLOGICAL EXAMINATION

Surname.....Hospital Number.....  
Address.....Date of admission.....

The guide is designed to enable hospital interns or trained attendants without special training in neurology, to conduct a detailed examination, thus eliciting the details of objective and subjective symptoms which would otherwise be overlooked.

#### DIRECTIONS FOR USING GUIDE

The Neurological Examination is divided into: I. General Neurological Symptoms. II. General Neurological Examination. III. Cerebral Localization. IV. Examination of Cranial Nerves.

Suggestive terms with their possible interpretations, and methods for testing, as well as the normal reactions, are placed in brackets. *An asterisk (\*) in the margin indicates examination in unconscious patients.*

The general sections are indexed by roman numerals, the chief symptoms or manifestations by capital letters, and the individual details by alternate letters and numbers.

(At the completion of the examination all outstanding symptoms and abnormal findings are recorded under *Résumé of Abnormal Neurological Findings* on first p., followed by the index numbers and letters according to the guide, enabling a ready reference to the exact location of each detail. Example: Papilledema, IV, B-1. When a general symptom has a direct bearing on the localization it should be cross-indexed. Example: Dizziness, I. (D.) and IV (F-)2d, III (D) 1.)

(a) *Résumé of history:*

(b) *Résumé of outstanding symptoms:*

(c) *Résumé of abnormal neurological findings:*

.....  
Signature of House Surgeon

Date.....

## I. GENERAL EXAMINATION

Pulse:                      Temperature:                      Respiration:                      Blood-pressure:

\*(A) *Head:*

- \*1. Wounds, description of:
- \*2. Scars, description of:
- \*3. Bleeding from ear: right,                      left,
- \*4. Discharge from ear: right,                      left,
- \*5. Bleeding from nose: right,                      left,

\*(B) *Neck rigidity:*\*(C) *Extremities:*

- \*1. Paralysis or paresis of arm: right,                      left,
- \*2. Paralysis or paresis of leg: right,                      left,
- \*3. Spasticity of arm: right,                      left,
- \*4. Spasticity of leg: right,                      left,
- \*5. Anesthesia of arm: right,                      left,
- \*6. Anesthesia of leg: right,                      left,

\*(D) *Reflexes:*

- \*1. Superficial: Epigastric:
- Abdominal:
- Cremasteric:
- \*2. Deep: Knee:
- Ankle:
- Babinsky:
- Kernig:
- \*3. Sphincters: Loss of bladder control:
- Loss of bowel control:
- 4. Vasomotor: Abnormal flushing:
- Abnormal sweating:

## II. GENERAL NEUROLOGICAL SYMPTOMS

(A) *Headache:*

Severity (mild, intense, bursting, throbbing, lacerating):  
 Location (frontal, homolateral, temporal [ear], between temples [pituitary], behind the eyes [petrous pyramid], occipital, vertex [sphenoid]):  
 In attacks or constant:  
 Initial appearance, date:

(B) *Pain:*

Situation (ears, nose, eyes, arms, legs, etc.):  
 Duration:  
 Initial appearance, date:  
 In attacks or constant:

\*(C) *Vomiting:*

Frequency:  
 Type (projective or regurgitant):  
 Appearance, dates:

(D) *Dizziness:*

In attacks or constant:  
 Duration (momentary or continued):  
 Degree (transient swaying, rotation, falling):  
 Initial appearance:  
 Description of attacks (if patient or objects appear to move):

(E) *Chills:*

Initial appearance, date:  
 Shaking:                      Chilly feeling:  
 Number of chills:                      Dates:

\*(F) *Convulsions:*

General or localized?  
 Associated with unconsciousness?  
 Preceded by aura?  
 Visual (flashes of light, objects seen)?  
 Auditory (tones or music heard):  
 Description of aura:  
 During attack did head turn to right?  
     did head turn to left?  
     did eyes turn to right?  
     did eyes turn to left?  
     did arm twitch right?  
     did arm twitch left?  
     did leg twitch right?  
     did leg twitch left?

- \*(G) *Stupor*:  
Initial appearance:  
Duration:
- (H) *Numbness*:  
Location: face, arm, leg,  
In spells or constant:  
Initial appearance, date:
- (I) *Tingling*:  
Location of:  
At times or constant:  
Initial appearance, date:
- (J) *Disorders of vision*:  
Diplopia, constant or transient:  
Date of initial appearance:  
Failing vision:  
Complete or partial:  
Initial appearance, date:  
Constant or by spells: associated with rampart edge?  
Blindness:  
Initial appearance, date:  
In attacks or constant:
- (K) *Disorders of hearing*:  
Deafness:  
Initial appearance:  
Complete or partial:  
Tinnitus:  
Initial appearance:  
Constant or in attacks:  
\*Discharge:  
Initial appearance:  
With or without pain:

## III. CEREBRAL LOCALIZATION

- (A) *Frontal lobe*:  
1. Memory for:  
(a) Names (father's name; street where lives):  
(b) Events:  
2. Orientation (where is at present?):  
3. Affections:  
4. Aphasia:  
(a) For names:  
(b) Ability to repeat sentence after examiner:
- (B) *Temporal lobe*:  
1. Aphasia:  
(a) Nominal ability to recognize objects and state use, but inability to name ("knife") ("key"):  
(b) Ability to recognize proper name and ability to repeat name:  
2. Hemianopsia:  
(a) Right-sided: left-sided:  
(b) For form:  
(c) For colors:  
3. Uncinate gyrus symptoms:  
(a) Hallucinations of smell:  
(b) Hallucinations of hearing:  
4. Dreamy state:
- (C) *Parietal lobe*:  
1. Sensory symptoms:  
(a) Paresthesia (touch, pain, temperature):  
\*(b) Anesthesia:  
(c) Astereognosis:  
(d) Muscle sense:  
\*2. Motor symptoms:  
\*(a) Paralysis: Face:  
Complete (peripheral):  
Only on spontaneous expression (supranuclear):  
Arm:  
Leg:  
\*(a) Paresis:  
\*(b) Spasticity:  
\*(c) Convulsive seizures (Sec. 1-4):

(1) Preceded by aura: sight, smell, dizziness:

(2) Parts involved:

(d) Consciousness lost:

(D) *Occipital lobe*:

1. Hemianopsia: Right-sided: left-sided:

2. Visual hallucination: uncinate gyrus:  
Right-sided: left-sided:

(E) *Cerebellum*:

1. Dizziness:

\*2. Nystagmus: Rotatory, horizontal, vertical,  
To right, to left,

\*3. Lateral deviation: To right, to left,

4. Romberg:

5. Disturbances of gait:

6. Ataxia:

7. Adiadokokineses:

8. Co-ordination: With eyes closed place finger to nose:

Heel to knee:

Finger to finger:

#### IV. CRANIAL NERVES

(A) *First* ("olfactory"; smell):

Smell (perfume, vinegar): Right, left,

(B) *Second* ("ophthalmic"; sight):

\*1. Fundus examination: Right, left,  
Papilledema.  
Atrophy.  
Dilated vessels.  
Arterial disease.  
left,

2. Vision: Right,

3. Field:

(C) \**Third, fourth, and sixth* (ocular movements):

1. Pupils:

\* (a) Equal: unequal:

\* (b) Contracted:

\* (c) Dilated:

\* (d) React to light:

\* (e) React to accommodation:

2. External ocular movements:

\* (a) Diplopia: Convergent (paralysis abducens):

Right, left,

Divergent (paralysis 3d.):

Right, left,

\* (b) Spontaneous nystagmus to right: to left:

\* (c) Conjugate deviation to right: to left:

(D) *Fifth* ("trifacial," sensory, and motor):

1. Sensory portion:

(a) Anesthesia of supra-orbital:

\*(cornea):

(b) Neuralgia of infra-orbital:

Neuralgia of inferior dental:

(c) Taste in anterior two-thirds of tongue: (sugar, salt):

(Technic: Have tongue protruded; apply moistened salt or sugar. Patient answers by signs before withdrawing tongue.)

2. Motor portion:

(a) Buccinator: (movements of jaw):

(b) Masseter:

3. Vasomotor portion: (herpes of brow):

\*(E) *Seventh* (facial, motor, sensory):

\*1. Motor portion:

Paralysis or paresis: Right, left,

Brow:

Lower eyelid:

Mouth:

Cheek:

Palate:

Wrinkle forehead, smile, whistle, show teeth; goes up in midline.

2. Sensory portion (taste in anterior two-thirds of tongue). See D, (c).

3. Vasomotor: (herpes of ear):

(F) *Eighth* ("auditory" [hearing] and "vestibular" [equilibrium]):

1. Auditory portion:

(a) Deafness: Right, left,

- Hearing: Whispered voice, right, left,  
 Watch, right, left,
- (b) Tinnitus: Pulsating or continuous:
- (c) Discharge: Right left,
- (d) *Special auditory tests conducted by specialist:*
1. Tuning-fork:
  2. Weber referred to: affected ear:  
 Weber referred to: good ear:
  3. Bone conduction: (Schwabach):  
 Duration in seconds: right, left,
2. Vestibular portion:
- \* (a) Spontaneous nystagmus:  
 Horizontal rotatory:  
 In all positions or only in outer corner:  
 Horizontal:  
 Rotatory:  
 Vertical:
- (b) Spontaneous vertigo (dizziness):
- (c) Spontaneous pointing deviation (downward from shoulder):  
 Right arm to right. left arm to left.
- (d) Lateral propulsion:
- \*(G) *Ninth* ("glossopharyngeal," swallowing, sensory motor of base of tongue and fossæ):
1. Taste: See D, (c).
  - \*2. Swallowing:
- (H) *Tenth* ("pneumogastric," motor and sensory to voice and respiration; motor, pharynx, esophagus, heart, stomach).
1. Hoarseness: (Say "ah," uvula resets):  
 (a) *Laryngoscope examination by specialist:*
  - \*2. Rapidity of heart:
  - \*3. Cheyne-Stokes respiration:
- (I) *Eleventh* ("spinal accessory," motor):
1. Trapezius muscle:
  2. Sternocleidomastoid: } Shrug shoulders.
  3. Shoulder dropping: } Turn head.
- (J) *Twelfth* ("hypoglossal," motor):  
 Tongue protruded in midline. (Note deviation or tremor.)

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## THE GRADENIGO SYMPTOM-COMPLEX

In 1904 Gradenigo first described a new symptom-complex occurring in the course of a middle-ear suppuration. At that time he reported a series of 6 cases, 5 of them being his own. Subsequently in 1907 he reported a series of 57 cases. In 1910 Perkins collected 88 cases in literature and added 6, making a total of 94. Since then many additional cases have

been reported, so that this symptom-complex now rests upon a firm foundation of accurate clinical observations.

**Definition.**—Abducens or sixth nerve paralysis, associated with a suppurative otitis media and pain limited to the affected side.

The *syndrome* consists of:

1. A purulent otitis media, with or without mastoid involvement.
2. Severe pain referred to the temporal and parietal regions on the affected side and explained by Gradenigo as being due to an involvement of the Gasserian ganglion.
3. Paralysis or paresis of the sixth, or abducens, nerve on the same side.

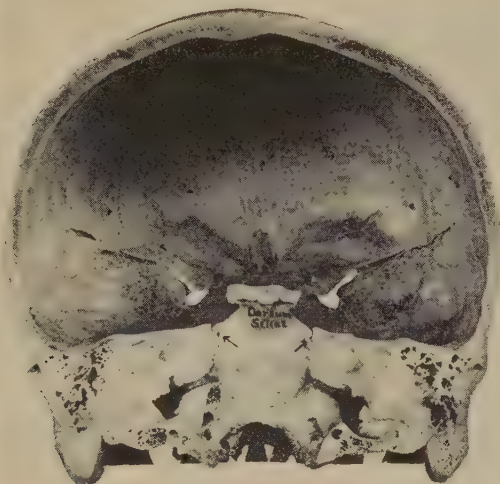


Fig. 332.—Vertical section through skull, posterior view. Arrows point to grooves at apices of petrous portion of temporal bones, in which the sixth cranial nerves lie. (Illustration loaned by Dr. John M. Wheeler.)

**Synonyms.**—The Gradenigo symptom-complex. Abducens paralysis, sixth nerve paralysis, accompanying a suppurating otitis media.

**Etiology.**—Gradenigo, who first called attention to this condition in 1904, ascribed it to “a circumscribed, simple, serous leptomeningitis, localized about the tip of the pyramid and caused by the diffusion of the infection in the tympanum.” He considered the infection to be “along pre-existing anatomical paths.”

Perkins describes these paths as follows:

1. “The infection may follow the sublabrynthine route, extending from the tympanum, below the labyrinth and internal auditory meatus to the mastoid tip.
2. “From the mastoid antrum it may extend through the subarcuate fossa, or petromastoid canal, which passes inward beneath the superior semicircular canal, and reaches a layer of cells sometimes lying above the internal auditory meatus, and thus arrive at the petrous tip.
3. “Or, this point may be arrived at by way of the carotid canal.
4. “Finally, the infection has been found in some autopsies to be through

a layer of cells extending along the Eustachian tube, thus passing from the tympanum to the petrous tip."

**Pathology.**—The paralysis is due to the passage of the sixth nerve through a minute canal (Dorello's) which is bridged over by the ligamentum petrosphenoidale (Gruber). Swelling, or edema, of this canal causes pressure and a consequent paralysis. The vulnerability of the sixth nerve

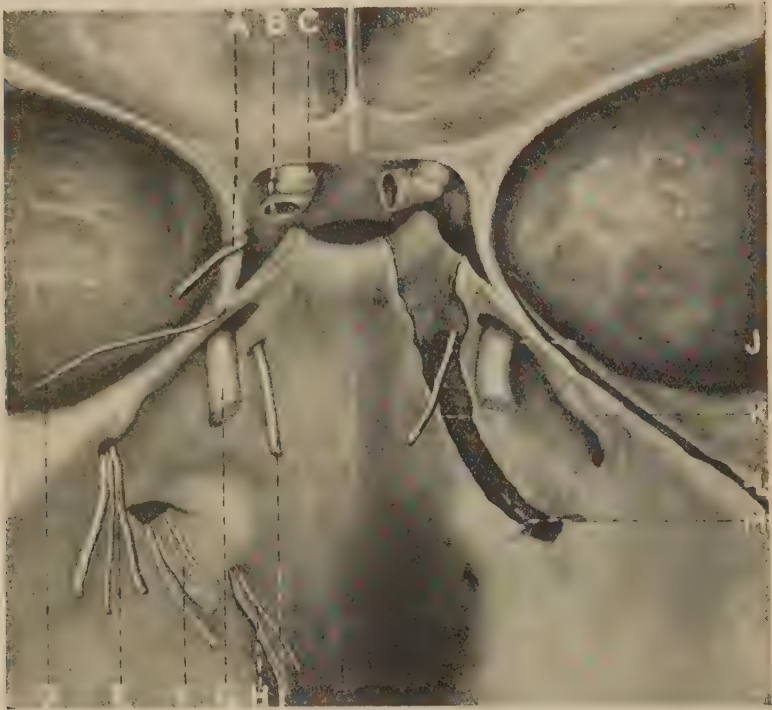


Fig. 333.—Drawing from wet specimen. The brain has been removed. On left side cranial nerves are seen entering dural openings. On right side dura has been removed to show sixth nerve entering connective tissue in posterior wall of cavernous sinus, a little below and behind apex of petrous pyramid. A, Third nerve; B, carotid artery; C, second cranial (optic) nerve; D, fourth nerve; E, seventh and eighth nerves; F, ninth, tenth, and eleventh nerves; G, fifth nerve; H, twelfth nerve; I, sixth nerve; J, fifth nerve entering Meckel's cavity for gasserian ganglion; K, sixth nerve entering connective tissue, dura has been removed; L, superior petrosal sinus; M, inferior petrosal sinus. (Illustration loaned by Dr. John M. Wheeler.)

is due to its exposed course, the basilar portion being approximately one inch in length.

The pain is due to the proximity to, and consequent involvement of, the Gasserian ganglion, the latter lying in a depression on the anterior surface of the apex of the petrous portion of the temporal bone.

**Symptoms.**—The essential symptom is, first of all, a suppurative otitis media, in the course of which there develops an abducens, or external rectus, paralysis on the side of the affected ear. This is accompanied by more or less severe pain limited to the same side of the head.

**Diagnosis.**—If, during the course of a suppurative otitis media, the patient complains of a diplopia, our suspicions should at once be aroused. In the earlier stages the paresis, or paralysis, may not be so evident, while

pain is not always so severe. Later the pain becomes a striking symptom and the paralysis is easily demonstrable.

Wheeler calls especial attention to the cases of abducens paralysis of otitic origin which may first of all come to the *ophthalmologist*.

The cases of toxic or syphilitic origin are so much more common that those of otitic origin are liable to be overlooked.

**Prognosis.**—Under prognosis and treatment Wheeler says, "The paralysis may clear up in a few days, and in a few cases has disappeared almost miraculously after a mastoid operation. Usually it persists for weeks and months, and there is a possibility of a permanent impairment of the nerve." As regards life there were 11 deaths in Perkins 94 collected cases, a mortality of almost 12 per cent.

**Treatment.**—Although there has previously been some diversity of opinion in regard to the advisability of operation, no doubt due to the recovery in many reported cases where no operation was performed, practically all authorities are agreed at present upon the performance of the *simple* mastoid operation in acutely discharging ears, and the radical operation where the otitic discharge has become chronic.

**Conclusions.**—1. The Gradenigo symptom-complex is of sufficiently frequent occurrence to deserve the attention of every *otologist*.

2. The *ophthalmologist* should be continuously on his guard to see that external rectus paralysis of otitic origin is not mistaken for the more frequent forms of abducens paralysis, with which he is more familiar.

3. A sixth nerve paralysis, associated with pain over the side of the head and a discharging ear, should have an early, if not an immediate, mastoid operation.

4. Although mastoid operation, with the free drainage which it affords, does not insure relief, it is the best means which we have and certainly lessens the dangers of intracranial involvement.



Fig. 334.—Further dissection to show sixth nerve entering cavernous sinus in relation to external wall of inferior petrosal sinus and in contact with apex of petrous pyramid: *A*, Posterior clinoid process; *B*, carotid artery; *C*, apex of petrous pyramid; *D*, sixth nerve; *E*, inferior petrosal sinus; *F*, ligament. (Illustration loaned by Dr. John M. Wheeler.)

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## SURGICAL COMPLICATIONS OF MIDDLE-EAR AND MASTOID SUPPURATIONS

The surgical treatment of complications of middle-ear and mastoid suppuration may be divided into two groups. First, cases which carry a menace of deeper extension, but are still external to the dura. This group includes necrosis in the cranial bones, infective labyrinthitis, and extradural abscesses. Second, cases where extension has taken place, the dura penetrated, and the brain coverings and tissue infected. The dura is a dense fibrous membrane, inelastic, insensitive to pain, and highly protective to the structures it encloses. Suppuration external to this membrane presents no special surgical difficulties, once the diagnosis is made.

When infection has passed the dura, and the brain and its coverings have become the seat of a suppurative process, attempts at relief involve conditions not met in other parts of the body. Accurate localization is seldom aided by examination of the outside of the skull as in soft tissues. The site of entering the skull must be chosen from clinical symptoms and neurological findings, frequently vague and contradictory. The approach is through a restricted field of hard dense bone, often a long and laborious procedure, with resulting jarring and shock to the nerve centers.

When the dura is opened, the pia-arachnoid spaces—highly sensitive to infection and trauma—are exposed. Through these spaces flows the cerebral fluid—the cerebrospinal circulation—bathing the entire brain surface. All manipulation within the brain must be carried on through some part of this area. To do this with a minimum of trauma and to protect these spaces from infection constitute the greatest obstacle to surgical success.

Infection of the brain structures is always followed by increase of the cerebral fluid and increased pressure. With normal cerebral pressure, the patient lying down, the cerebral fluid will rise in a glass tube to a height of 120 to 150 mm. Under stimulation of infection this pressure may be greatly increased. As the skull and dura are unyielding and brain tissue incompressible, this extra pressure is expended upon the venous sinuses, the capillaries, and the other blood-vessels, flattening the convolutions and obliterating the fissures. This interferes with the nutrition of the brain cells and, with the added trauma of pressure, lowers their vitality.

"Cerebral tissue, the seat of infection, does not tolerate manipulation and injury as well as normal cerebral tissue. The suppurative process with the associated compression so lowers its resistance that slight trauma may occasion extensive edema and encephalitis."<sup>1</sup>

The determination of the pathway of an infection to the brain materially aids in locating the operative field and indicating the nearest approach to the infected area. This infected area, whether superficial as a localized collection of cerebral fluid from a circumscribed meningitis, or more or less deep within the brain as an abscess, is generally in close proximity to the primary external infecting focus. Infection from the frontal sinus would be in the corresponding frontal lobe. When of otitic origin not only is it on the same side as the affected ear, but the point of penetration will determine its location above or below the tentorium. When it penetrates through the tympanic or mastoid vault the temporosphenoidal lobe or its coverings are infected. When it penetrates through the posterior

mastoid wall the cerebellum and sigmoid sinus are exposed to infection. The results of the invasion of the cranial cavity from the labyrinth are determined largely by anatomical factors within the bone. The cochlear and endolymphatic aqueducts, and the internal auditory canal, open on the posterior surface of the pyramid bone in close relation to the larger arachnoid spaces at the base of the brain and the anterior surface of the cerebellum. Infection through these channels more often results in meningitis, especially so if the infection passes through the internal auditory canal. Abscess when it occurs is generally in the anterior part of the cerebellar lobe. The small veins following the two aqueducts may occasion sinus thrombosis by way of the inferior petrosal sinus and jugular bulb.

Infection of the brain substance through the dura may be by direct extension to the cortex or by a small vessel to the deeper brain tissue. A cortical infection may cause a deep abscess by secondarily involving a blood-vessel entering the brain from this point. As the vessels in the brain substance run nearly perpendicular to the surface, the abscess would be in this relation to the cortical lesion.

The pathological result of the direct extension of an infection to the cortex will be determined largely by: (a) Nature's reaction to the invasion and ability to wall-off the infected area and limit its spread; (b) the size of the arachnoid spaces involved. If the invasion occurs near or at the base of the brain, or over a fissure where the cerebral fluid is abundant, the infection would be carried into the general cerebral fluid circulation, resulting in a diffused meningeal infection. When the infection passes to the brain surface where the arachnoid spaces have thinned out, the chances of the infection becoming circumscribed are greatly increased.

**Lesions Resulting from Infection.**—The complications resulting from cerebral infection are arranged in three groups:

1. Infections involving the veins and sinuses, producing sinus thrombosis.
2. Infections involving the coverings of the brain, resulting in infectious meningitis.
3. Infections involving the substance of the brain with resulting necrosis, causing brain abscess and encephalitis.

#### SURGICAL DRAINAGE OF THE LABYRINTH

The object of labyrinth operations is the removal of infection from within the labyrinth, and thus prevent its spread to the meninges with a resulting meningitis or abscess. Judging from society discussion and the literature, the trend in recent years has been toward marked conservatism in opening the labyrinth. It was early recognized that a fistula in the horizontal canal, uncovered during a radical mastoid operation with no other symptoms from the labyrinth except vertigo, did not constitute an indication for invasion of the labyrinth, and that better results followed when the fistula was not even curetted or probed, as Nature had already blocked it off from the vestibule.

Later experience would seem to indicate that in acute suppurative labyrinthitis drainage has been disappointing as a preventive of meningitis; that in active inflammatory processes meningeal extension has already taken place before operative measures could have been instituted. In case the process was circumscribed the operative breaking of protective

barriers may open up pathways to a fatal meningitis. The best operative results are obtained after the subsidence of the acute condition.

The vestibule is the vulnerable part of the labyrinth, its inner wall being in close relation to the internal auditory canal and the cochlea. The primary object of all labyrinth operations is the free opening of the vestibule for drainage. The small semicircular canals have no free pathways to the cranial cavity, but open freely into the vestibule. Their exposure is not for the purpose of drainage, but for a guide in entering the vestibule behind the facial nerve. The cochlea has many connections with the dural spaces through the channels for the passage of vessels and nerves. Any attempts at extensive exposure of the whorls will probably result in fracture of the modiolus at the base, its weakest point, with escape of spinal fluid and the opening of direct connection to the meninges (Fig. 335). The opening of the first whorl usually gives satisfactory drainage.

A **radical mastoid** is a necessary preliminary to any attack upon the labyrinth. To obtain as wide a field as possible, free removal of bone is necessary. The posterior meatal wall is cut down to the lowest surgical limit of safety. The hump in the external canal is lowered and the anterior wall of the attic removed.



Fig. 335. — The modiolus, the base of which is liable to fracture by injudicious chiseling of the cochlea shell. (From Phillips, *Diseases of the Ear, Nose, and Throat*, F. A. Davis Co., publishers.)

**Surgical Landmarks.**—There should now be brought into view the oval window, the niche of the round window, the horizontal portion of the facial canal, and the prominence of the anterior half of the horizontal canal. Posterior to and slightly above the horizontal canal is the “solid angle,” an area of bone enclosed within the plane of the horizontal and two vertical canals.

There are four especial dangers encountered in opening the labyrinth. First, injury to the facial nerve while endeavoring to open drainage both internal to and below this nerve. The horizontal part of the nerve is 3 mm. above the upper margin of the oval window, and in close relation to the anterior half of the horizontal semicircular canal directly above it. Great care must be taken in opening this part of the canal because of the danger of a transverse fracture of the facial canal. Second, fracture of the modiolus, from too extensive operation on the cochlea. Third, injury to the dura from attempts to curet within the vestibule. Fourth, injury to the jugular bulb and carotid artery from abnormal positions. This last should be easily avoided by careful work.

**The Jansen Operation.**<sup>2</sup>—The upper half of the anterior portion of the horizontal canal is removed, using a small chisel or gouge. It is important to use the chisel as a plane to take off shaving after shaving. With care it is possible to preserve the lower half of the anterior crus. This establishes a guide below which one cannot go without injury to the facial canal. The posterior portion of the horizontal canal, behind the downward curve of the facial nerve, is wholly removed. Two routes are now available: (a) Following the anterior crus forward, above and internal to the facial nerve, to the ampullar end of the canal, which enters the roof of the vestibule above the fenestra ovalis. The possibility of fracture of the facial canal is present unless due care is used. (b) The posterior crus of the horizontal

canal can be followed to its non-ampullated end on the lateral wall of the vestibule, and the opening enlarged upward and outward to the ampulla. This procedure does not endanger the facial nerve. The stapes is then removed, ending the operation. The fenestra ovalis is not enlarged unless there is bone necrosis on its edge or the exposure of the cochlea is indicated.

**The Hinsberg Operation.**—On account of the possibility of losing the landmarks by filling of the small lumen of the canal with bone chips or dust if a burr is used, Hinsberg advises beginning the operation with enlarging the fenestra ovalis by removal of the bone between the fenestra ovalis and rotunda. With a small curet or burr the window is further enlarged by the removal of the outer shell of the promontory forward and downward. This opens the lower whorl of the cochlea (Fig. 336). A small probe, bent at a right angle, is introduced through this opening, and the level of the roof and depth of the vestibule is ascertained. The vestibule outlined, the next step is the opening of the ampulla of the horizontal canal



Fig. 336.—Hinsberg's labyrinthine operation. (From Kerrison, *Diseases of the Ear*, J. B. Lippincott Co., Publishers.)

above the fenestra ovalis, and above and internal to the knee of the facial ridge. This is accomplished by the removal of the spongy bone above the ridge of the horizontal canal. This can be done with a small, straight, sharp chisel, removing thin layers of bone. This is continued until the upper half of the anterior crus of the horizontal canal is removed, as in the Jansen operation, and the ampulla opened. Kerrison has devised a special curet for this purpose<sup>3</sup> that diminishes the danger of this procedure. The canal opened, a dental canal probe can be passed through it and the vestibule roof definitely located, which is then perforated with a small chisel or burr and enlarged downward and backward. Judgment must be exercised in the use of the curet for the removal of granulations and necrotic tissue within the vestibule.

**Richard's Operation.**—This has for its object the exposure of the entire labyrinth. The horizontal canal is first exposed, then the two verticals are uncapped. The vestibule is entered through the "solid angle," a depression formed by the junction of the three canals. This opening is enlarged by the removal of the greater portion of the posterior wall and roof of the vestibule. The cochlear whorls are uncovered by first exposing

the lower, then the apex, and the intervening shell between the two openings is carefully removed. This operation when completed is a beautiful piece of work, but it should be undertaken only by artists in otology.

**Jansen-Neumann operation** differs from the intratympanic operations in that the approach to the vestibule is from the posterior fossa. The radical mastoid completed, the sigmoid sinus and the dura over that part of the cerebellum internal to the sinus are separated from the pyramid bone. The dura is then elevated over the posterior surface of the pyramid bone to the depth of  $\frac{3}{4}$  of an inch or more, care being taken that it is not torn where the endolymphatic sac lies in a depression on this surface. A thin spatula is used gently to retract the cerebellum from the pyramid bone and to protect the dura from injury (Fig. 337). If the dura over the

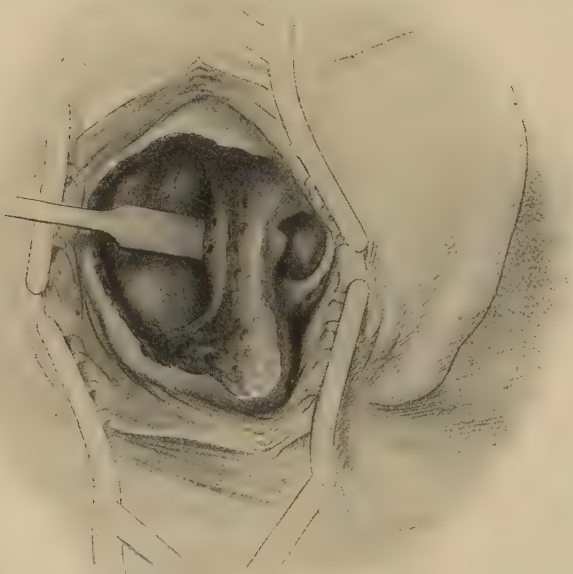


Fig. 337.—Jansen-Neumann operation. Sinus and cerebellar dura retracted. Cross-section of the posterior vertical canal, showing openings of upper and lower limb. (From Loeb, Nose, Throat, and Ear, C. V. Mosby Co., Publishers.)

cerebellum is tense a lumbar puncture should be done to gain working space. The posterior wall of the pyramid is cut away parallel to its surface, using a sharp chisel or gouge, and avoiding the superior petrosal sinus that runs along its upper edge and the deep portion of the sigmoid sinus below. As successive thin layers of bone are removed the bone is closely observed for the opening of the posterior vertical canal which, when the arch is uncapped, will appear as a groove. The next section of bone removed, the groove is replaced by two round openings, one nearly above the other, being cross-sections of the upper and lower arms of the posterior vertical canal. Continuing the bone removal, the arch of the posterior limb of the horizontal canal appears. When this is sectioned a third orifice is seen midway between and external to those of the posterior vertical canal (Fig. 338).



Fig. 338.—Jansen-Neumann operation. Openings of the upper and lower line of the posterior vertical canal and the posterior limb of the horizontal canal. (From Loeb, *Nose, Throat, and Ear*, C. V. Mosby Co., Publishers.)



Fig. 339.—Jansen-Neumann operation. Posterior wall of the vestibule removed. (From Loeb, *Nose, Throat, and Ear*, C. V. Mosby Co., Publishers.)

This is a cross-section of the posterior part of the horizontal canal which enters the vestibule at its lateral wall. The position of the vestibule can now be ascertained by its location behind and above the foramen ovale and a fine probe introduced into the horizontal canal and the openings of the posterior canals. The center of approach is in the area embraced between the three orifices. The vestibule exposed, the opening is enlarged by removal of the posterior wall of the vestibule (Fig. 339). The foramen ovale is enlarged and the first whorl of the cochlea exposed as in the Hinsberg method.

**Comparisons of the Different Operations.**—The approach to the vestibule in the Jansen and Hinsberg operations is practically the same, the difference being in the treatment of the oval window and promontory. Jansen does not take advantage of this for freer drainage. Hinsberg begins his operation with enlargement of the foramen ovale, and exposing the first whorl of the cochlea, and can thus outline the vestibule before approaching the vestibule from above. It affords good drainage for the labyrinth and the technic is not difficult.

*Richards operation* gives extensive exposure of the canals, vestibule, and cochlea. It is a difficult operation, with increased liability of injury to the facial nerve and of opening the dural spaces at the internal auditory canal.

*Neumann's operation* has the advantages of increased operative space; the opportunity to inspect the posterior surface of the pyramid bone for inflammatory changes or localized abscesses; and the greater safety of the facial nerve from injury. Its disadvantages are the chance for accidental tearing of the dura, and the fact that with a high jugular bulb, or the sigmoid sinus located well forward, it is impracticable.

### SINUS THROMBOSIS

The **prognosis** in infective sinus thrombosis is favorable when operative measures are undertaken in the early stage. The sinus, passing over the brain surface between the layers of the dura, is easily accessible, and no exposure of the meninges is required in opening the vessel to remove the clot.

**Treatment of Lateral Sinus Thrombosis.**—If conditions found at the mastoid operation, or subsequent symptoms, indicate a probable sinus infection, the sinus should be uncovered for inspection. There seems to be a strong reluctance with many otologists to exposing the sinus for diagnostic purposes. The procedure is not difficult or dangerous. We cannot too strongly emphasize that it shows better judgment to establish a diagnosis of thrombosis early on moderate symptoms and an exploratory opening, than to wait for strong positive symptoms, only to find that the infection has been already carried to other organs.

*Preparation.*—If, before opening the mastoid, sinus involvement is suspected, attention to preliminary details will shorten the operative time, and give clearer results. Separate trays of instruments should be prepared for the mastoid, sinus, and jugular. The patient is prepared before going to the operating room. The field is sterilized over the mastoid, sinus, and neck, and protective dressings applied to each region separately. Each area can thus be dealt with independently without loss of time. The surgeon's and assistant's gowns, as well as the instruments, should be changed between each step of the operation.

The mastoid operation completed, the cavity, copiously flushed with

normal salt solution, should be packed with gauze strips saturated with alcohol, to remain until the space is needed for the deep sinus exposures.

The *technic of approaching the sinus* will depend on conditions found in the mastoid. The position of the sinus is subject to marked variations in relation to the mastoid cavity, often situated beneath its floor. If the bony wall is thin the exposure can be made with a sharp curet or small rongeur. I have found no instrument as useful for this bone work as the small Stille bone forceps. When the sinus is found exposed at the mastoid operation, it offers an inviting point from which to commence the removal

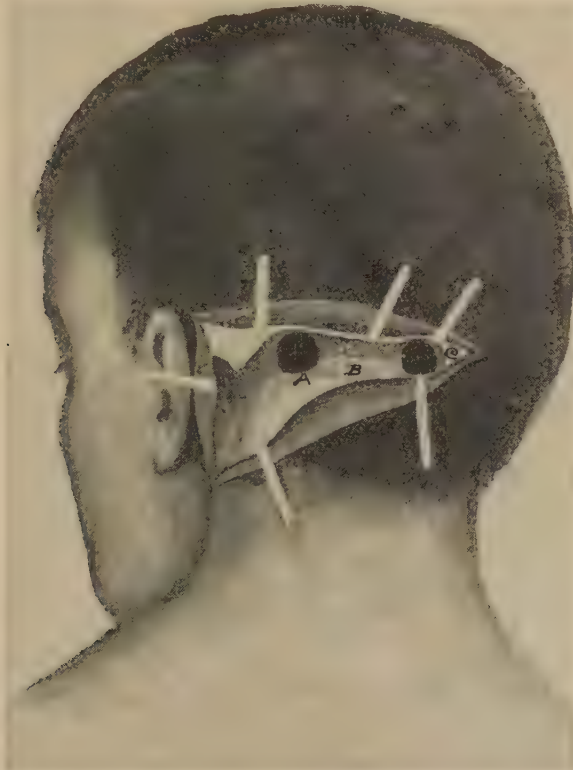


Fig. 340.—Showing rapid method of exposing the lateral sinus. A, Trephine opening behind knee of cervix. B, Groove through outer table. C, Trephine opening for opening for extensive thrombus backward.

of the sinus wall. This must, however, be undertaken with caution. The sinus wall is cemented to the groove by the inflammatory process and is soft, friable, and easily torn at this point.

Where the bone is intact and of definite thickness, much time may be consumed in cautious bone removal, expecting each stroke to uncover the sinus and fearing to perforate its wall. To obviate this the following procedure has the advantage of rapidity and safety. The soft parts over the lateral portion of the sinus are incised and bleeding controlled, a hasty search is made for the emissary vein, the condition of which often gives valuable information. With a Hudson drill the skull is now perforated  $\frac{1}{2}$  inch behind the location of the knee, and the sinus exposed (Fig. 340).

The upper level of the knee can be located by a line drawn from the upper attachment of the auricle to the occipital protuberance. We can now rongeur rapidly forward to the knee, uncovering the sinus, working parallel to the vein and toward the mastoid cavity. The bone is usually too thick for easy biting; a wide groove cut through the outer table, with a large gouge, the gouge being held a little less than parallel to the skull and the groove wider than the width of the sinus, saves time and danger of tearing the wall (Fig. 340). The knee being uncovered, the sigmoid portion is quickly exposed working from the sinus into the mastoid.

*Tearing of the sinus wall* may occur during bone removal, and is wrongly ascribed to nipping the wall between the blades of the rongeur. This accident, I believe from experience, is caused by the operator, when the bone does not bite readily, using the forceps handles as a lever, thus partly breaking the bone loose, the rough edges penetrating the sinus wall from the downward tilting movement. This accident can be avoided if the bone is first thinned by the groove made through the outer table which permits clean cutting by the forceps. Tearing of the wall, when it does occur, is embarrassing, but should not cause the abandonment of our search. A small pad placed over the tear will control the bleeding, and can be held in place a short time by an assistant, while the operator proceeds. The whole exposure should ordinarily be done in a few minutes, and for diagnosis should be at least 1 inch in length.

*Inspection of the Sinus.*—The sinus wall is now carefully examined for changes in color, thickness, or necrotic spots. When there is any departure from normal, inspection of the sinus gives little reliable information, and its interior must be examined. We make one exception to this rule. When operating on a mastoid, and the sinus is found exposed through a necrosis of the inner mastoid wall and covered with granulations, in the absence of clinical symptoms, it is not molested. Experience shows that the granulations on the outer surface may have thus far been a protection to the interior. The opening in the bone should be enlarged until healthy sinus wall and dura are uncovered. Close observation of the patient should follow with blood examinations and bi-hourly temperature records.

*Opening Sinus for Diagnosis.*—When in doubt the interior of the sinus should be examined. It is without risk if negative, without damage to the function of the sinus, and the bleeding is negligible.

The use of the needle and punctures are unreliable and, moreover, will not show a mural clot, and their use should be discarded. An incision through the sinus wall in the descending portion, the most frequent location of a beginning thrombosis,  $\frac{1}{4}$  inch or more in length, made with a paracentesis knife, *layer by layer*, is most satisfactory. When the intima is reached and before cutting through, it can generally be demonstrated if a clot is adherent to the wall. On opening the intima, with an obstructive clot beneath, no bleeding occurs; with a thin mural deposit, the clot will present in the opening before rupturing. Loss of blood is immediately controlled by placing the finger over the opening while pressure is made on both the distal and proximal ends of the exposed sinus. This is done with two short cylinders of gauze tightly rolled, held with artery forceps (Fig. 342). If the whole width of the sinus has been exposed this pressure properly and firmly applied will control bleeding and allow an examination of the interior walls. If a clot is not present, the alternate releasing of the pressure,

first above and then below the opening, will show whether the sinus is free or obstructed beyond the opening, by the rate of the blood flow. A free flow of blood is usually diagnostic of a free sinus. A source of error will be present when a partial clot exists in the jugular end or the bulb, causing a reversal of the blood-stream from the inferior petrosal and vertebral veins. This condition cannot be diagnosed at this time. A primary clot posterior to the entrance of the mastoid vein would be so rare that it can be disregarded. If no thrombus is present, a small pad placed over the opening will control all hemorrhage, permit the sinus to continue its function, and

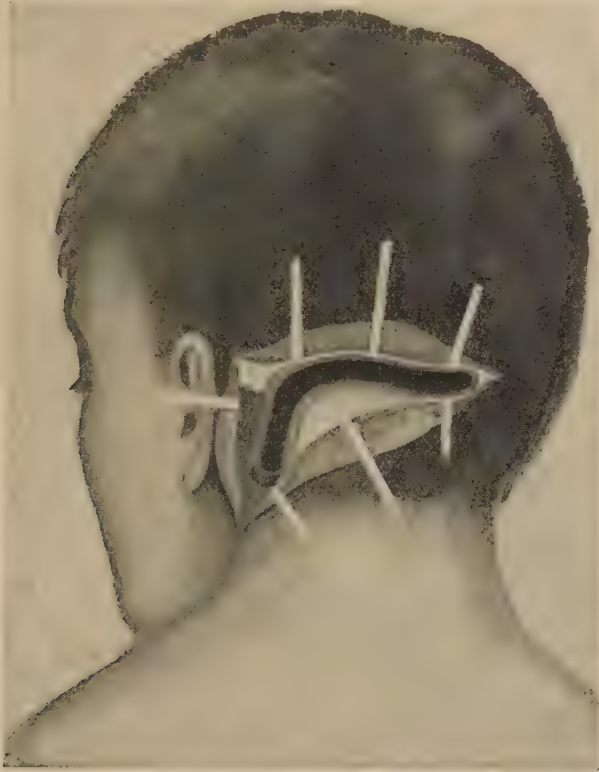


Fig. 341.—Complete exposure of sinus. Outer sinus wall removed.

the opening through the vessel wall will quickly repair. Both sinuses can be explored in this manner at the same operation without harmful effects.

A *positive diagnosis of thrombus* being demonstrated, the exposure should be now continued posteriorly until healthy sinus wall is uncovered, and then well down toward the bulb. Exposure of the bulbar end is the most trying part on account of the confined space and difficult angle. The extent of the thrombus determined, the manner of handling the clot must now be decided.

Specific rules cannot be laid down. The treatment must be modified to meet the condition present. If healthy sinus has been exposed toward the torcular end, a plug of gauze is introduced between the bone and sinus to obliterate its lumen and prevent bleeding when the clot is removed. This control of primary hemorrhage is most important. The amount of

blood that can be lost in a few moments is great, and this sudden withdrawal of the blood from the brain can only produce increased shock.

If, however, healthy wall has not been exposed posteriorly, it is better to make a second opening with the Hudson drill well toward the torcular end of the sinus (Fig. 340). If the wall is normal in this opening, it makes an excellent point to obliterate the sinus; this obliterating plug can be left *in situ* for several dressings, until we are satisfied an organized clot is formed.<sup>4</sup> If we find a thrombosed condition beneath this second trephine opening, we are reasonably sure the disease extends to the torcular; in which case the bridge of bone anterior to the second trephine opening must be cut away. The posterior limits of the thrombus having been outlined, the outer sinus wall is opened from our exploratory incision, posteriorly

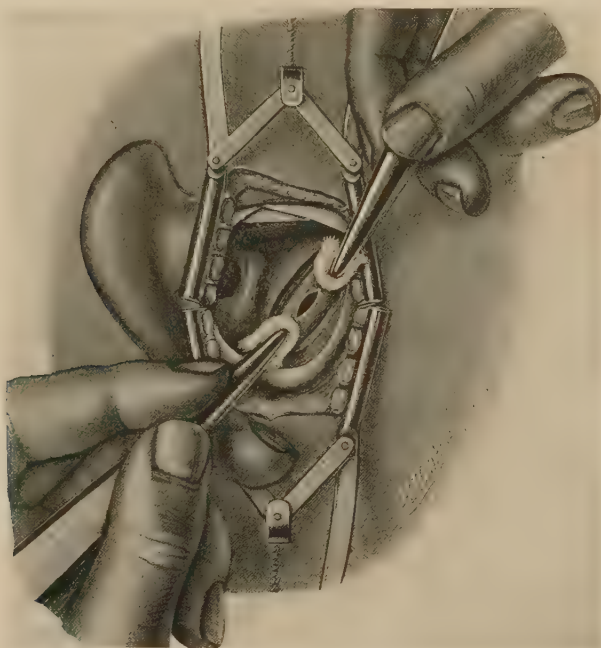


Fig. 342.—Method of controlling hemorrhage in testing for clot above or below opening in sinus. (Barnhill and Wales.)

to the limits of the clot, with a pair of small dural scissors, and the outer loose edges of the wall cut away. The clot now lies exposed as in a trough and can be removed with a large size curet without danger to the inner wall (Fig. 341).

With the compression plug in position in the second trephine opening, the portion of the sinus beneath the bridge of bone, if not cut away, can be emptied with a dull curet or by suction, bearing in mind that the inner wall is equally diseased as the outer, and that a perforation opens an entrance to the meninges.

*The Use of Suction.*—In our clinic the use of suction has displaced the curet as more effective and safer in removing thrombi from the jugular bulb and all unopened parts of the sinus. A flexible rubber tube, smaller in diameter than the lumen of the sinus, is connected to an electrical suction

pump. The end of this tube introduced into the sinus will from the negative pressure attach the clot firmly to the end of the tube and, if the clot is firm, permit of its complete withdrawal. When the thrombus is disintegrating, the sinus can be thoroughly emptied of its contents.

*Indications for Exposing the Jugular Vein.*—Thus far the lower sigmoid portion of the vein has not been touched. When the appearance of the sinus wall indicates that the thrombus approaches the bulb, or changes in the sinus wall are present, it is conservative to suppose that a mural clot or infected intima is present in the jugular bulb, and to proceed to isolate this region by ligation or resection of the jugular vein. Thus we hope to confine the infection between our torcular plug and the closed jugular.

It is not justifiable to carry out any manipulations around the bulb with an open jugular vein. Whether a jugular should be ligated or resected is a much debated question; both have their strong advocates. Statistics seem to indicate equally good results from both. Personally we have thought conditions called for resection in but three of our cases.

Ligation is quickly and easily performed through a small wound. Resection is a much more extensive operation, requiring time, and careful dissection. Our plan has always been to expose the vein as for ligation, and if the appearance of the vessel is good and no thrombus present, to ligate. If, however, the walls appear much damaged or a clot is present, the resection is called for.

*Ligation of the Jugular.*—The point of election for ligation is in the upper carotid triangle on a level with the thyroid cartilage, above the facial vein. There are two ways of making the skin incision for this exposure. The one usually used is a longitudinal incision,  $1\frac{1}{2}$  inches long, over the anterior border of the sternocleidomastoid muscle. The neck is made prominent by a small sand-bag under the shoulders. The second approach is by a transverse incision, following the most marked skin fold.<sup>5</sup> This should be well marked with a scalpel, with the chin in the median line, before placing the sand-bag. The incision is carried through the skin and platysma. With the use of retractors this gives good working space, and the resulting scar is hidden. With either the vertical or transverse incision after the exposure of the sternocleidomastoid muscle the technic is the same. Curved Mayo scissors is inserted just in front of the sternocleidomastoid muscle, and by blunt dissection the carotid sheath is uncovered, which is opened, and the jugular exposed and the facial vein located. If the vein is patent, a double chromicized catgut ligature is passed around it above the facial and tied, and the blood expressed before tying the second a short distance below. Nothing is gained by cutting the vein between the ligatures, and it exposes the neck wound to infection if the intima is contaminated. The wound is closed with metal clips and a neck dressing applied.

*Indications for Jugular Resection.*—If, however, the jugular is thrombosed or evident phlebitis present, the wound is freely extended upward behind the angle of the jaw and downward to the clavicle. The lower limit reached, the vein is ligated by a double ligature at least  $\frac{1}{2}$  inch apart and all contributing veins tied off with double ligatures. The upper jugular end is now doubly ligated as high as possible. Before severing the vein, a strip of gauze is placed beneath it to catch any drip that might infect the

wound, and the severed ends immediately sterilized with pure carbolic acid and alcohol. This technic is carried out on all contributory veins until the jugular is completely removed (Fig. 343). Several strands of silkworm-gut are twisted together and laid in the bottom of the wound, coming out at the lower angle, and the wound closed with metal clips. With no evidence of infection, this drain is removed about the third day.

We now return to the deep bulbar end of the sinus. The wall is slit toward the bulb and the clot removed. If this is followed by bleeding from the inferior petrosal veins, the sinus is plugged with gauze.

*Emptying the Jugular Bulb.*—A dissection of the region of the jugular bulb shows that the mechanical obstacles to be overcome in curetting this

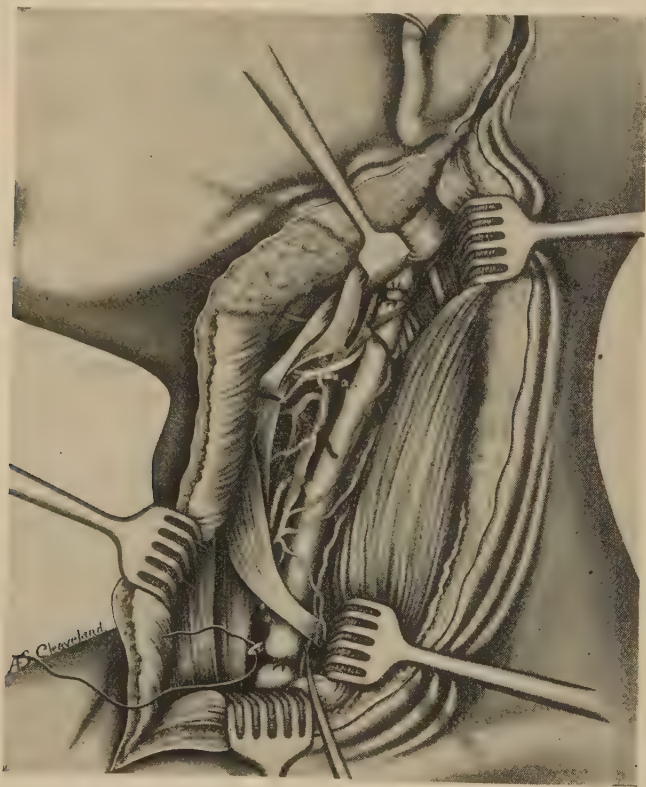


Fig. 343.—Resection of internal jugular vein for thrombosis.

part of the vein are always great, and often insuperable.<sup>6</sup> Attempts to curet the bulb only result in clearing the deeper end of the sigmoid sinus. The use of suction, referred to before, has been most satisfactory in removing large clots, which apparently filled the bulb, from this most difficult location. If the clot is firmly adherent to the walls, suction may fail; but this would indicate that organization of the clot, and obliteration of the space, were taking place. By the use of suction, the jugular bulb can be emptied quickly and safely in a large proportion of the cases, and renders unnecessary a more extended operation for this purpose.

*Closure of Wound.*—A catgut drain of several twisted strands is intro-

duced into the bulb, and the whole field filled with iodoform gauze. Compression plugs introduced between the bone and the sinus to control hemorrhage should have attached a free end of black ligature for identification. Avoid the use of short pieces of gauze pushed under the bone. They might be overlooked at subsequent dressings, with the formation of granulations and bleeding. The scalp flaps are drawn together over the dressing, in the lateral portion of the sinus, and held together with three or four metal clips. This prevents shrinking of the flaps, and lessens the space to be filled by granulations, with correspondingly less final cicatrix. Drainage is not interfered with by this closure.

*At the first dressing*, usually the fourth to sixth day, the incision can be torn open to remove the compression plug, if this has not been introduced in a more posterior trephine opening. If the plug has been used, the gauze is removed from the sinus region without disturbing the flaps, and new gauze inserted in the resulting tunnel. The flaps should be reunited if opened. It not infrequently happens that bleeding will occur on attempting to remove the compressing plug, and it may be that it cannot be dispensed with until after several dressings.

The infection which was confined in a closed tube has now been mechanically blocked off, and a larger part laid open and its contents evacuated. The closed upper part of the jugular vein, left after ligation or resection, has fair drainage to the bulb, which is the most dependent point with the patient in the recumbent position.

The *complications* that follow will be mainly caused by infection that has invaded the blood-stream. The most common and least dangerous is an acute arthritis. More dangerous are meningitis, endocarditis, and septic pneumonia.

A continuation of the septic symptoms suggests metastasis to other locations. Acute arthritis is most common, one or more joints becoming swollen and tender. Areas of central pneumonia or lung abscess are frequent sequelæ and difficult of diagnosis. Cerebral involvement is rarely of metastatic origin, but usually due to direct extension by thrombosis of the small veins emptying into the sinus. In a recurrence of high temperature following a marked improvement after operation, the local field should be examined and the neck wound reopened on slight indications. The possibility of an extension of the thrombus to the torcular must be kept in mind. This can be determined by a trephine opening over the sinus near the occipital protuberance.

The *transfusion* of properly typed blood—repeated as necessary—has been successful in many reported cases in combating the bacteriemia. The intravenous injections of mercury preparations are of doubtful value.

*Successful results* in the treatment of lateral sinus thrombosis will be inversely proportionate to the delay in making the diagnosis and instituting the surgical remedy.

**Cavernous Sinus Thrombosis.**—Spontaneous recoveries from this condition, though few, would seem to be relatively more frequent than from thrombosis of the lateral sinus. On account of its anatomical location, no surgical technic has given a sufficient number of favorable reports to result in its being generally followed.

Operations on the cavernous sinus must be done early before extension to the opposite sinus has taken place. The two sinuses are closely con-

nected by the circular and transverse sinuses and collectively form a venous plexus.

Ballance<sup>7</sup> recommends approaching the cavernous sinus by the Hartley-Kraus method for exposing the gasserian ganglion in the middle fossa. An osteoplastic flap is not necessary as the temporal fascia is so thick and tense that with careful repair ample protection is secured. The middle meningeal artery will require ligating, and venous bleeding, mostly from the diploë, may be troublesome. The sinus is exposed by elevating the dura and lifting up the brain. Under good reflected light the cavernous sinus or plexus can be seen anterior and internal to the position of the gasserian ganglion. The first and second branches of the fifth nerve are

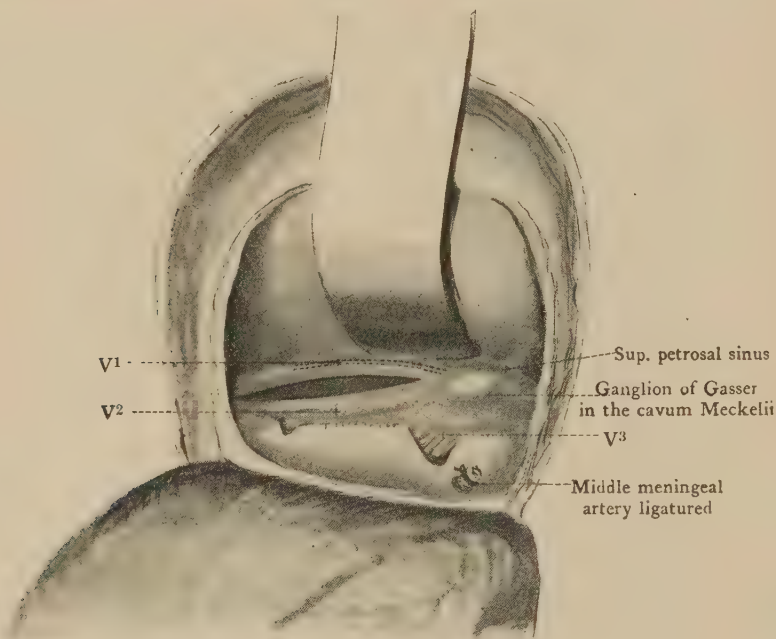


Fig. 344.—Exposure of left cavernous sinus by the Hartley-Krause method. (From Ballance, *Surgery of the Temporal Bone*, Macmillan & Co., Ltd., Publishers.)

infolded in its wall, and they can be used as a guide to incise the sinus between them (Fig. 344).

The *orbital approach to the cavernous sinus*, recommended by Mosher, does not seem to have been given the consideration that it merits.<sup>8</sup>

*Technic.*—The globe of the eye is removed, the orbital cavity cleaned out and the ophthalmic artery tied. The periosteum is cleaned from the posterior half of the floor of the orbit and the groove for the superior maxillary nerve recognized (Fig. 345). The periosteum from the orbital surface of the great wing of the sphenoid is elevated and the outer end of the sphenoidal fissure located. With a chisel a vertical cut is made through the orbital plate of the great wing of the sphenoid from the notch for the superior maxillary nerve below, to the outer end of the sphenoidal fissure above. The bone is thin along this line and easily removed. The bone

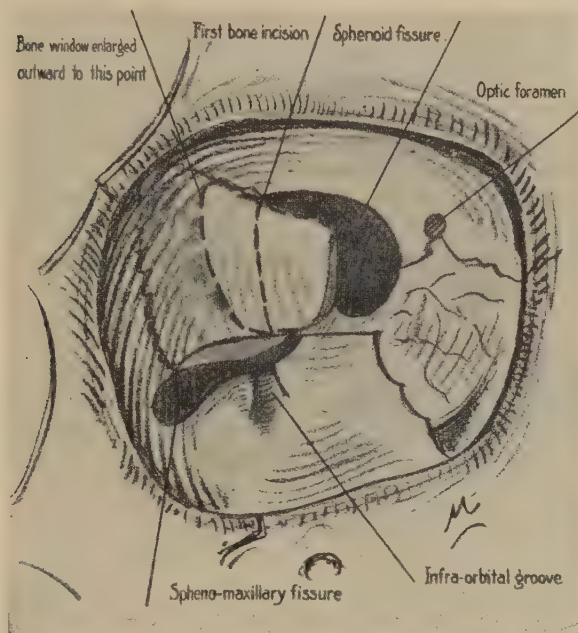


Fig. 345.—Showing the bone incisions for reaching the cavernous sinus through the orbit. (Courtesy of Dr. Harris P. Mosher.)

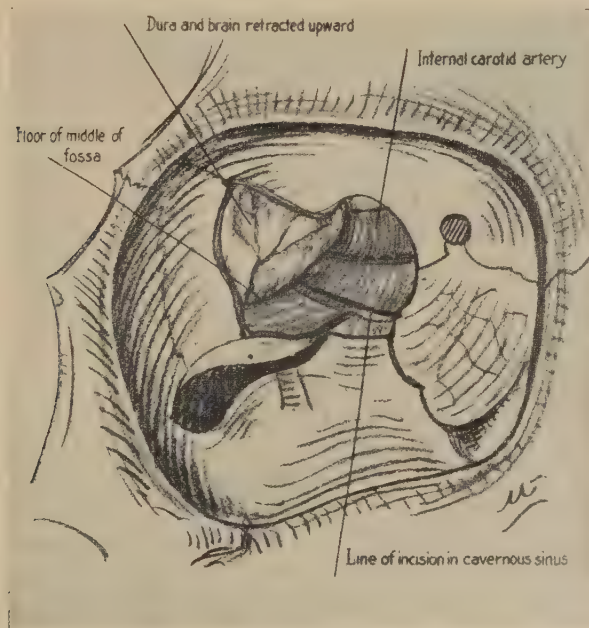


Fig. 346.—Showing the cavernous sinus exposed and incised. (Courtesy of Dr. Harris P. Mosher.)

opening is enlarged outward  $\frac{1}{2}$  cm., and downward flush with the floor of the orbit (Fig. 346). The dura is elevated from the floor of the middle

fossa, working from the outer border of the bone window inward. The dura can be separated from the outer wall of the cavernous sinus for about 1 cm.; beyond this point a pinhole perforation of the sinus will occur from an entering vein. A blunt-pointed knife is placed against the outer wall of the sinus on a level with the floor of the orbit and the knife-blade carried inward toward the body of the sphenoid until stopped by the bone. The incision opens the wall of the sinus for 1 cm. and is well below the internal carotid artery. Through this incision a small curet can be carried to the openings of the superior and inferior petrosal sinuses; or its contents evacuated by the use of the suction pump.

The drawback to this procedure is that it necessitates the removal of an eye.

#### LUMBAR PUNCTURE

Lumbar puncture is done: (a) To obtain cerebrospinal fluid for examination; (b) to measure intracranial pressure; (c) to relieve excessive pressure by the withdrawal of cerebrospinal fluid; (d) for the introduction of remedies; (e) in connection with subarachnoid irrigation; (f) diagnosis of spinal cord tumors.

**Technic.**—Lumbar puncture can be done without pain by superficial infiltration of the skin, and deep injection of novocaine. The patient should be on his side with the back well bowed to separate the vertebræ. The puncture is made between the fourth and fifth lumbar vertebræ, found at the point where a line on a level with the iliac crests crosses the spinal column. The needle is placed at a distance of  $\frac{3}{4}$  inch on either side of the line of the spinous processes and slowly forced in at an angle that will cross the median line at the center of the spinal canal. For purposes of laboratory examination  $\frac{1}{2}$  to 1 c.c. of fluid is sufficient.

The pressure is generally estimated by the rate of flow from the needle. This is a crude and unreliable method, subject to many errors, and bears no relation to real pressure. The flow from the needle may be drop by drop with high intracranial pressure, due to obstruction at the base of the brain or in the spinal canal. Repeated pressure readings have been made where it required time for the fluid to slowly filter into the tube, to obtain a true reading. The fluid should be drawn off slowly to give time for the adjustment of the cerebral circulation. The relief of cerebral pressure by the withdrawal of fluid is only temporary, done in the hope that it will give Nature a chance to gain control. When serums are to be injected into the spinal canal, it should be done slowly by gravity, and the amount introduced should be less than the amount of fluid removed.

**Measuring Cerebral Pressure.**—It has been our routine for several years in every case of lumbar puncture to make a manometer reading. Two types of spinal manometers are available. One depends on the displacement of a column of mercury. It is compact and easily handled by one operator, but has the disadvantage that minor movements of the fluid cannot be appreciated, the study of which may be of diagnostic value.

In the other type the reading is made from the height the fluid rises in a graduated open tube. It consists of the needle with a socket for receiving the glass tube and an independent outlet for drainage without entirely withdrawing the stylet (Fig. 347). Pressure can be taken with the withdrawal of the very small amount of fluid required to fill the tube. Normally this registers from 120 to 150 mm. During the withdrawal

of fluid for drainage the decrease in pressure can be accurately noted. The rapidity with which the fluid enters the tube indicates the absence or presence of obstruction to the flow above the puncture. Two distinct oscillations can be noted: One synchronous with the pulse, with a rise and fall of 1 to 2 mm.; the second with respiration, with a swing of 1 cm. or more. In making a reading the patient must completely relax and breathe quietly. Muscular contractions, deep breathing, or holding the breath, increases pressure. The absence of the respiratory wave is highly suggestive of internal hydrocephalus, confirmed, however, by only two autopsies. In one, opinions differed between right frontal lobe abscess and internal hydrocephalus. Cerebral pressure was 450 mm., with a respiratory wave of 2 cm. At operation an abscess 3 by 4 cm. was evacuated. Autopsy showed an open aqueduct of Sylvius. The second, with equally high pressure but no respiratory wave, showed both the inlet and outlet of the fourth ventricle blocked by a thick exudate.

**Diagnosis of lateral sinus thrombosis** by the use of the spinal manometer was reported by Drs. Ayer and Tobey, of Boston, at the American Otological Society, 1925. In a limited trial this has proved to be a rational and accurate procedure in complete blocking of the sinus.

With an occluding thrombus in one lateral sinus, the manometer reading will be higher than normal, approximately between 200 and 250 mm. If now firm pressure is made over the jugular vein of the occluded sinus there will be no change in the cerebral pressure. When pressure is made over the jugular vein of the free sinus, thus blocking both sinuses—one blocked by the clot, the other by pressure on the vein—there will be a marked and rapid increase of cerebral pressure, the fluid probably overflowing at the top of the tube. This is diagnostic of complete blockage of the sinus opposite to the one on which pressure is made. With a parietal clot, the finding will be less definite or very doubtful. This procedure is especially valuable in determining which sinus contains the clot in cases of double suppurative otitis media.

*Caution.*—In making this test, complete relaxation of the patient is necessary; no struggling or holding the breath. It is best done under complete anesthesia. Pressure must not be maintained too long, or damage to the brain tissue may result.

#### VENTRICULAR PUNCTURE

Ventricular puncture is for relief of cerebral pressure from internal hydrocephalus. The posterior horn of the lateral ventricle can be reached through an opening made with a Hudson drill 1 inch above the occipital

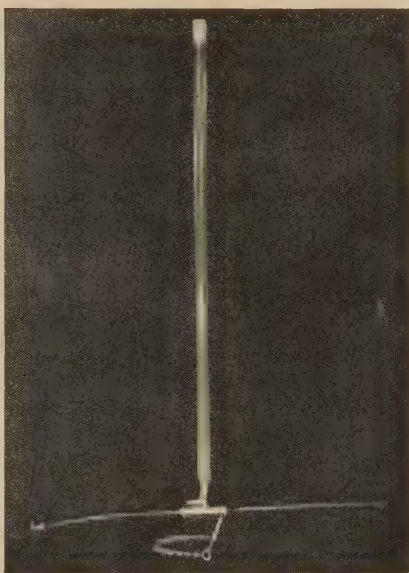


Fig. 347.—The Kraus manometer for taking the cerebral pressure.

protuberance and  $\frac{3}{4}$  inch from the median line. Through a nick in the dura the needle, passed straight forward parallel with the midline of the brain, will enter the ventricle at a variable distance, depending on the amount of distention.

#### PUNCTURE OF THE CISTERNA MAGNA

Puncture of the cisterna magna is performed: (a) For one point of entrance in subdural irrigation; (b) for the introduction of serums in spinal block; (c) in diagnosing compression of the spinal cord. The needle enters the cistern between the occipital bone and the atlas, through the connecting ligament. The technic, though not difficult, should first be tried on the cadaver as it carries potential danger.

**Technic.**—The patient is placed on his side so that the spinal processes are straight with the midline of the skull. The needle is introduced at a

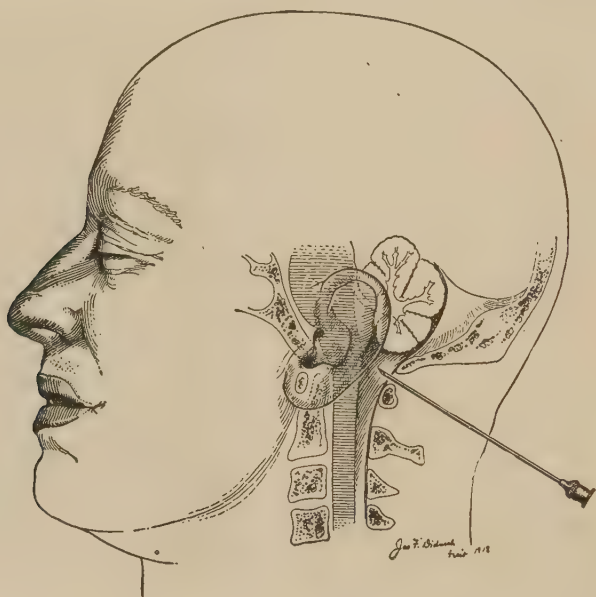


Fig. 348.—The landmarks in performing cisterna puncture (Ayer).

point in the midline of the neck, bisected by a line from the glabella and upper edge of the external canal, which line passes through the atlo-occipital ligament. A straight edge held on the skull over these two points will assist in giving the direction. The needle following this imaginary line to the glabella in the midline of the head will penetrate the ligament at an average depth of 4 cm.<sup>9</sup> (Fig. 348). The least distance to the cisterna magna from observations made on cadaver was 3 cm.; the greatest 5.5 cm., with a fairly constant distance of 4 cm. in the greater number. The resistance of the ligament to the passage of the needle can be felt by one accustomed to making lumbar punctures.

#### MENINGITIS

Infective meningitis occurs, not only as the most frequent of the graver complications of purulent otitis media, but also as a secondary result from operative measures for brain abscess. At the present time no successful

method has been developed that offers more than an occasional recovery. The outstanding symptoms of meningitis are those resulting from infection and from increased intracranial pressure. This pressure can be crudely relieved by drainage, but the control of the infection seems still far distant. All treatments must have for their objective the freeing of the meningeal surfaces from infection and the prevention of injury to the brain from pressure.

The anatomical structure of the brain coverings with their innumerable spaces, recesses, and pockets, for the most part inaccessible, makes the application of drainage as applied to other parts of the body impossible here except to a *limited* degree. Internal medication is without effect, and intraspinal injections of remedies are as a rule unsuccessful.

The trying and retrying of old procedures with many modifications has resulted in establishing certain facts: (a) That meningitis is at first a localized condition with a more or less latent stage; (b) that surgical drainage of this area while still localized would result in many recoveries; (c) that later, with the formation of exudate and blockage of the cisterns at the base of the brain, the case becomes hopeless; (d) that the symptoms of meningitis as now accepted are those of the terminal stage, or *death symptoms*.

The facts that prevent early operations are: (a) The early symptoms are vague and uncertain and may be caused by many minor disorders; (b) the patient may not come under observation during the prodromal stage; (c) the natural reluctance of both patient and surgeon to assume grave risk, by opening the cranium, without strong clinical indications.

A patient with suppurative otitis or following a mastoid exenteration, complaining of malaise and slight headache, should be under close observation, and manometric readings of the cerebrospinal pressure are indicated. Clinical observations indicate that the reaction of the vertical semicircular canals are early sensitive to increased pressure below the tentorium. An abnormal reaction of these canals to test suggests a beginning meningitis below the tentorium.<sup>10</sup> Close observation and study will often reveal slight symptoms that otherwise would be overlooked.

The surgical treatment of infective otitic meningitis aims at: First, eradication of the primary source of infection; second, drainage of localized collections of cerebral fluid and pus; third, irrigation of the intradural spaces.

**Primary Focus.**—It is well known that a necrosing focus in the adjacent temporal bone may, from the toxins of suppuration, cause an increase in the cerebral fluid, with pressure and irritative symptoms, constituting a serous meningitis, diffused or localized. The early removal of the primary bone focus, and relief of pressure by lumbar puncture, is followed by a high percentage of recoveries.

If these measures are delayed and infection of the accumulated fluid follows, the removal of the primary source will in no way exert a curative influence on the now intracranial infection. The meningeal infection thus starting is now an independent focus in a favorable medium for disseminating through the arachnoid spaces. By the successful reaction of a protective meningitis,<sup>11</sup> there may be thrown around this area a barrier of leukocytes, which, if permanent, may form the beginning of a pia-arachnoid abscess; but if this barrier of leukocytes gives way, the infective process spreads into a diffused leptomeningitis. It is during this localized or latent walled-off period that local subdural drainage is effective.

**Treatment.**—*Subdural Drainage.*—It has been shown that subdural drainage is effective over a limited space (Fig. 349) but without value when the infection has spread beyond the drainable area or to the cisterns at the base of the brain (Fig. 350). The most frequent location for this localized

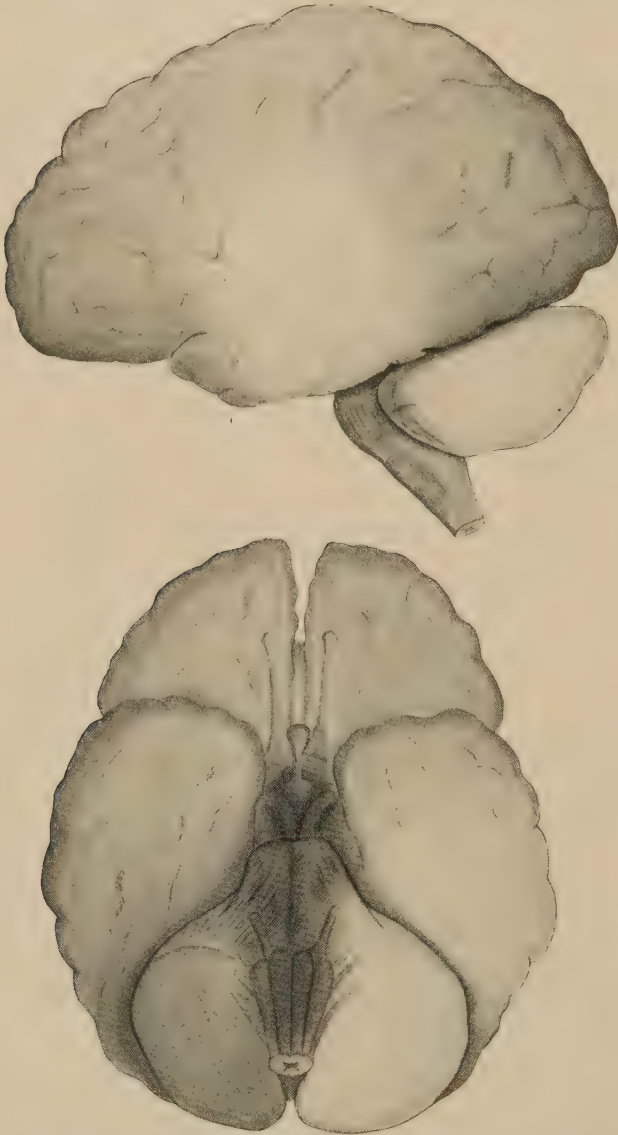


Fig. 349.—Extent of effective subdural drainage in temporal and cerebellar region. Death and autopsy two years after drainage for influenzal meningitis.

collection of infected fluid is in the arachnoid spaces on the under surface of the temporal lobe which overlies the tympanic and mastoid vault and the upper surface of the pyramid bone, and the anterior surface of the cerebellum adjacent to the posterior surface of the pyramid bone. The real problem is not how but when to interfere.

*Draining from the Temporal Region.*—The technic for exposure of the temporal lobe in general is that for temporosphenoidal abscess, except that it is necessary to open the skull low down near the upper border of the external auditory canal to give access to the under surface of the temporal lobe. The procedure should be through a clean field unconnected with any previous mastoid work. Intracranial pressure must be reduced by lumbar puncture to the point where the dura is no longer tense on palpation. A large dural opening is not necessary and only favors the formation of a cerebral hernia. A horizontal incision  $\frac{3}{4}$  inch in length is sufficient. A folded strip of rubber tissue is slowly introduced, following the direction of the pyramid bone between the dura and meninges to its apex, and the projecting end of the drain stitched to the skin. Unnecessary space in the dural incision is closed, and the scalp opening closed excepting at the drainage outlet.

The maximum efficiency of the drain is in the first twenty-four hours, requiring changing of the outer dressings several times. The drain once in position is not disturbed, but allowed to remain until it is no longer effective, when it should be removed and the scalp outlet closed. Several recoveries in severe cases have been reported by this method.<sup>12</sup>

*Subtentorial local drainage* is done on the anterior surface of the cerebellum, internal to the descending portion of the sigmoid sinus. This space is exposed by removal of the posterior wall of the mastoid cavity. The drain is passed within the dura well down toward the internal auditory canal. As the dura is opened from the infected mastoid cavity, there should be reasonable evidence that the localized condition exists before the arachnoid spaces are exposed.

Subdural drainage when done through a clean field involves no special dangers, but its usefulness is distinctly limited to blocked areas and its application limited.

*Irrigation of the Subarachnoid Spaces.*—Many attempts to irrigate and drain the meningeal surfaces and cisterns have been tried and abandoned from lack of satisfactory results. These attempts are mainly valuable for their demonstration of some of the things that should not be done. A better knowledge of the physiology of the cerebrospinal circulatory system<sup>13</sup> has resulted in the presenting by Eagleton of an improved technic for the irrigation and drainage of the basal cisterns. He reports 3 cases of recovery from traumatic meningitis.<sup>14</sup> The operation is highly technical and should not be undertaken without due preparation. The procedure has for its object the drainage of the basal cisterns to outlet needles in the cisterna magna and lumbar regions, and the replacing of the cerebral fluid thus re-

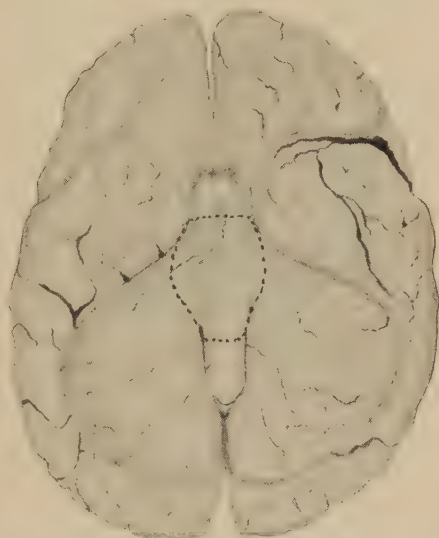


Fig. 350.—Streptococcic meningitis. Successful sterilization of the area within the dotted lines is essential for recovery.

moved by a warm non-irritating Ringer's solution at a temperature of 99° F. at the point of entrance to the meninges. To be successful it must be done before the pathways are blocked by exudate. Any benefits obtained will be derived from the first irrigation. A second irrigation is not recommended. The procedure is based on the assumption: (a) That the meningitis begins in, and for a time is confined to, the basal cisterns; (b) that the whole cerebrospinal system is but slowly involved; (c) that dissemination of the infection is aided by the pulsations of the arteries, passing through the cisterns; (d) that recovery from meningitis is made possible by the withdrawal of infected cerebral fluid and its replacement by a non-irritating physiological solution, and placing the parts at rest by ligation of the common or internal carotid artery. (No reports have been made on the possible damage to the meninges from lack of blood-supply until anastomosis takes place, due to this ligation of the carotid artery.)

*Equipment for Subdural Irrigation.*—(a) A gravity irrigator with an inner and outer chamber—the inner chamber for the irrigating solution; the outer chamber for the water-bath to maintain this irrigating solution at an even temperature; (b) a rubber tube attached to the outlet of the inner chamber, in the distal end of which is inserted the dural cannula for introducing the solution into the arachnoid spaces or cistern. A tip used by ophthalmologists to irrigate the anterior chamber of the eye makes a good dural cannula for this purpose. (c) A thermometer to verify the temperature of the irrigating solution before it enters the meningeal spaces is placed in the upright arm of a glass T-tube inserted in the rubber irrigation tube near the dural cannula. (d) Two lumbar puncture needles, the one for the cistern puncture having a short beveled point.

*Technic.*—1. Ligation of the common carotid artery on the affected side. This places at rest the inflamed subarachnoid spaces, lowers cerebral tension, and renders bloodless the approach to the basal cistern.

2. The rapid cutting away of the pyramid bone with a heavy burr, and exposing the cisterna pontis at the apex of the pyramid bone. No attempt is made to preserve the seventh and eighth nerve. This procedure is much facilitated by the previous ligation of the carotid artery.

3. The placing of drainage needles in the cisterna magna and lumbar canal. The cisterna magna must be the most dependent point of the body.

4. The replacement of the cerebral fluid as it drains from the cisterna magna and spinal canal with Ringer's solution, introduced into the cisterna pontis through the dural cannula. This solution must be introduced by gravity from a height of but a few inches, to prevent overdilatation of the arachnoid spaces. The dural cannula is inserted in the cisterna pontis, and the solution allowed to flow through the spaces at the base of the brain, and out at the needles in the cisterna magna and possibly the lumbar region. This procedure removes a large amount of infected cerebrospinal fluid from around the base of the brain and replaces it with a sterile physiological substitute.

5. The increase of the immunity of the patient by the transfusion of blood from professional donors who previously have been immunized to the particular strains of streptococcus present in the meninges.

*Conclusions.*—1. To be successful any measures must be undertaken early.

2. Subdural drainage is effective in localized meningeal infection which, unrelieved, carries a potential danger of a general leptomeningitis.

3. The curative effects of subarachnoid irrigation and replacement are not at this time fully established.

4. Operative measures are hopeless when the pathways are blocked, as in the terminal stages, or where the pneumococcus is the infecting organism.

5. The diagnosis of infective meningitis made, operation on the mastoid alone is useless.

**Extradural abscess** is a collection of pus between the bone and dura, adjacent to a primary focus, most often found in the region of the mastoid over the tympanic vault or cerebellum. Symptoms are absent, or mild and indefinite, and it is usually discovered while operating by a visible fistulous tract leading to it, or a small bead of pus appearing on the posterior mastoid wall (Figs. 351, 352).

*Extradural Abscess With Protecting Granulations.*—The dura is usually covered with granulations, which with its natural resistance to invasion has effectually protected the meninges. This condition adds little to the gravity of the case and presents no surgical difficulties. The bone should be removed over the infected area until healthy dura is exposed, and treated

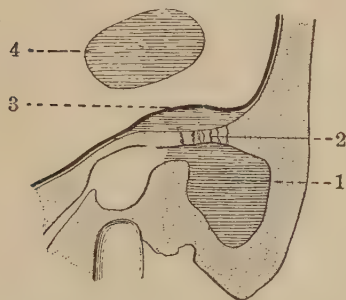


Fig. 351.—Mechanism of intracranial infection by the venous route during otitis and mastoiditis: 1, Mastoid abscess; 2, roof of mastoid traversed by veins; 3, extradural abscess; 4, brain abscess. (Laurens.)

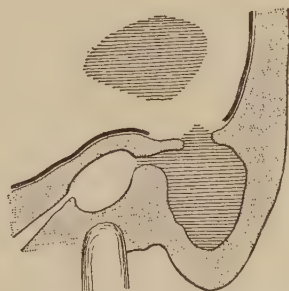


Fig. 352.—Abscess by direct extension. (Laurens.)

as part of the mastoid. Most authorities advise against interference with this granulating surface but I have never been able to resist the temptation to remove the redundant granulations, with no after-regrets.

*Extradural Abscess Without Granulations.*—When this rare condition occurs, if not of recent origin, it is suggestive of a possible associated infective process within the dura, especially if marked changes in color or consistency of the dura have taken place. If no such complication is evident from inspection, the dura should not be opened until evidence of meningeal involvement is confirmed by taking the cerebral pressure, examination of the spinal fluid, and clinical symptoms.

#### BRAIN ABSCESS

A *brain abscess* is a localized intradural collection of pus, usually referred to by its anatomical location as frontal, temporosphenoidal, or cerebellar abscess.

A brain abscess is usually in juxtaposition to an infected area, and is referred to as an *adjacent abscess*, differentiating it from a *metastatic abscess*, which is embolic in origin, the primary source of infection of which is in a

distant part of the body. The infection, being carried by the blood-stream as a small embolus, may find lodgment in any part of the brain with or without evidence of localization. Metastatic abscesses are frequently multiple, and may be widely separated, as in the cerebrum and cerebellum. They most frequently develop in the white substance of the brain, in which, having less resistance to infection, they run an acute course without forming a capsule.

With reference to the brain surface an abscess may be *pia-arachnoid* or *intracerebral*.

A *pia-arachnoid abscess* is the result of direct extension from without the dura or from a localized meningitis becoming infected. It does not extend

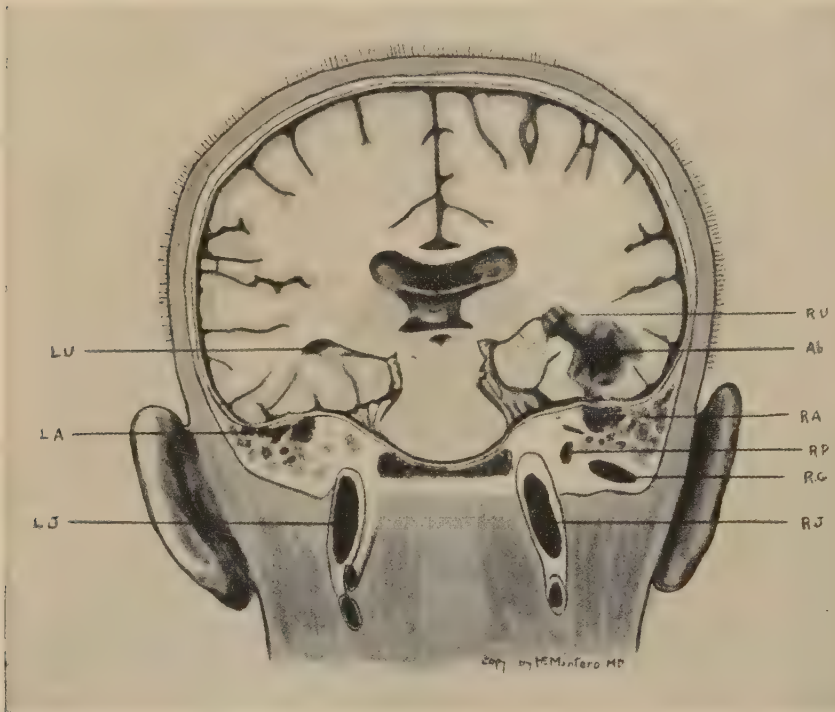


Fig. 353.—Showing uniform position of adjacent abscess low down in temporosphenoidal lobe above antrum or middle ear. (From Eagleton's Brain Abscess, Macmillan Co., Publishers.)

directly into the brain tissue, but may cause superficial pressure necrosis of the cortex. Thrombosis of a blood-vessel entering the brain from this point can give rise to an intracerebral abscess. When this takes place it may result in the formation of a stalk.

Pia-arachnoid abscess, uncomplicated with a deeper abscess, more properly should be classified as a localized purulent meningitis.

Intracerebral abscess may be either acute or chronic.

In an acute abscess tissue necrosis is active and evidence of septic infection present. No limiting membrane has formed. Cerebral pressure is increased by an area of edema of the brain around the abscess, and the increase of cerebral fluid. In a case of acute temporosphenoidal abscess

at Camp Lee the manometer showed a pressure of 600 mm. at which point the fluid overflowed the measuring tube. After evacuating the abscess the pressure still registered 400 mm., the pus pressure only accounting for 200 mm. plus—the general cerebral edema for much of the remaining pressure.

*Chronic abscess* may be *with* or *without* a capsule. Adjacent abscesses are most frequently encapsulated, while those without a capsule are more frequently metastatic abscesses. Abscess with a capsule may be: (a) Without macroscopical connection to the dura or meninges—the most frequent variety in otological cases—or (b) with a stalk connecting the capsule with the brain surface or attached to the dura (Fig. 353). This stalk can be formed by direct extension inward along a vessel entering the brain or by a retrograde thrombosis from within out.<sup>16</sup> The formation of a stalk is a chronic process, and when found should be utilized to enter the abscess. These cases show the highest percentage of recoveries.

Occasionally a fistulous tract from an abscess may be found draining from a stalk attached to the dura.<sup>17</sup> In the absence of increased cerebrospinal pressure, interference with this tract should at least be delayed and work limited to increasing drainage by enlarging the bony opening.

While the presence of a stalk is the greatest factor in successfully evacuating an encapsulated brain abscess, the capsule in an abscess without a stalk may cause failure to obtain proper drainage, either through unpreventable conditions or faulty technic. The abscess may be emptied so rapidly on first penetrating the capsule that partial collapse follows, and the point of entry is lost. Subsequent efforts to enter the cavity for drainage usually fail, and the tissue external to the capsule is exposed to infection, resulting in increased edema of the surrounding brain.

That surgeons differ as to the proper method of treatment of brain abscess is shown by the different methods employed. Certain basic conditions should be remembered.

1. All operations for brain abscess are more or less exploratory.
2. Failing to find an abscess, closure of the parts without the risk of secondary infection is of the utmost importance.
3. The ideal entrance to an abscess is through a well developed stalk. When not present, exploration should be through a *clean field*.
4. The dura is highly protective but, once opened, is an open gateway to meningitis.
5. Brain tissue has a low resistance to trauma.
6. The end sought is the complete evacuation of the pus from within the brain with the least trauma and the avoidance of secondary infection.
7. The shock and time required in an extended exposure may offset the advantages over one less complicated.

**Treatment.**—The treatment of brain abscess is essentially surgical and, once the diagnosis is made, no delay should occur.

Brain-cells are the most sensitive and highly organized tissues in the body. They are the most sensitive to trauma and shock, and have the least regenerative power. It follows that the utmost pains and care must be used to avoid unnecessary manipulations and trauma. The brain cannot be entered without a certain amount of trauma and shock to the nerve centers, which should be reduced to the minimum. When a patient enters the operating room fully or partly conscious, and does not return to

this former mental state in a few hours after the operation but remains in coma for an indefinite time, the operation should be analyzed to ascertain how much the condition is probably due to necessary technic and how much to failure to realize the intolerance of the brain to trauma. "More deaths are due to a faulty conception of what the surgeon is called upon to attempt than to lack of operative skill."<sup>15</sup>

**Preparation of Patient.**—The patient is prepared as soon as operation is decided upon. The entire head should be shaved and the old mastoid cavity cleaned, if present, painted with tincture of iodine, repacked, and dressings so applied that they can be removed without disturbing the final head dressing. The prominent landmarks on the scalp are marked. The lines for the incision are plainly outlined and the points for puncture of each lateral ventricle indicated. This can be done with a scalpel if immediate operation is to follow, otherwise the lines will soon disappear. If the patient is conscious, the local application of ethyl chloride will render this outlining painless. When, for good reasons, an extended delay will occur, a solution of nitrate of silver can be used for this purpose, but the scalp must be exposed until the lines become dark. This outline can be done much more quickly and accurately with the entire scalp exposed than after the patient is on the table and the field cloths placed. The head is now covered with a moist bichloride pack and temporarily bandaged so as not to interfere with the mastoid coverings. At this time it is good practice to take the cerebrospinal pressure. Even when the lesion is below the tentorium, it can be done without harmful symptoms. The amount of fluid withdrawn is exceedingly small, being only what flows into the measuring tube. The information so derived may lead to a change in operative technic. When the pressure is great, marked herniation of the brain and increased bleeding from the diploic veins of the skull would be expected. From the respiratory wave of the fluid in the manometer (see pressure reading) the presence or absence of an internal hydrocephalus may be reasonably suspected.

As most brain abscesses are a complication of otitic suppuration, more bone work in a contaminated mastoid may be necessary. On its completion the mastoid should be douched, cleansed with alcohol, packed with gauze, and covered from any adjoining field. The temporary scalp dressing applied in the dressing room is now removed, and the cranial field again cleansed by an assistant, while gloves and gowns are changed. The coverings are pinned securely to the scalp with retention clips, but so arranged that the field for a ventricular puncture, if needed, can be exposed with a minimum of disturbance.

**Anesthetic.**—A trained anesthetist is essential. All struggling and straining by the patient is to be avoided as it increases cerebral pressure—already too high. A quick primary anesthesia with ethyl chloride, blending gradually with ether, then ether alone, makes a quick and satisfactory method.

**Local Anesthesia.**—The brain can be explored under local anesthesia. The dura is not sensitive. A drop of weak solution of cocaine on the meninges over the point of exploration will produce a local anesthesia on the surface, through which the brain substance, which is non-sensitive to pain, can be explored. In a case in which we were forbidden to go beyond the mastoid when operating, at the first dressing the dura was incised with no

anesthetic and pus evacuated without the knowledge of the patient. Local anesthesia is not, however, recommended except in stupor. The physical pain can be controlled, but there exists in the mind of the patient a state of anxiety and suppressed fright, which is depressing and, in an emergency, may break out into active resistance.

*Technic.*—The scalp is infiltrated by the Schleich method over the proposed lines of the incision, using a solution of  $\frac{1}{2}$  per cent. novocaine with 1 : 10,000 adrenaline. This is followed by deep infiltration beneath the periosteum over the same area. A stout needle and high-pressure syringe are required. The point of the needle must be well under the periosteum before pressure is applied to the piston, and slowly advanced as the solution is expelled.

**Special Instruments.**—A Hudson drill for perforating the skull. Braatz dural separators of different curvatures, and one flexible separator. Gigli saws and protector, if a bone-flap is to be made. Large and small bone forceps. Avoid forceps with long jaws or short handles. A small dural hook, knife, and dural scissors, for opening the dura. Brain cannula, forceps, or knife, for exploring the brain. A rubber catheter with wire stylet. Cushing silver wire clips for the small pia blood-vessels. Bone-wax for control of bleeding from the diploic veins.

**Operative Approach.**—Governed by the location of the field and the exposure to be made, the division of the soft parts may be by a straight or a curved incision or by a flap thrown back.

*Control of Hemorrhage.*—The hemorrhage encountered in large incisions of the scalp is free and, with increased cerebral pressure, may be menacing. Among the several mechanical means for its control, the use of rubber tubing, as a tourniquet, is the simplest and most effective in a measure. Its use, however, is restricted. In cerebellar cases it is ineffective, and in the temporosphenoidal region it may interfere with the operative field.

The line of the incision outlined, an assistant makes firm pressure with his finger-tips on each side, and the scalp is incised between them to the skull. The galea is elevated and seized with artery forceps at close intervals, the weight of the forceps pulling the galea over the cut edges. This makes sufficient pressure to control the free oozing; vessels spurting on removal of the finger-pressure should be seized with forceps or ligated. This is repeated through the length of the incision.

*Dural Exposure.*—The soft tissues retracted, the skull is penetrated with a Hudson drill, and the opening enlarged with bone forceps to the desired size. In doing so, free bleeding may occur from the veins in the diploë. This can usually be controlled by the use of bone-wax, but if it fails, a carpenter's nail-set placed over the bleeding-point and tapped lightly will be effective and give a better grasp for the wax. Bone bleeding must be entirely controlled before the dura is opened.

When the operation is exploratory, requiring a large dural exposure, an *osteoplastic* flap may be used. This permits of aseptic closure when nothing is found. In outlining an osteoplastic flap, the scalp portion of the flap must be formed so that it will be larger and extend beyond the bony portion. The flap outlined and incised, the scalp is retracted along the incision. Drill-holes are made at the four corners of the proposed flap and, if large, in the center of the three sides. In making the drill-holes, severe bleeding may be encountered from the veins of the diploë, requiring packing of that

section, leaving its completion to the last. The dura is separated from the bone between the drill holes, and the bone sectioned with a Gigli saw or cranial forceps. Injury to the dura from the saw is prevented by the introduction of a metal protector or elastic metal sound. The saw permits of forming a bevel flap, which cannot be obtained with the various cranial forceps. In the presence of dangerous hemorrhage, while cutting the flap, it may be necessary rapidly to sacrifice the bone, by rongeurizing it away, to control the bleeding. The bone, severed on three sides, is pried up and fractured at the base. When the base is broad or thick it may be necessary to pry up the bone sufficiently to permit the introduction of a Gigli saw and partially cut it through. In the presence of sepsis, infection of the flap will probably follow, with necrosis and partial or complete loss of the bone. In large exposures, by complete removal of the bone, the amount of subsequent muscular support to the brain over the bony defect must be considered. Over the cerebellum and temporal region this is well provided for, while over the frontal region it is slight.

The dura exposed, it is palpated for the presence or absence of pulsation. As a rule pulsation is absent with an abscess, but its presence does not signify that no abscess exists. It may be small or located deep in the white substance.

*Incision of the Dura.*—The crucial incision of the dura should be avoided. With increased intracranial pressure, a crucial incision favors the formation of, and the strangulation of, a hernia cerebri in small exposures. A straight incision lessens the chances of this complication and gives equal working room. When extensive inspection of the meninges is desired, a dural flap of large area should be made.<sup>18</sup> Unnecessary exposure of the meninges increases the risk of infection and trauma.

Great care must be used in incising the dura to avoid injury to the pia-arachnoid membrane. This should be done by repeated light strokes of the knife between two ligatures or small sharp hooks caught in the dura, lifting it slightly from the parts beneath. The primary opening made can then be extended with dural scissors. A dural flap, when used, should have an outer dural margin  $\frac{1}{4}$  inch from the bone to facilitate after-closure.

Depending on the amount of pressure, bulging of the brain occurs when the dura is opened. With marked herniation, lumbar or ventricular puncture is necessary before proceeding. This should be prepared for in advance. Even with moderate pressure this procedure should be followed if search is to be made for a stalk or collection of pus beneath the dura, beyond the cranial opening. With the exposure of the subarachnoid spaces all manipulations must be slow and painstaking.

The brain surface is constantly bathed with cerebral fluid. On exposure it becomes dry and more susceptible to injury and rupture of the pia capillaries from stretching by the bulging brain.<sup>19</sup> When kept moist its resistance to trauma is increased. Warm, moist cotton sponges should be used and gently pressed, not wiped, to clear the field.

*Control of Bleeding.*—Large vessels may be divided between double ligatures when they cannot be avoided. Bleeding from the small pia vessels may be troublesome. The application of warm, moist cotton pledgets, pieces of muscle, or moist cotton smeared with vaseline may succeed; if not, the application of Cushing's silver clips are effective. The small vessels of the pia are too delicate to be seized by forceps or ligature.

*Intracerebral Exploration.*—All instruments for this purpose should be of known length, so the depth of penetration can be ascertained. A brain cannula with a large opening on the side close to the tapering point is preferable. Held between the thumb and finger, it is slowly pushed into the brain—the exposed meninges protected by moist cotton from expected leakage or overflow—and changes in resistance can usually be appreciated when encountering a capsule or entering an abscess. The initial puncture being unsuccessful, it is repeated in different directions until the adjacent area has been explored. Under perfect asepsis this involves little danger. These punctures are often made through a single dural opening, but this procedure is open to the objection that, if pus is found, infection may extend from the dural outlet along the previous punctures and result in secondary abscess formation. To obviate this a separate parallel incision in the dura is made for each puncture.<sup>20</sup> The abscess entered, the flow of pus is quickly checked to prevent loss of the opening by collapse of the capsule. With the cannula still in the abscess cavity a pair of brain forceps with thin blades,  $\frac{1}{4}$  inch wide, semisharp and semipointed, are introduced alongside the cannula to the same depth as the cannula and opened, the cannula then being withdrawn. The forceps once spread and locked is not removed until the cavity is emptied and drainage placed. The forceps can be used alone, but require repeated opening and closing while advancing. Many operators recommend the use of a long narrow brain knife, as causing less trauma. The loss of the opening to an abscess cavity may occur with the best technic, but is none the less disastrous. The protection formed around the abscess has been penetrated and leakage into non-infected tissue results, and the drainage placed outside the cavity irritates and favors extension of the infection.

*Complete evacuation* of the abscess at the primary operation is important. When pus no longer appears, rotation of the head to different positions may cause it to again flow, and at the same time gives an idea of the position of the opening in the cavity. Routine attempts to see, douche, or pack the interior of the cavity are inadvisable and productive of trauma. In exceptional cases with caseous or necrotic material the mechanical removal, when the abscess is subcortical, can be advantageously done through the Whiting speculum.

*Drainage* has a twofold purpose—to empty the abscess and to establish a fistulous tract by which it can be maintained until the cavity is obliterated. Gauze is the least desirable for this purpose. It requires frequent changing, and pressure forces cerebral tissue into its meshes, causing irritation on removal. Pleated rubber tissue is free from this objection, but the pressure and pulsation of the brain tend to expel it. When rubber tubing is used it should project an inch beyond the scalp. This end is split lengthwise to the cranial opening. The drainage tube is anchored to the scalp by a suture through the divided ends, one on each side of the incision. It does not require removal, and can be kept in position by a stitch to the scalp until the cavity is obliterated.

Cahill reports good results from the use of Mosher's wire basket drainage tubes.

When a flap has been used, the drains must be carried to the surface by the shortest route, through a button-hole opening in the flap.

*Dural Repair.*—It is, of course, necessary to leave a free opening around

the drainage; other than this, the dura edges should be closely approximated by sutures. When unable to do so without injury to the cortex, fascia lata should be sutured in the defect.

*Cerebral hernia* is more frequent over the vertex and cerebellum than the temporosphenoidal region. Its appearance, when of considerable size, is a grave complication, and with brain abscess usually forecasts a fatal termination from meningitis. Care at time of operation will to an extent prevent this condition. The reduction of cerebral pressure by lumbar or ventricular puncture and careful closure of the dura, leaving an opening sufficient for drainage, is the most effective guard against its formation. When a hernia forms the treatment given for induced hernia in the operation for "unroofing" a brain abscess should be followed.

**Temporosphenoidal abscess**, secondary to a chronic or acute purulent otitis media, is the most frequent form of brain abscess. The eradication of the primary focus in the middle ear and mastoid, with the exposure of the roof of the tympanic cavity and mastoid antrum for necrosis or fistulæ by a radical mastoid operation, is first indicated. Having in view the probable need to explore the temporal lobe through an opening in the squama, and the desirability of a clean field for this purpose, it is preferable to modify the usual radical technic. The encircling incision above the ear is omitted, and the cartilaginous portion of the external canal not elevated from the bone on the lower anterior portion. A wide tongue-flap is turned outward from the posterior part of the cartilaginous canal to give room for work in the tympanic cavity, and is used later for final placement in the mastoid cavity. This procedure will lessen the chances of infection if a subsequent temporal exposure requires extension downward to the upper border of the bony external canal. Freshly sharpened chisels and gouges should be used with as little jarring as possible. Waste no time on the finer technic of the radical operation, as shock increases in proportion to the time required. The radical operation completed and the cavity cleansed, search is made for exposed dura or a fistulous opening in the antrum or tympanic roof; then remove this bone, exposing the dura above. This area offers the most favorable site for draining a temporal abscess in the lower or middle convolution, and has given the highest percentage of recoveries where the dura is adherent to the brain cortex. The exposure, limited in extent, may show an extradural abscess, evidence of an intradural collection of fluid, or the dura adherent to the brain cortex. With these findings, the mastoid incision is now extended, encircling the upper part of the ear, the canal and ear deflected forward and down, exposing the upper bony auditory canal. The bone over the external canal is then removed to expose the lower temporal convolution. An extradural abscess with protective granulations unaccompanied by cerebral symptoms would be treated as an extradural abscess, and the dura not incised. Without granulations, but with gross changes in color and thickness of the dura and tension on palpation, it would be suggestive of an intradural collection of fluid or a meningeal abscess, and should be drained from this point. Intracerebral punctures through such an area would result, if no abscess was found, in infecting the exploratory tract and the formation of an abscess or encephalitis. As it is impossible to distinguish by the symptoms between an intradural and intracerebral abscess, after the finding of pus subdurally in quantity, the intracerebral exploration should be postponed awaiting developments.

When the dura is found necrotic and adherent, with a fistula or evidence of a stalk, the probabilities of a chronic encapsulated abscess are great, and puncture should be made along this tract, which would be walled-off both from the cerebral tissue and the meninges. The object is to establish and maintain drainage with the least danger of breaking down the protective adhesions. Unfortunately the cases with a stalk are in minority.

In the majority of cases there is *no visible evidence of dural involvement*. In this case the cerebrum should not be entered from this infected mastoid field. The mastoid cavity is now cleansed and packed, draperies, gowns, gloves, and instruments changed, and a new field in the temporal region prepared. The objective being to obtain a clean field, the extension of the mastoid incision over the temple does not meet this requirement; infection will spread along the soft tissues. A separate incision following the direction of the fibers of the temporal muscle is now made; the tissues are elevated and retracted; and the skull is perforated with a Hudson drill

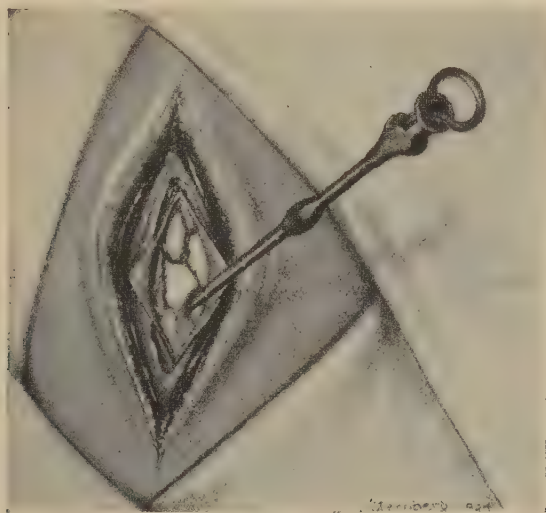


Fig. 354.—Exposure for temporosphenoidal abscess. Dura incised and cannula in abscess cavity. (Courtesy of Dr. Charles A. Elsberg.)

at a point  $1\frac{1}{4}$  inches above the external auditory canal and enlarged downward with the bone forceps, until an exposure of an inch or more is obtained. With a little care with the primary incision and dural opening, this exposure can be extended to the decompression operation if required. With marked increase of intracranial pressure, herniation of the brain would occur on opening the dura and, to prevent this, a lumbar or ventricular puncture should be done. This could be anticipated by measuring the cerebrospinal pressure prior to the operation; this might also indicate which procedure would be the most effective. If the manometer tube filled slowly with absence of the respiratory wave, an obstructed lumbar flow would be probable and a ventricular puncture indicated. It is best done on the opposite ventricle, as a large temporal sphenoidal abscess may by pressure or distortion cause partial or complete obliteration of the ventricle on the side of the abscess.

*In acute abscess* the exploring is preferably done with a short rubber

catheter carrying a wire stylet of small diameter that will not obstruct the flow of pus. The abscess located, the catheter is not withdrawn, but cut to the desired length and retained for permanent drainage.<sup>21</sup> Excepting in acute abscess, ridged semiblunt searchers should be used. The first puncture should be directed toward and internal to the tympanic and mastoid vault; this failing, then in different directions until the lobe has been thoroughly explored. A puncture carried beyond  $1\frac{1}{4}$  inches perpendicular to the surface must be done with great caution, as puncture of a distended lateral ventricle might result.

In *chronic abscess* a capsule of greater or less density may be encountered. With ridged searcher lightly held and slowly advanced resistance may be felt when the capsule is reached or entered (Fig. 354).

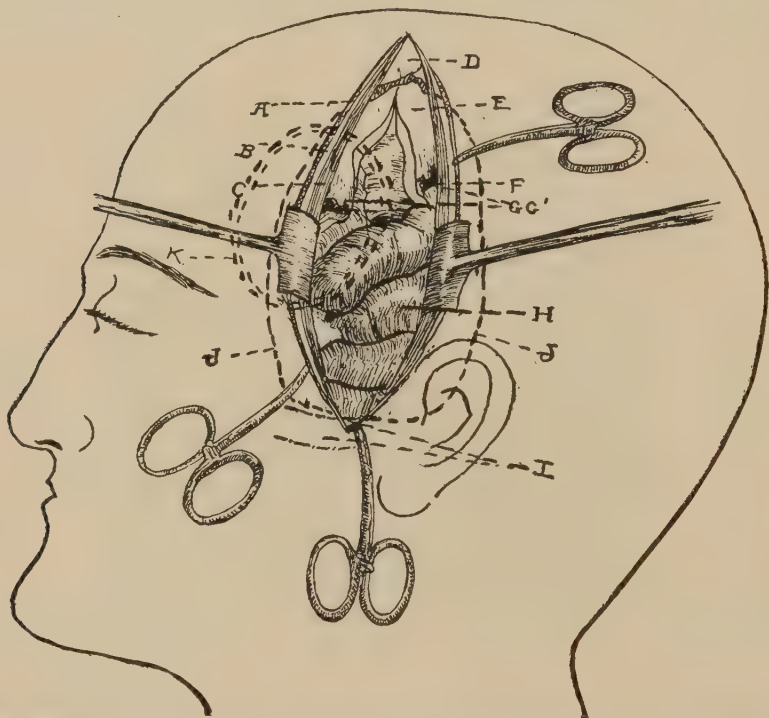


Fig. 355.—A, Skin and superficial fascia; B, temporal fascia; C, temporal muscle—its fibers divided longitudinally; D, skull; E, dura incised and its flaps retracted; F, branch of middle meningeal artery divided; GG, silver clips on artery; H, cortex; I, zygoma; J, decompression 3 by  $2\frac{1}{2}$  inches; K, location of abscess. (Courtesy of Dr. William Sharpe.)

The *temporal decompression route*, advocated by Sharpe,<sup>22</sup> has much to recommend it in cases where localization is doubtful and the dura in the mastoid region not adherent to the cortex. The temporosphenoidal, ipsolateral, frontal, and parietal regions can be explored, and an abscess, if found, drained. No abscess or infection being present, the opening (Fig. 355) can be closed with little danger to the patient.

The incision begins above the posterior end of the zygoma and extends vertically for 3 inches. The bone is removed over 3 by  $2\frac{1}{2}$  inches, and the dura opened vertically for  $2\frac{1}{2}$  inches and retracted. A wide field is exposed

for exploring. After repair the temporal muscle gives support as in a temporal decompression for dangerous intracranial pressure.

*Eagleton's osteoplastic flap*<sup>23</sup> has for its object a large exposure low enough to allow complete examination of the tympanic and mastoid vault and upper surface of the pyramid bone for intradural collections of pus, a proceeding imperfectly accomplished by other methods, and permitting an aseptic closure if no abscess is located. The lower skin incision is 4 inches long, beginning 1 inch in front and  $\frac{3}{4}$  inch above the external auditory canal, extends horizontally back 2 inches to a point above the posterior edge of the mastoid, turns upward at an angle of 30 degrees, to avoid the



Fig. 356.—Osteoplastic flap for exploring for abscess of middle fossa. (From Eagleton's Brain Abscess, Macmillan Co., Publishers.)

lateral sinus, for  $1\frac{1}{2}$  inches to the posterior inferior angle of the parietal bone. The anterior incision extends up 4 inches at a right angle to the anterior part of the lower border. The posterior incision is made to the height of the anterior and  $2\frac{1}{2}$  inches from it at its upper end. Seven drill holes are required. Free bleeding will be encountered at the postero-inferior angle. The bone flap is elevated upward and the dural flap down (Fig. 356). The brain can be elevated and a good exposure of the floor of the middle fossa obtained. Some small veins entering the dura will be torn in the posterior part. With the close proximity of the infected mastoid a careful technic is necessary. This osteoplastic flap exposure permits of

inspection and palpation over large areas for intradural or cerebral abscess. Disadvantage—the accompanying shock, the length of time required, and the higher technical skill in brain surgery. In case an abscess is located, the resulting infection of the flap with more or less necrosis of the bony portion is probable.

**Cerebellar Abscess.**—To explore the cerebellum the patient is placed on the table, face down. Shoulder supports or sand-bags under the shoulder raise the chest free of the table to prevent interference with respiration. The head extends over the end of the table supported in a head rest attached to the table, or by an “outrigger.” The neck should be arched upward without interfering with breathing.

Internal hydrocephalus is usually present from plugging or distortion of the aqueduct of Sylvius. The affected lobe may be crowded across the median line, compressing the opposite lobe. The danger of medullary compression is great and cerebellar hernia usually occurs. To partially reduce this pressure, before opening the dura over the cerebellum, the lateral ventricles should be drained.

An abscess from extension of a labyrinth infection is usually located in the anterior-lateral lobe adjacent to the affected ear. When abscess develops from a later sinus thrombosis the infection is carried by way of the small vessels entering the sinus from the cerebellum and is located in the lateral lobe posterior to the infected sinus. To inspect the anterior surface of the cerebellum the posterior mastoid wall is removed, and the posterior part of the pyramid bone cut away with a burr or chisel. A somewhat limited triangular exposure is thus obtained internal to the descending portion of the sigmoid sinus. The possibility of the formation of a stalk connecting the abscess with the dura on the anterior surface of the cerebellum, when secondary to a labyrinthitis, is as great here as in a temporo-sphenoidal abscess, and a search should be made for this condition. Should changes in the dura be present, indicating adhesions or fluid, exploration at this point can be made. As this is at a difficult angle on account of the limited space, a curved cannula is useful for this purpose. No extended exploration can be made from this limited exposure, and an abscess, other than one in the immediate vicinity of the triangle, will probably be missed. If no abscess is located, unless the dura is adherent to the cortex, a meningitis from the septic mastoid cavity through the dural incision generally follows. The tendency for the formation of a hernia, after incision of the dura on the anterior surface of the cerebellum, is much less than in the lateral or posterior portion.

The sigmoid sinus crosses the cerebellum at the most advantageous point for exploring and draining the abscess by the shortest route if it is located in the anterior part of the lateral lobe. Where a thrombosis of the lateral sinus is associated with cerebellar abscess, the inner wall of the sinus should be chosen as the starting point for exploration. The arachnoid spaces beneath the inner sinus wall are usually obliterated by the phlebitis. To avoid entering the cerebellum from the infected mastoid cavity for an abscess in the anterior part of the lobe in the absence of obliterated subarachnoid spaces, the lateral sinus can be approached from behind, to obtain a clean area unconnected with the mastoid cavity. The sigmoid portion of the sinus is obliterated by invulsion of its outer wall into its cavity at its upper end, and by an encircling ligature at its lower, tied over

a small firm compress. Incision can then be made through the sinus wall.

*Exploring Posterior to the Sinus.*—A clean field is important; neither the incision of the soft tissues nor bone removal should join the previous mastoid operative field. Due to the pressure, small bone openings are especially liable, in this region, to be followed by the formation of a brain hernia with strangulation.

To be in a position to prevent injury to the cortex and to avoid paralysis of the respiratory centers should that threaten, a free removal of the bone over the entire lobe is advisable. When only one lobe is uncovered by the bone removal, the search for the abscess is limited to exploring within that lobe. Should it be desirable to examine the surfaces of the lobe by elevating or depressing the lobe, it is necessary to remove the bone over both lobes and form a dural flap including the surface of both lobes as in operations for tumor. This requires ligation of the occipital sinus and division of the falx cerebelli.

*Technic.*—When only *one lobe* is to be exposed, a flap of the soft parts is formed by an incision extending from the occipital protuberance to the second cervical vertebra. A second incision beginning at the upper end of the first one extends horizontally along the superior curved line of the occipital approaching the open mastoid process, but ending a sufficient distance from it to prevent the spread of infection. If necessary to properly retract this flap, a short vertical incision may be made downward behind the mastoid. The bone is cut away with forceps over the lobe. The lateral ventricle is now drained and the lobe explored through a small incision in the dura. The operator must be prepared, if on opening the dura he encounters an uncontrollable or explosive hernia, to rapidly evacuate the abscess and form a large dural flap.

To expose *both cerebellar lobes*, the horizontal incision is extended to the opposite unopened mastoid—cross-bow incision—and the flap turned down. The bone is cut away over both lobes extending downward to include the posterior part of the foramen magnum, to guard against paralysis of respiration by prolapse of the medulla into the foramen, as in operation for cerebellar tumor (Fig. 357).

Before opening the dura over the cerebellum the ventricles are drained by ventricular puncture. This should be done slowly, drop by drop, to allow gradual partial readjustment of the brain-stem, crowded from its normal position by the combined pressure of the abscess, edema of the surrounding tissues, and the confined cerebral fluid.

Lumbar puncture is contraindicated. With internal hydrocephalus not only would pressure not be relieved, but the withdrawal of fluid from the basal cisterns would favor the forcing and strangulation of the medulla into the foramen magnum.

**Frontal Lobe Abscess.**—Abscesses in this region are especially fatal. The frontal lobes being a silent area involvement does not cause focal symptoms, and late diagnosis results. This may account in part for the high mortality. A fistulous tract or stalk occurs more frequently than in otitic abscesses. When present it should be utilized for drainage. In the absence of a stalk the operation should be through a clean field, as in the temporosphenoidal region, and the same technic is applicable. The decompression should not involve the frontal sinus. When an osteoplastic

flap is employed it should be on the outer portion of forehead, base outward. With this exposure, after ventricular puncture, the floor of the frontal fossæ can be seen by suspending the head over the end of the table allowing the brain to fall into the skull vault. A minimum of manipulation is advisable as the frontal lobe does not tolerate trauma. To compensate for the secondary edema of the brain that follows frontal lobe operation, it has been advised that a temporal decompression be a part of the operation.<sup>24</sup>

**After-treatment of Brain Abscesses.**—Frequent interference after operation is to be avoided. The drainage-tube should not be disturbed, and the

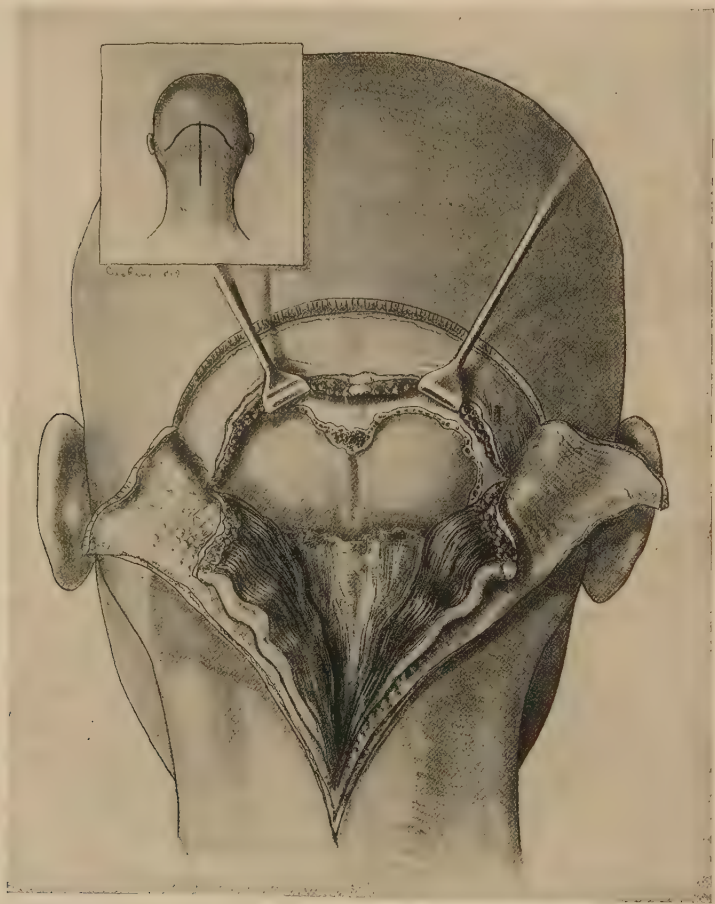


Fig. 357.—Exposure of both cerebellar lobes, showing opening partly made. (Cushing in Keen's Surgery.)

external dressing changed as soiled. The tube is retained until drainage entirely stops, then withdrawn gradually, to allow the tract to close without the formation of pockets. If it is found at the time of the operation that rotating the head in any position facilitated the evacuation of the abscess, such position should be utilized to aid after drainage.

The mortality in brain abscess is very high, given at 75 per cent. If the cases of all surgeons who have attempted operating for brain abscess were reported, the percentage of recoveries would probably be reduced one-half.

*Fatalities* incident to operative measures are: (a) *Immediate*, due to shock; (b) *later*, to meningitis, encephalitis, cerebral or medullary compression. Shock depends on the magnitude of the procedure, the amount of manipulation involved, hemorrhage, and time required by operation. In a report of 410 cases of surgical intervention on the brain at Eiselberg's clinic, surgical shock caused 50 per cent. of the deaths.<sup>25</sup> Edema and encephalitis may result from operative trauma, or trauma plus infection in heretofore sterile tissues. Meningitis may follow from contamination of the meninges and often as a sequela to cerebral hernia.

The value of the bone and dural openings for exposing the meninges depends largely on the judgment and experience of the different surgeons. Many fail to appreciate the intolerance of the brain to repeated slight traumata continued throughout the period of a long operation. For the occasional operator the simplest procedure is the best.

The various procedures for brain abscess heretofore employed, in which various forms of drainage were used, have been followed by a high mortality. In all, the prevention of hernia cerebri has been aimed at. Based on observations of brain injuries in the World War, and four consecutive recoveries following operation for brain abscess,<sup>26</sup> King has devised a new technic for dealing with brain abscess, which does violence to older methods. Drains are discarded. The brain tissue between the cortex and abscess cavity is removed. The dura is so incised as to favor eversion of the cavity by the formation of a hernia cerebri, combating the infection by the use of Dakin's solution.

*Treatment by Unroofing and Herniation.*—A three-limbed or crucial incision is made through to the skull; the flaps are elevated, reflected outward and held by self-retaining retractors. The apices of the flaps are turned backward and sutured loosely to the scalp, to prevent their becoming adherent too early to the hernia which will form. A trephine opening is made in the center of the exposure with a Hudson drill, bits A and B. A small incision  $\frac{1}{4}$  inch long is made in the dura and the brain explored with a small brain cannula. The abscess entered and its location indicated, the cannula is withdrawn, and the trephine opening enlarged to the size of a silver dollar or larger. The enlargement is so directed that the abscess will lie beneath the center of the cranial opening. The dura is incised in a stellate fashion, making six flaps which are deflected over the bony margin and sutured to the underlying surface of the scalp flaps. Three narrow strips of iodoform gauze are lightly packed beneath the margin of the circular dural defect to prevent the extension of infection to the subarachnoid spaces (Fig. 358). The cannula is again introduced to locate the abscess definitely, and a  $\frac{1}{2}$  inch incision is made with a sharp knife through the brain tissue and capsule, if present, overlying the abscess. Through a soft rubber catheter, with a Luer syringe attached, most of the contents of the cavity are withdrawn to prevent soiling of the field. The incision in the cortex or "roof" of the abscess is extended to the margin of the cavity, then around it in a circular manner, so that the entire roof of the cavity is removed, using a short knife. Only slight oozing occurs. If dural adhesions are present at the cortical margins they should not be separated. The cavity is thoroughly irrigated with Dakin's solution and gently sponged with cotton pledgets to remove its contents. No attempt is made to remove the capsule if present. No drainage is employed. A.

fenestrated rubber-dam is placed over the cavity, its margins extending over the operative field, over which pieces of gauze, wet with Dakin's solution, are placed so the rubber-dam dimples somewhat into the cavity. Three Dakin tubes are placed over the gauze in order that the area can be kept

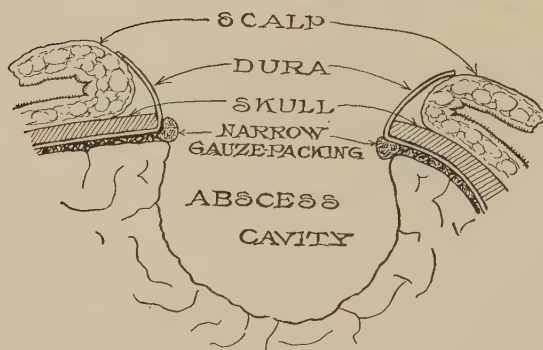
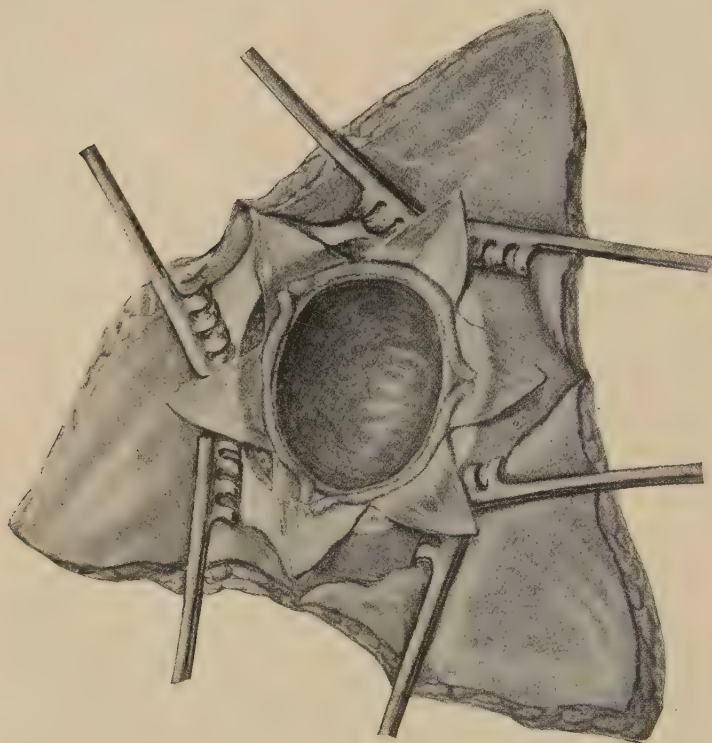


Fig. 358.—Three-limbed flap of soft parts reflected; bone removed over abscess cavity; stellate flaps of dura reflected over bony margins of skull; iodoform gauze insinuated between dura and cortex. The "roof" of the abscess cavity has been removed and can herniate or invert through the cranial defect. (King in *Surgery, Gynecology, and Obstetrics*.)

thoroughly wet with the solution, and a copious Dakin gauze dressing is applied. The ears, eyes, and scalp are protected by layers of vaseline gauze.

*After-treatment* is most important. Dakinization is commenced immediately, instillation being made every hour. Dressings are changed

every day by the operator or assistant. About the sixth day the hernia will form and assume a mushroom shape, overlapping the strips of iodoform gauze inserted at time of the operation which are now carefully removed. It is advisable to lightly place gauze strips, wet in Dakin's solution, between the flaps and the brain hernia so as to allow the flaps to first become gradually attached to the hernia at the base. When this takes place the sutures at the apices are removed, and the flaps are allowed to become adherent over the lateral surface of the hernia. The hernia should not be cut away or compressed. To prevent trauma, a ring made of cotton wrapped in gauze and strapped in place to prevent displacement is employed. Excessive herniation is prevented by lumbar puncture. Slight areas of superficial sloughing may occur. These should not be removed until completely loosened. After the first month healthy granulations will cover the hernia and epithelium extend from the margins of the skin flaps, and the size of the hernia diminishes. At this stage light pressure with perforated adhesive plaster is applied, and the frequency of the use of the Dakin solution lessened. The time required for the hernia to completely recede and the surface to become covered with epithelium is about three months (Fig. 359). After a period of a year a scalp plastic may be performed.

#### SURGERY OF THE SEVENTH NERVE

In the treatment of facial paralysis it is desirable that the activity of the muscles be regained by restoration of the function of the facial where this is possible. This will depend largely on the causative factor.

Paralysis of toxic origin, following exposure to cold or infectious diseases, usually recovers in a few weeks. When due to either an acute or chronic suppurative otitis media the appropriate mastoid operation should be done to remove the focus of infection. A facial paralysis with a chronic suppurative otitis media is suggestive of an erosion or necrosis of the fallopian canal and, if found, the canal should be opened and freed of granulation and cholesteatoma by careful wiping. Facial paralysis due to operative trauma, either from division of the nerve or injury from fracture of the fallopian canal, is not infrequent.

A paralysis coming on shortly after an operation, muscular contraction of the face muscles being noticed on recovery from the anesthetic, is not due to direct operative injury, but the result of pressure from a blood-clot or swelling of the sheath in the facial canal. When, however, paralysis is present when the patient regains consciousness, the probability of a permanent injury is great. A search should be made for the site of the injury,

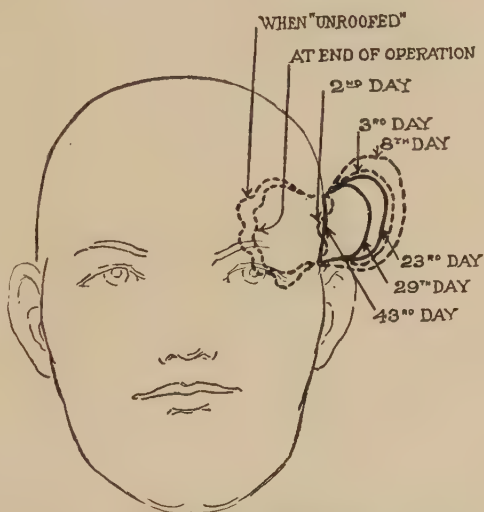


Fig. 359.—Schematic representation of progression and recession of brain hernia after "unroofing" operation. (King in Surgery, Gynecology, and Obstetrics.)

and the facial canal opened, and any spiculæ of bone that may have been driven into or between the ends of the severed nerve removed. In the absence of infection the nerve should be exposed as far as possible in both directions and an attempt made to join the two ends by a suture. In the presence of suppuration this would probably fail. Spontaneous recovery may take place in a divided nerve where the distance is not greater than the process of repair will bridge over. As a reuniting nerve invariably produces a swelling at the site of the reunion, it follows that the facial nerve, lodged within its canal, will in itself produce compression which makes functional restitution impossible unless space is provided for the process by opening the facial canal. This procedure is not contraindicated by the presence of sepsis in the tympanic cavity, as a passage has already been opened for infection by the operative injury.

The duration of the paralysis is variable; when due to pressure, recovery may be complete in a few weeks, whereas, in a lesion due to severe traumatism, the paralysis may persist for months and yet result in complete recovery.

When doubt exists whether the nerve will recover its function, it is best to delay three or four months before doing an anastomosis, and com-

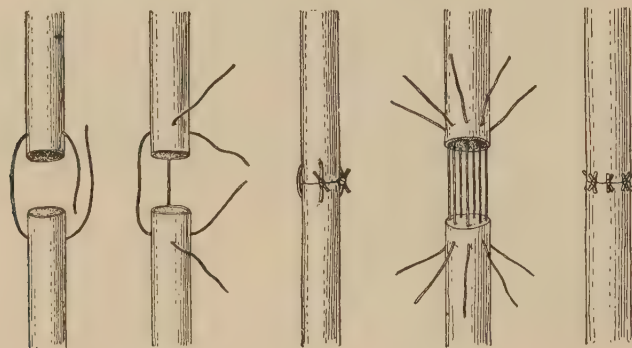


Fig. 360.—Method of suturing in end-to-end anastomosis of nerve trunks. (From Phillips, Diseases of Ear, Nose, and Throat, F. A. Davis Co., Publishers.)

bat the tendency to muscular atrophy by the daily use of electricity and massage. Any increased electrical reaction toward normal, or the retention of normal reaction, indicates that the treatment should be continued, as restoration by Nature is always better than that obtained by surgical means.<sup>27</sup>

**Indications for Nerve Anastomosis.**—With lessened muscular reaction or the signs of contractures, an anastomosis with another motor nerve offers the only hope for re-establishing the function of the peripheral branches of the injured nerve. In cases where no muscular response can be obtained with a strong current, or when complete atrophy of the muscles has taken place, no benefit would be derived from a nerve anastomosis.

The nerves available for anastomosis with the facial are the spinal accessory and the hypoglossal.

The two methods usually employed for nerve union are: (a) End-to-end anastomosis; (b) lateral implantation.

*End-to-end anastomosis* is simpler in technic and more fully restores muscular control to the paralyzed muscles. Its disadvantage is the loss of power to the muscles supplied by the peripheral fibers of the divided nerve (Fig. 360).

*Lateral implantation* is recommended by most American writers to overcome this drawback. This is done by grafting the peripheral end of the facial nerve into the trunk of the chosen nerve, through a longitudinal slit. This avoids extensive injury to muscles supplied by the receiving nerve, but restoration of the facial muscles is usually less complete than a successful end-to-end union. Ballance strongly condemns the lateral-implantation method for the facial nerve,<sup>28</sup> and overcomes the resulting paralysis of the muscles supplied by the spinal accessory or hypoglossal by a further anastomosis. If the spinal accessory is taken for the facial nerve, the peripheral end of the spinal nerve is joined to the descending hypoglossal nerve. When the hypoglossal is used, its distal end can be united with a portion of the spinal accessory, the descending hypoglossal, or a cervical nerve.

The successful lateral implantation of the facial with the hypoglossal results in restoration of facial expression, avoidance of atrophy of the muscles of the tongue on the same side, and favors disassociation of facial and tongue movements. When the facial is anastomosed with the spinal accessory, the end-to-end method is preferable; the distal end of the spinal accessory should, however, be joined with the descending hypoglossal. Co-ordinate movements of the shoulder and face muscles are permanent.

#### Technic of Facial-hypoglossal Lateral Implantation.

—An incision is made through the skin, fascia, and platysma, extending from the level of the stylomastoid foramen, along the anterior border of the sternocleidomastoid muscle, to the level of the thyroid cartilage. The anterior border of the sternocleidomastoid is freed and retracted backward, and the posterior belly of the digastric muscle divided when exposed. Dissect bluntly well down between the anterior border of the mastoid process and the parotid gland until the styloid process can be easily felt. The parotid gland is retracted forward and the facial nerve located at its entrance into the gland. Retraction of the gland stretches the nerve which can be felt as a ridge in the gland. Verify the findings by stimulating with an electric battery.

The nerve is traced and freed to its exit at the stylomastoid foramen. A black ligature is placed (not tied) under the nerve for identification and the hypoglossal searched for.<sup>29</sup>

The occipital artery is located by blunt dissection, where it is given off from the external carotid artery. This is facilitated by a curved incision

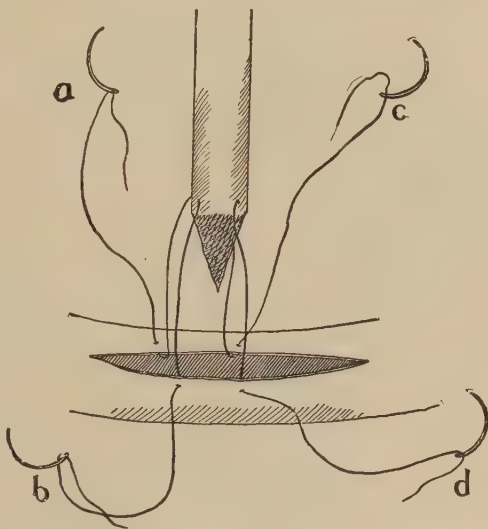


Fig. 361.—Showing the method of suturing in lateral implantation: *a-b* and *c-d*, Double-needled anchor sutures. (Ballinger, Diseases of the Nose, Throat, and Ear, Lea & Febiger, Publishers.)

from the upper and middle third of the first incision to the great cornu of the hyoid bone. The hypoglossal curves around the occipital artery to go transversely across the neck.

The occipital artery is divided between double ligatures and the hypoglossal carefully separated from the surrounding tissues as far posterior as possible, to allow its being brought forward to a point within easy reach of the facial nerve for lateral implantation.

Ascertain if the facial is of sufficient length to graft into the hypoglossal without traction. If not, remove the mastoid tip and open the facial canal for  $\frac{1}{2}$  inch to gain increase in length.<sup>30</sup> For an end-to-end anastomosis the hypoglossal must be freed forward to the hyoglossus muscle before cutting the nerve.

The facial nerve is drawn from its canal as far as possible and severed close to the opening with small scissors.

A longitudinal slit is made in the hypoglossal nerve at a point nearest to the facial, to receive the facial stump. The facial stump is trimmed of its sheath so that its axis-cylinders will be in direct contact with those of

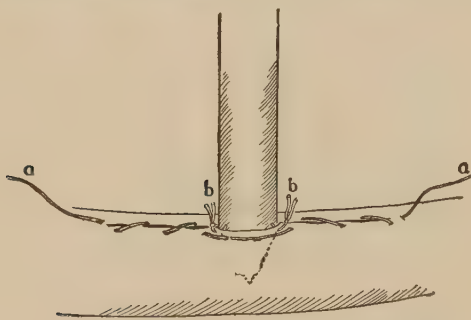


Fig. 362.—*b-b*, Anchor sutures holding the implanted facial nerve in position. *a-a*, Loose running sutures closing the longitudinal incision in the receiving nerve. (Ballinger, *Diseases of the Nose, Throat, and Ear*, Lea & Febiger, Publishers.)

the hypoglossal (Fig. 362). A very fine silk suture with a small round needle on each end is passed through each side of the sheath above the stump, and each needle passed through the sheath of the hypoglossal from within the incision outward. The slit in the hypoglossal is opened with a pair of fine spring forceps and the facial stump entered, and the anchor sutures tied with great care. A secondary continuous suture should be passed through the lip of the hypoglossal incision to close it on each side of the facial and drawn tightly

but not tied. Cover the anastomosis with Cargile membrane or a muscular flap. Reunite the digastric muscle and close the wound.

**Facial and Spinal Accessory End-to-end Anastomosis.**—The same incision along the anterior border of the sternocleidomastoid, as before described, is used. When the anterior border of the sternocleidomastoid muscle is freed, the spinal accessory nerve can be felt as a ridge where it enters the sternocleidomastoid muscle, about 2 inches below the mastoid process. On retraction of the muscle the nerve is easily cleared of the fascia to the level of the transverse spinous process, and marked by a ligature passed beneath it.

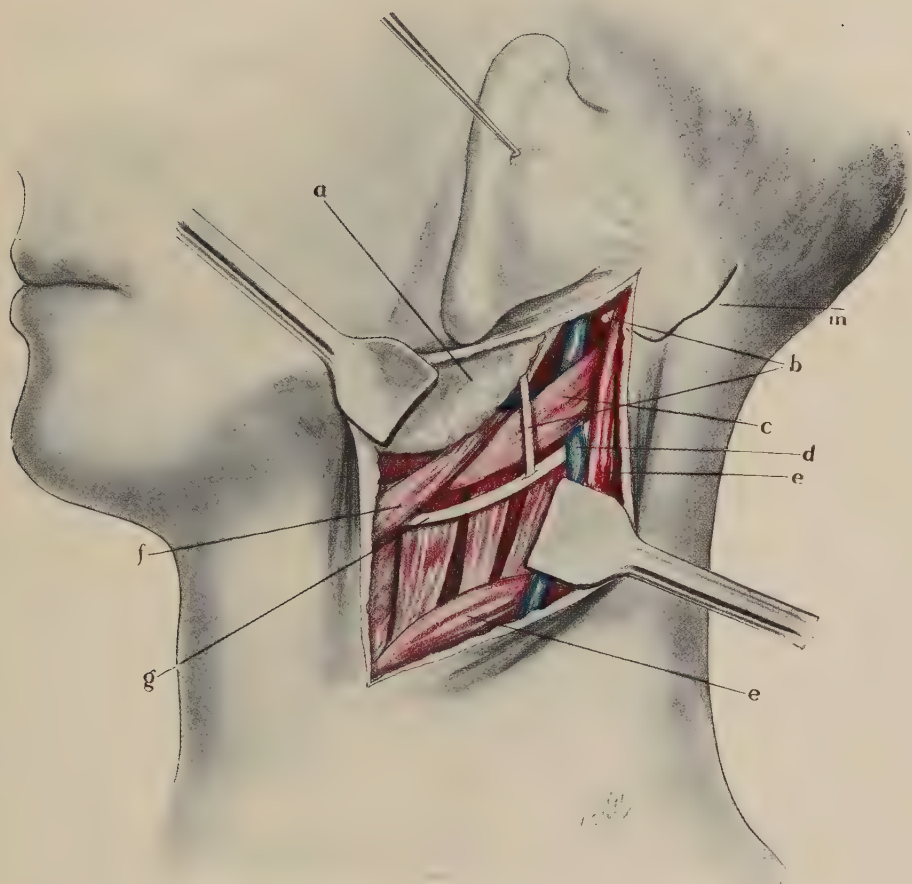
The facial and hypoglossal nerves are exposed as in hypoglossal anastomosis, and the facial marked with a ligature beneath it.

Locate the descending branch of the hypoglossal where it leaves the main nerve and passes downward on the common carotid artery, and mark with a third ligature.

Ascertain if the facial and spinal nerves will meet without tension and, if not, open the facial canal for  $\frac{1}{2}$  inch before cutting the facial.

Divide the spinal accessory before it enters the sternocleidomastoid

# PLATE VI



The Anastomosis of the Facial with the Hypoglossal Nerve. *a*, The parotid gland; *b*, the stump of the facial and the facial anastomosed with (*g*) the hypoglossal nerve; *c*, the posterior belly of the digastric muscle; *d*, the external jugular vein; *e*, the sternomastoid muscle retracted to expose the hypoglossal nerve; *f*, the omohyoid muscle; *g*, the hypoglossal nerve; *m*, the mastoid process. (From Ballenger "Diseases of the Nose, Throat and Ear," Lea & Febiger, Publishers.)



muscle. Trim the ends of the two nerves so they will accurately approximate end to end, unite with two or three fine silk sutures, and cover the union with Cargile membrane. The descending branch of the hypoglossal is now divided at a level that will permit its approximation with the peripheral end of the severed spinal accessory nerve where it enters the sternocleidomastoid muscle, to which it is united by the same technic as employed to unite the central end to the facial. The ends of the digastric muscle are sutured together and the wound closed without drainage.

**After-treatment.**—The judicious use of electricity and massage will materially aid in recovery, but both the patient and surgeon must be prepared to wait for months for full restoration of function.

E. W. DAY.

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## DISEASES OF THE INTERNAL EAR

### (*Labyrinthitis*)

Diseases of the labyrinth or internal ear have been classified as primary and secondary on the assumption that some of the inflammations might be regarded as primary. Politzer<sup>1</sup> speaks of diseases of this section of the ear that arise "idiopathically or without discoverable cause," and mentions among them "primary hemorrhages and inflammations . . . on the whole, rare"; but on good authority it can be stated that no case of undoubted primary disease of the labyrinth has yet been reported.<sup>2</sup>

**Etiology.**—The etiology of labyrinthitis is varied. The older observers described minutely the non-suppurative inflammations of the labyrinth, particularly those due to external "harmful influences" such as cold, sun-strokes, diving, indirect injuries to the labyrinth and traumatic influences, hyperemia, and hemorrhage.

Hyperemia and hemorrhages in the labyrinth may be due to sudden changes in air pressure in the middle ear, as in caisson workers, divers, and aviators; and the effect on the labyrinth of the shock from detonations, explosions, and gunfire has been definitely shown.<sup>3</sup> Peculiar affections of the labyrinth are found to be prevalent among boiler-makers, blacksmiths, locksmiths, plate workers, and others whose ears are subjected to the repeated action of noise and sound-waves.

The writer reported the case of a man in whom external sounds and the sound of his own voice produced vertigo and nystagmus in the plane of the right superior semicircular canal. The cause of the hypersensitiveness to sound in this locality was not discovered, and it was only relieved by removal of the canals and obliteration of the labyrinth on that side.<sup>4</sup> There are cases authentically reported in which tinnitus and marked deafness have followed mental disturbances. Politzer speaks of such a case caused by fright in which he attributed the disturbance in the labyrinth to the effect of the mind on the vasomotor nerves (vasoconstriction and dilatation).

Circulatory disturbances, by producing hyperemia or anemia of the labyrinth, exudations, hemorrhages, or nutritive changes, have been found to cause temporary or permanent disease of the labyrinth, depending on the severity and permanence of the disorder. The peculiar susceptibility of the auditory nerve, more than any other nerve, to the action of drugs circulating in the blood, is well known. Quinine, salicylic acid, morphine, chloroform, tobacco, and alcohol often cause temporary disturbance of function which may become permanent when they are used in large doses over a long time. Kirchner's experiments with quinine and salicylic acid in rabbits, referred to by Politzer,<sup>1</sup> produced hemorrhage in the labyrinth which he attributed to vasomotor disturbances conducive to stasis and exudation produced by those drugs.

Distressing functional disorders of the labyrinth may abnormally persist in susceptible individuals as a result of mechanical disturbances, such as those met with in the rotation and caloric tests, and in car- and sea-sickness. Inflation of the middle ear with the eustachian catheter has caused disturbance of the labyrinth through sudden change in the intratympanic air pressure. These disturbances of function occur without apparent injury of any sort to the structures of the labyrinth.

There are other conditions which produce functional disturbances of the internal ear and signs of labyrinthine irritation, but beyond a hypersensitiveness, tests reveal no organic change during the attack. Similar transient, or recurrent, disturbances that sometimes become permanent are attributed to intestinal toxemia, and the toxemia from focal infections in tonsils, teeth, and elsewhere in the body. A reasonable assumption based on clinical experience is the basis on which such a diagnosis is made. The same can be said of hemorrhage and exudation in the labyrinth. Ménière's disease, the name applied to the group of symptoms found by Ménière to be the result of hemorrhage into the labyrinth, cannot be diagnosed with the same certainty in

the ear as intra-ocular hemorrhage can be with the ophthalmoscope in the eye.

Syphilis, leukemia, and cerebrospinal meningitis, the exanthemata, nephritis, mumps, and other diseases are known to produce labyrinthine complications that may end in its partial or total destruction. In fracture of the base of the skull the line of fracture may pass through the labyrinth. Hemorrhage into the labyrinth may result from other head injuries. Penetrating wounds of the labyrinth, from pistol shots and stab wounds from knives and hat-pins, sometimes occur.

In a paracentesis or myringotomy for middle-ear abscess, injury to the outer wall of the labyrinth or dislocation of the stapes may occur. Accidental dislocation, or removal of the stapes, sometimes occurs in ossiculectomy, with subsequent infection of the labyrinth.

In the simple mastoid operation the semicircular canal may be cracked or entered. In the radical mastoid operation the stapes may be dislocated, a semicircular canal or the promontory cracked or penetrated.

The danger of labyrinthitis developing from dislocation of the stapes or injury to the tissue around the oval window during the radical mastoid operation is not inconsiderable. (In Dr. John D. Richard's opinion, dislocation of the stapes, when it occurs during the radical mastoid operation, is so often followed by labyrinthitis and fatal meningitis that when it occurs he advocates immediate opening of the labyrinth to lessen the probability of extension into the meninges.)

The writer once, during a radical mastoid operation, found the stapes so loosened in the oval window by surrounding cholesteatoma that it fell away from its seat without rupture of the window or any subsequent change in the activity of the labyrinth.

**Types.**—The types of labyrinthitis have been divided into *metastatic*, *meningeal*, and *tympanic*.

In the *meningeal* cases the infection spreads from the meninges to the labyrinth through the (1) internal auditory meatus; (2) aqueductus cochleæ; (3) aqueductus vestibuli (rarely); (4) necrosis in the inner labyrinthine wall; (5) hiatus subarcuatus.

Poltizier calls attention to the fact that the meningeal cases are more prevalent in children, because of the freer communication that exists between the meningeal spaces and the labyrinth in children as compared with adults.

The most common cause for this type of labyrinthitis is epidemic cerebrospinal meningitis. From 15 to 50 per cent. of the cases of cerebrospinal meningitis are said to result in deafness. In 19 temporal bones, from 10 patients who died from cerebrospinal meningitis, Goerke<sup>2</sup> found inflammatory changes in the internal ear in 17. Meningitis that has been caused by middle-ear infection on one side may spread to the other side and infect the opposite labyrinth.

In a case from the writer's practice, operation for acute middle-ear abscess and mastoiditis was followed on the third day by slowly developing facial paralysis and signs of meningitis, with sudden loss of hearing four days later in the opposite ear. The hearing was markedly impaired in the ear operated on though not entirely lost. As there was never any sign of vestibular irritation, invasion of the meninges was thought to have taken place along the facial nerve, and loss of hearing in the opposite ear to be

due to affection of the auditory nerve without invasion of the static labyrinth.<sup>5</sup>

The pathways of infection between the labyrinth and meningeal spaces are the internal auditory canal and the aqueductus cochleæ. Cases in which infection passes through the aqueductus vestibuli are rare.

The *tympanic* type of labyrinthitis develops from extension of infection or inflammation from the middle ear.

The middle-ear inflammation may be acute and sometimes of only a few hours duration when signs of labyrinthitis appear. More often, however, it is from the acute exacerbation of chronic middle-ear suppuration than from the acute or subacute form.

In the tympanic cases infection from the tympanum may pass: (1) Through the oval window; (2) the round window; (3) fistula in one of the semicircular canals; or (4) fistula in the promontory.

The involvement in the labyrinth begins at the site of invasion and may become walled-off and limited to that part as *circumscribed labyrinthitis*, or the infection may spread through the whole labyrinth and cause *diffuse labyrinthitis*. The character of the inflammation may be *serous* or *purulent*.

In *circumscribed labyrinthitis* it is possible for almost any part of the labyrinth to be affected. The involvement begins where the invasion occurs, most often in the external semicircular canal because of that canal's proximity to the mastoid antrum, and the erosive action on it of cholesteatoma that has a tendency to form there. While this is the most usual way for infection of the labyrinth to occur it is the least dangerous, for the invasion is necessarily slow through the hard bony wall of the canal and, owing to the small lumen and the attachment of the membrane at the ampulla, the infection is more apt here to become walled-off than anywhere else in the labyrinth.

The perilymph spaces may become involved to a greater or less extent, but the infection may not pass beyond them to involve the endolymph.

Infection through the oval window, on the other hand, is less apt to become circumscribed. Nevertheless, when the inflammatory progress is gradual and the infection not too virulent, it may pass through the oval window and be confined to the cisterna perilymphatica of the vestibule. Inflammation in the cochlea, when circumscribed, is said to be usually located at the first half of the basal whorl. The explanation of this, given by Ruttin, is the mechanical arrangement of the cochlea and the position of the scala vestibuli of the basal whorl below the oval window through which the infection passed. Infection through the round window reaches the scala tympani and, if mild, may be confined to the lower half of the basal whorl. Consequently, it has been observed<sup>2</sup> that a graver prognosis in regard to the return of function would be indicated (if it were possible for the fact to be known) when infection passes through the oval window than when it passes through the round window, for the reason that in the former both vestibule and cochlea are involved, and in the latter the cochlea alone is infected. It is not likely, however, that during the life of the patient it would be discovered whether or not the invasion was through the oval or through the round window.

*Perilabyrinthitis* is the term generally used to describe that condition in which the cellular bone around the labyrinthine capsule is inflamed, as

it may be in acute mastoiditis, or in an acute exacerbation of chronic middle-ear disease. With it facial paralysis sometimes occurs, and vestibular symptoms attributed to congestion through more or less involvement of the bony labyrinthine capsule itself. The term is also applied to inflammation of the bony capsule with possibly mild involvement of the perilymph spaces, as might be the case in fistula of the semicircular canal.

In *fistula of the canal* the external semicircular, because of its proximity to the aditus and mastoid antrum, is the one most frequently attacked. Its prominent external wall is slowly eaten away down to the endosteum or lining of the canal. During this stage the bony labyrinth or bony capsule of the labyrinth is the only part involved. When the endosteum is broken through, the perilymph spaces are entered but, owing to the gradual progress of the inflammation and the inflammatory thickening of the endosteum and septa between it and the membranous canal, the lesion may become walled-off or circumscribed and be prevented from spreading throughout

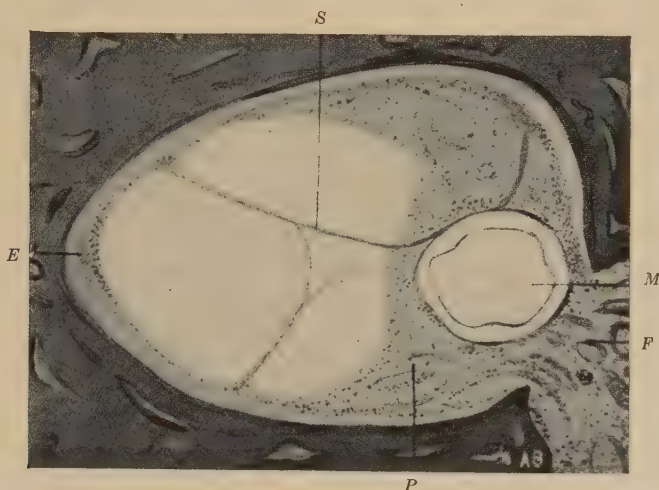


Fig. 363.—Fistula in external semicircular canal. Suppurative perilyabyrinthitis. Membranous canal is normal. *F*, Fistula in bony capsule filled with granulation tissue. Endosteum is destroyed in this location. *M*, Normal membranous canal. *P*, Granulation tissue and pus in perilymph space. *E*, Endosteum. *S*, Connective-tissue septum in perilymph space. (After Braun and Friesner.)

the labyrinth. Sections have been made that show this involvement of bone and perilymph spaces with the walls of the membranous canal and endolymph uninvolved<sup>2</sup> (Fig. 363). The membranous canal may later give way and infection invade the endolymph, through which it may spread throughout the whole labyrinth and produce an acute or chronic diffuse labyrinthitis.

*Diffuse labyrinthitis* may be *serous* or *purulent*. "Serofibrinous" is a term also used for a certain variety, though clinically this cannot be differentiated from the former with certainty during an attack. In *diffuse purulent labyrinthitis* the bone may or may not be involved. *Panlabyrinthitis* indicates involvement of the whole labyrinth, bony as well as membranous. *Panotitis* is the name applied to inflammation that involves the middle ear and labyrinth, either simultaneously or in rapid succession. *Diffuse serous labyrinthitis* may be secondary to a circumscribed labyrinthitis with a

fistula, or it may occur with the bony wall intact. That which occurs with the labyrinthine wall intact is thought by some to be due to inflammatory edema of the labyrinth from inflammation in the middle ear. It is also thought to be due to extension of infection in the middle ear through the minute vessels in the outer labyrinthine wall.

From cases reported of acute purulent inflammation of the middle ear, in which on section bacteria were found accumulated in the recesses of the oval and round windows, it is thought that toxins elaborated by these passed into the annular ligament and through the membrane of the round window into the perilymph and endolymph spaces to cause in them inflammation and exudate that, however, were free from bacteria.<sup>2</sup> "Toxic labyrinthitis" has been suggested as a name for this condition though it adds confusion to the thought when other forms of toxic irritation of the labyrinth are considered. If necrotic changes take place in the bony capsule and perforation occurs, the bacteria break through and cause purulent labyrinthitis. Serous inflammation of the endolymph spaces may be induced from infection in the perilymph spaces just as it is induced from purulent middle-ear infection.

The frequency of labyrinthitis is said to be one in every 100 cases of middle-ear suppuration.<sup>2</sup> Diffuse, serous, or serofibrinous labyrinthitis is usually acute, resolving in a week or two or resulting in either partial or complete destruction of the labyrinth.

*Diffuse purulent labyrinthitis* may be acute or chronic. In the acute fulminating cases the bone destruction is rare. In the chronic cases some bone destruction usually occurs and, with necrosis of the labyrinth, 80 per cent. are said by Neumann to develop facial paralysis. In this type of labyrinthine invasion, the infection usually passes through a rupture in the labyrinthine wall, but there has been evidence found that bacteria may pass through an unruptured annular ligament or an unruptured round window to cause purulent labyrinthitis.<sup>2</sup>

Return to normal may occur in the serous and serofibrinous forms. A certain number, however, as well as a large number of the purulent cases, have atrophic and obliterative changes. In a large proportion of the purulent cases the exudate in the labyrinth becomes organized and filled with connective tissue in which bony transformation may occur in time.

Non-suppurative diseases of the labyrinth are difficult to distinguish from affections of the auditory nerve itself, and many disturbances attributed to lesions in the end-organ may be due to irritation of the fibers leading from the end-organ.

Symptoms of eighth nerve affection may simulate those of the labyrinth. Neuritis may develop from many causes that have no connection with the middle ear or the usual causes of labyrinthitis. In such cases the cochlear and vestibular fibers are apt to be affected separately and not in combination. When the stimulation or destruction of both together develops rapidly it is more indicative of labyrinthine involvement than of retrolabyrinthine disease.

By the caloric test the comparative sensitiveness of the two labyrinths can be determined, and by the galvanic test the irritability of the two vestibular nerves can be compared. A neuritis which involves both divisions of the eighth and, with it, the facial nerve is often found to be due to syphilis.<sup>2</sup>

**Symptomatology.**—As the functions of the auditory and static portions of the labyrinth are separate and distinct, it is possible for signs of disturbance in one portion to be present without signs of the disturbance in the other. In the cochlea, nerve irritation manifests itself by hypersensitiveness to sounds, subjective noises, or impairment of hearing, and nerve destruction by deafness and tinnitus. In the static portion the symptoms of nerve irritation may be so similar to those of nerve destruction that the differentiation between the two may be difficult. Both stimulation and destruction of the static labyrinth cause vertigo, ataxia, nausea, vomiting, and nystagmus, for normally the labyrinths on each side respond equally and simultaneously to stimulation, and if there is hypersensitiveness, irritation or sluggishness of one side, an imbalance is produced which gives rise to symptoms of labyrinthine disturbance. When the difference in the action of the two sides is caused by irritation or stimulation of one side, it has been called a *stimulation disharmony*. When the disharmony is the result of destruction or paralysis of one side, it has been called a *destruction disharmony*.<sup>2</sup>

Irritation or destruction of the vestibular apparatus, whether artificial or from acute labyrinthine disease, is invariably accompanied by spontaneous nystagmus, vertigo, and disturbance of equilibrium.

As nystagmus may occur with certain ocular lesions, with cerebellar abscess, with tumors occupying the posterior fossa of the skull and as a physiological anomaly, it is important to distinguish from nystagmus of this kind—the true *vestibular nystagmus*.

Vestibular nystagmus is characterized by a slow movement in one direction and a quick movement in the opposite direction. The significance of the slow movement and its association with the movement of endolymph will be recalled. The quick is a recovery movement only. Nevertheless, the direction of the nystagmus has been from the beginning designated from this conspicuous, though less significant, movement. Where there is overwhelming involvement of all nerve endings in one labyrinth, the nystagmus is rotary and to the opposite side because of the overbalancing influence of all the canals on that side. When the overbalancing or disharmony, however, is due to the overstimulated end-organ, the nystagmus is in the plane and toward the canal in which the chief impulse rises.

In a mild disturbance the nystagmus may be noticeable only when the eyes are turned extremely in the direction of the rapid nystagmic movement. It is less and may even disappear when the eyes are turned as far as possible in the direction of the slow movement. First, second, and third degree nystagmus are the terms used. The third degree is that applied to the exaggerated form, or that in which the nystagmus persists no matter in what position the eyes may be.

Turning the eyes in the direction of the slow component always retards the nystagmus, while turning them in the direction of the quick component increases the movement and excites the other labyrinthine symptoms.

*Vertigo* as a symptom of labyrinthitis is rotary in character, and with it is the subjective impression of the rotation of surrounding objects always in the plane corresponding to the plane of the nystagmus.

The tendency of the body to fall in the direction of the slow component follows, no matter in which direction the head is placed. This is important in determining whether disturbances of equilibrium are of vestibular origin or not. If the tendency to fall is not in the direction of the slow component

of nystagmus, the disturbance should not then be considered of vestibular origin. An exception to this is shown by Kerrison to exist when the person's head is lowered forward to an angle of 90 degrees, the head then in following the tendency to rotate in the direction of the slow movement causes the body to fall in the direction of the nystagmic movement.

Bárány's law is stated as follows: "(1) Spontaneous vertigo of vestibular origin is always accompanied by some degree of spontaneous vestibular nystagmus, and is always increased when the eyes are voluntarily turned in the direction of the quick nystagmic movement. (2) Vestibular ataxia is always accompanied by nystagmus, and is always influenced by the position of the head. (3) A person exhibiting vestibular nystagmus tends to move within the plane of the nystagmus, and to fall in the direction opposite to the quick nystagmic movement." Kerrison suggests that a better understanding of the principle involved will be obtained, if it is said that "A person exhibiting vestibular nystagmus tends to rotate within the plane of the nystagmus and in the direction opposite to the quick eye movement," and that "this tendency to rotation is about an axis passing through his head, and he falls, or tends to fall, in the direction in which this rotation throws his body, and this, as we shall see, is not always in the opposite direction to the quick nystagmic movements."<sup>6</sup>

A person with vestibular nystagmus to the left tends to fall to the right. If the head is turned to face the left, he falls forward which is in the direction of the slow nystagmic movement. But if the head is bowed to an angle of 90 degrees, it tends to rotate in the direction of the slow movement and to cause the body to fall not in that direction but in the opposite one. In vertigo from other causes, cerebellar abscess for example, the tendency to fall is independent of the nystagmus.

It is believed that the disturbance that results from the destruction of the labyrinth is due to an overbalancing of the impulses from the remaining sound side, notwithstanding the fact that this view does not seem to agree with observations made on experimentally destroying first one and then the other labyrinth in the same animal. When one labyrinth is destroyed typical disturbances of equilibrium follow, and nystagmus is directed to the sound side. When, after a time, compensation for the loss of this labyrinth takes place and the animal no longer shows signs of imbalance or disharmony, if this remaining labyrinth is destroyed or the eighth nerve is severed, disturbances of equilibrium as severe as those which followed the first operation will occur, and the nystagmus will be directed to the side on which the labyrinth was destroyed first, despite the fact that this labyrinth has been for some time totally without function.<sup>2</sup>

Since this is the case it is argued that the disturbance cannot depend exclusively upon the impulses from the labyrinth. It seems, however, that it is quite definitely due in such a case to the loss of the normal impulses from the labyrinth, for while the sight, the muscle, arthrodial and tactile senses are used in orientation and are called upon to compensate for the loss of the labyrinths, the loss of one of them, sight for example, causes no disturbance of equilibrium when the labyrinths are normal but, when they are destroyed, sight plays a large part in compensating for their loss. When there is loss of sight, or darkness, with one or both labyrinths destroyed, further education and compensation through the muscle, joint, and tactile senses are called for.

Kerrison<sup>6</sup> describes the experiments of R. T. Slinger and Sir Victor Horsley in measuring the muscular and arthroclial senses, and quotes their conclusions that "if the information gained by sight is permanently blotted out, the muscular sense under necessity can by education be brought to a point at least one-fourth better than that learned by the normal person."

In the writer's case of paracoustic vertigo referred to,<sup>4</sup> in which not only an active but a hypersensitive labyrinth was removed, the severest vertigo, vomiting, and nystagmus with great prostration followed and continued for a week or more. The patient was fully cautioned and warned of the danger of losing his balance unexpectedly any time he happened to make a movement he had not prepared for. Shortly after he left the hospital, on crossing the street, he looked up suddenly and consequently sat down in the middle of the street. His compensation, however, in time became apparently complete but, through overconfidence and in spite of warning, he lost his life in an accident several years later as the result of driving his automobile off a mountain road *at night*.

When both the labyrinths are destroyed simultaneously no nystagmus occurs because the impulses aroused through the simultaneous destruction of both end-organs oppose each other completely in the same way that caloric or galvanic irritation of the labyrinths applied simultaneously neutralize each other. However, with totally destroyed end-organs, disturbance of equilibrium may occur from irritation or some effect on the retrovestibular paths.

(Bárány and Voss believe that the disturbances in equilibrium that follow the labyrinthitis and deafness, which complicate cerebrospinal meningitis, are due to the involvement of the cerebellar cortex, and not to the destruction of the labyrinth.<sup>2</sup>)

Before any marked involvement of the labyrinth occurs symptoms of labyrinthine irritation may be caused by perilabyrinthine involvement from mastoid inflammation, in which case with active functional tests of the labyrinth there may be symptoms of vertigo, nausea, and vomiting with nystagmus to the irritated side. This may even occur from retention of fluid in the middle ear in acute middle-ear abscess and be relieved by myringotomy. It may follow the radical mastoid operation and be relieved by the removal of tight packing from the cavity. With involvement of the labyrinth itself, however, the nystagmus in the majority of cases is directed to the sound side. Wide variation in the intensity of the vertigo and ataxia occurs. With gradual loss of function in the labyrinth, only a slight unsteadiness may be noticed and no nausea or vomiting.

In *diffuse labyrinthitis*, as in sudden destruction of the labyrinth, there are usually violent vertigo, nausea, vomiting, tinnitus, and deafness. The nystagmus is rotary in character with the quick movement directed to the sound side. It is similar to that produced by irrigating a normal ear with cold water. The patient tends to fall in the direction of the slow movement, or, when standing, usually to the diseased side. Lying in bed, the nausea and vertigo are increased by any movement of the head, and the patient lies in a characteristic position with head and body turned as far as possible in the direction of the quick nystagmic movement. Even after several days, when the symptoms are beginning to subside, they will return if the eyes are turned in the direction of the quick nystagmic movement. As compensation for the destruction of the labyrinth takes place, the symp-

toms subside, and in time even rotation tests will fail to show great difference in the duration of the nystagmus after rotation to the right and to the left.

In *circumscribed labyrinthitis* it rarely happens that the infection is limited to the cochlea. When the invasion takes place in that region it almost invariably spreads to the static labyrinth, and symptoms of general labyrinthine involvement develop.

When the invasion is the result of erosion of the external semicircular canal the hearing may remain present, but the symptoms of vestibular disturbance are usually active and similar to those that occur in diffuse labyrinthitis. The nystagmus is to the sound side, with the usual vertigo and ataxia. The severity of the symptoms depends on the rapidity of the involvement. The presence of hearing indicates that the process is limited to the static portion, and distinguishes the circumscribed from the diffuse type.

The acute symptoms gradually subside in a week or two, and during the latent or quiescent stage the vestibular reactions can be tested. They are usually found to be absent. In some cases, however, there may be a partial return of function and some response to the caloric test, and possibly the fistula reaction may be present.

The *fistula symptom* occurs as a result of disease gradually thinning and perforating the bony wall of the labyrinth, and the site is usually the external semicircular canal. The membranous labyrinth remains intact, and increase or decrease of pressure over the fistula causes disturbance of the fluids of the labyrinth, and produces movement of the eyes and a sense of vertigo. When the fistula is exposed, the symptom can be produced by mechanical pressure over it. When it is not exposed, it may be produced by changing the air pressure in the external auditory canal. A convenient instrument for this purpose is the Siegel otoscope. Once the symptom has been recognized, it is dangerous to continue to produce it because of the possibility of rupturing into the membranous labyrinth and causing diffuse labyrinthitis.

*Diffuse serous labyrinthitis* occurs usually with a suppurative process in the middle ear. The symptoms are those of complete suppression of both the vestibular and cochlear functions, and it cannot be distinguished during the acute stage from diffuse purulent labyrinthitis. Later, there may be partial or almost complete return of function, while in the diffuse suppurative form there is permanent destruction. No definite distinction between the two can be made during life. If the caloric test reveals a partial or complete return of function *it is assumed* that the involvement was of the serous type.

In uncomplicated cases of labyrinthitis, there is usually no rise of temperature. A rise of temperature in a case of labyrinthitis that has signs of destruction of the labyrinth is strongly suggestive of involvement that has passed beyond the labyrinth to the meninges, and immediate opening of the labyrinth to promote drainage away from the meningeal spaces may possibly be indicated.

Symptoms are often no indication of the seriousness or permanence of the lesion. Profound functional disturbances may be transient and leave no permanent injury, while less disturbing symptoms may be followed by permanent loss of function.

With a still imperfect knowledge of the internal ear as the chief organ of balance, though with somewhat improved methods of testing the motility of the labyrinthine fluids and the sensitiveness of the nerve endings, in many cases hardly more than a reasonable surmise of the condition as it exists in the labyrinth can be made. Tests will show whether the labyrinth is functionally active or not, but whether the function will return when it has been lost cannot be determined. A surprising return of function sometimes occurs.

The case referred to, in which the stapes fell from the oval window at the time of the radical operation, was seen first in an active attack of labyrinthitis with vertigo, vomiting, and nystagmus to the affected side. This was followed shortly by evidence of total loss of function in the ear and nystagmus to the sound side. Total loss of hearing had occurred. There was no caloric reaction to prolonged use of heat or cold. Nevertheless, two months later when the case returned for operation on the diseased ear, there was an active caloric reaction and considerable return of hearing.

Great caution should be observed in testing cases with acute labyrinthitis. Except in chronic or latent cases, tests that involve manipulation and disturbance of the patient should not be used, for, aside from causing discomfort, there is danger in their use of increasing the possibility of extension of the infection to the meninges. Dr. Arthur B. Duell<sup>7</sup> particularly calls attention to the fact that such tests are not necessary for diagnosis in the acute cases. The Bárány noise apparatus will determine whether the cochlea is functioning or not, and the caloric test the condition of the static labyrinth. The fistula test, if positive, should not be used a second time. Rest and sedatives are of prime importance until it is decided whether or not surgical interference is indicated.

**Treatment.**—Rest in bed in quiet surroundings is essential in the treatment of acute labyrinthitis, and careful observation is necessary to determine whether or not surgical interference is indicated. Drugs that might mask the signs of beginning meningitis should be avoided if possible. However, the extreme irritability and nervousness that accompany the discomfort from such vertigo and nausea necessitate the use of sedatives. Bromides or chloral by mouth or rectum are often more satisfactory than morphine or codeine. An accurate record should be kept of the temperature and careful watch for any approaching sign of meningitis. Frequent leukocyte counts are of value. An increase in the number of leukocytes, like a sudden rise of temperature, is highly suggestive of dangerous meningeal involvement. Frequent lumbar punctures are sometimes advocated in order that early signs of meningitis through spinal cell counts may be detected. In the writer's opinion, however, the possibility of inducing the passage of infection from the labyrinth to the meninges through reduction of intraspinal pressure is not inconsiderable and, therefore, the withdrawal of spinal fluid is dangerous, particularly in the early stages of labyrinthitis before the avenues between the meningeal spaces and the labyrinth have had time to become occluded. Tests and examinations that are disturbing to the patient should be avoided for they not only add to the discomfort of the patient but increase the possibility of meningeal involvement.

Simple hyperemia, or anemia, of the labyrinth is apt to be transient, and a return to normal may be expected when the cause of it is removable or amenable to treatment. Some cases of vertigo and nystagmus, in which

the cause is obscure, are relieved for a varying length of time by lumbar puncture and the withdrawal of 15 to 20 c.c. of cerebrospinal fluid. The good effect, when it is obtained by this procedure, has been attributed by Bárány to the breaking up of adhesions that may be present in the cerebello-pontine angle by the reduction of intraspinal pressure.

In certain cases of persistent vertigo operation on the labyrinth itself has been advocated by Lake and others. In the writer's case of violent paracoustic vertigo, some relief for two or three months was gained by lumbar puncture, but permanent relief was afforded only when the labyrinth was destroyed.

*Ménière's disease* or hemorrhage in the labyrinth gives the classical symptoms of a sudden attack of labyrinthitis, and in the absence of middle-ear suppuration the diagnosis may be reasonably assumed in cases where the general physical condition indicates weakness of the blood-vessels or excessive vascular strain. When signs of labyrinthitis are associated with *leukemia*, *nephritis*, *syphilis*, *drug poisoning*, *toxemia from various sources*, etc., the direct treatment of the labyrinthitis is secondary to the treatment of the disease to which it is due.

When hemorrhage or exudate in the labyrinth is suspected, in addition to the complete rest and quiet always indicated until the acute symptoms have subsided, cold compresses or other local applications comforting to the patient may be applied to the head and neck, and later, in the second or third week, potassium iodide and pilocarpine sweats are often given to aid absorption and increase elimination.

The *complications of labyrinthitis* occur as a result of intracranial extension along the avenues already mentioned. Meningitis, brain abscess, cerebellar abscess, sinus thrombosis, or facial paralysis may occur.

Facial paralysis is said by Neumann to occur in about 80 per cent. of the cases with labyrinthine necrosis.<sup>2</sup>

When the infection passes rapidly through the labyrinth to the intracranial cavity, it usually results fatally in a diffuse purulent leptomeningitis. If gradual, it may become circumscribed in a cerebellar, epidural, or intradural abscess. Extension usually occurs along the auditory nerve or through the aqueductus cochleæ, in which case purulent meningitis usually results. When it occurs through other channels, such as the aqueductus vestibuli, the fossa subarcuata, through necrosis of the superior or posterior surfaces of the petrous pyramid or by metastasis, the process may be sufficiently gradual to become localized.

In the case referred to, in which meningitis was preceded by slowly developing facial paralysis with apparently no disturbance of the static labyrinth, there was reason to believe that the passage of infection was along the facial nerve. Fortunately, the avenues of communication that lead from the labyrinth to the meningeal spaces are often closed by inflammation, and the infection is confined to the labyrinth. Otherwise, every case of diffuse purulent labyrinthitis would end in meningitis.

The diagnosis of labyrinthitis *per se* is not difficult as the symptoms are generally known and easily recognized. The differential diagnosis between intracranial complications of labyrinthitis and other intracranial lesions is more difficult.

The internal ear as a key to intracranial diagnosis becomes of greater importance as the pathways from it to the cerebral centers become more

definitely traced. As the chief organ of balance, these pathways are numerous to the many centers from which are distributed impulses that govern the whole body. A lack of normal response to vestibular stimulation along a certain line may lead to the discovery of a lesion along that line.

The lesion may be central or peripheral. If it is peripheral the labyrinthine tests will very likely reveal it. If central, certain observations have been made that are now regarded as having definite significance in relation to them.<sup>8</sup> Spontaneous vertical nystagmus upward is believed to be indicative of a central lesion.

Lyons reports 9 cases of supratentorial tumors, diagnosed independently by the Bárány tests and proved either by operation or necropsy.<sup>9</sup> Loss of vertigo responses suggests disturbance in the cerebellum, and the so-called "angle lesion phenomenon complex," consisting of deafness and total failure of vestibular responses on the affected side, together with, on the opposite side, failure of the vertical responses but with good responses from the horizontal canal, has been found so often in proved angle lesions as to be regarded as a most important diagnostic sign.<sup>8</sup>

The response to stimulation in the vertical canals, however, is always less active than the response in the horizontal canals, and loss of reaction in these canals is sometimes met with where there is probably no organic central lesion.

While such phenomena cannot properly be included among the diseases of the labyrinth, reference here to some of them may excite an interest to find a proper presentation elsewhere.

JOHN R. PAGE.

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#### TESTS OF THE VESTIBULAR APPARATUS

This presentation of the subject of functional testing of the so-called "vestibular apparatus" aims: (1) To point out the most important items concerning its functions; (2) to describe procedures required to assemble necessary findings; and (3) to aid in understanding their meanings concerning the patient's condition. Text-book and monograph subject matter

includes such a multitude of observations, emanating from such a large number of sources, and representing disjointed attempts to arrive at conclusions from such widely differing viewpoints, that an attempt to reduce this available material to any semblance of orderly consideration would be beyond the objects of the chapter.

**General Considerations.**—In order to test any mechanism one must have some reliable indicator by whose use results of testing may be observed and compared. In the case of pneumatic or hydraulic mechanism some form of gauge may indicate pressures accurately; in the case of electric mechanism, some form of voltmeter or amperemeter. In the case of nerve mechanism, such as the internal ear apparatus, no gauge or meter can suffice. One must use the living sensory and motor mechanisms in conjunction with any physical means of testing. Our knowledge of the ear mechanism is so meager it must be realized at the start that test measurements must prove difficult, unsatisfactory, and at best only roughly approximate; and further that a considerable amount of fundamental physiological information concerning living mechanisms must first be possessed before making any attempt to test the internal ear.

It is undoubtedly because of this discouraging aspect of functional ear testing that so few serious scientific studies of internal ear conditions are undertaken in ordinary otologic practice. It is true that certain tests have been worked out to the point of their being applicable somewhat according to "rule of thumb"; but it frequently happens that deductions drawn from these tests lead to erroneous conclusions, not because of the difficulties of testing or because the tests themselves are at fault, but because of the ease of misconstruing test findings. Certain it is that far less than the full measure of practical value is to be had from functional tests of the ear, unless the observer has previously become familiar with many necessary prerequisites to a real understanding of the findings. These prerequisites include, in addition to a working knowledge of physics, anatomy, physiology, and biophysics, a familiarity with the gross and microscopical anatomy of the entire ear mechanism, the vascular and hydrostatic conditions within the internal ear cavities and cranium, the comparative anatomy and embryology of the middle and internal ears, the pathology of the ear and its associated structures, and the physiology and pathology of the motor and sensory apparatus utilized in observing the effects of ear stimulation.

Functional testing of the ears involves two sense apparatuses, one concerned with motion, the other concerned with sound.\* To approach the

\* Certain writings relating to functions of the internal ear, in approaching the problems from different viewpoints, contain observations couched in such different terminology as to confuse the student. When one compares the written descriptions of observations and the comments made by physiologists, by physicists and by psychologists, the resulting impression is one of more or less radical differences. These differences are in many instances more apparent than real, at least with respect to fundamentals.

For instance, many psychological writings express doubts that the vestibular apparatus is a sense organ. Ogden states (Hearing, 1924, pp. 24-25): "The vestibule seems to be the most primitive organ of the ear, for we find analogous structures far down in the scale of animal life. . . a special sense of equilibrium is sometimes attributed to the vestibule and semicircular canals but no agreement has been reached on this point. . . The specific function of these organs as reflex mechanisms of equilibrium is, however, evident." At the same time he states clearly that it is "the organ of equilibration." Equilibration is not a sense function such as touch, nor a motor function such as contractility, nor indeed a *function* at all, but a complex *performance*, involving sense functions of many kinds and motor functions of intricate and complementary characters. The internal ear mechanism

subject from a more promising avenue it is better to say that the ear contains two end-organs, one normally stimulated by motion, the other by sound. In order to "test" an ear, the results of stimulating an individual ear must be observed and compared with known normal results.

In general certain truths are common to all end-organ apparatuses of the body. As the result of the operation of a living *physical mechanism*, such as the tympanic or the refractive mechanism, some specific form of exogenous energy is brought to bear upon end-organ cells. These cells thereupon discharge nerve impulses as the result of this stimulus; these impulses are transmitted by nerves to certain portions of the central nervous system, where they produce certain reactions and become available as conscious or subconscious information concerning that particular energy. In a civic community various manufacturing or industrial plants are constantly accomplishing transmutations of energy, as a result of which in-

undoubtedly occupies a very important place in the complex neuromuscular mechanism through the operation of which impulses originating in the internal ear find expression, on the one hand, in the reflex or automatic movement of a fin, or of a wing, or of an extremity; on the other hand, in equilibration.

Sensory motor nerve connections of various orders (from the simplest reflex arcs to the most complicated [subconscious] automatism "patterns," and [conscious] intention performance "patterns") are more or less intimately connected with the afferent pathways from the internal ear; many of these afferent impulses are constantly by-passing consciousness or attention in reaching normal effector goals; but it seems unavoidable to regard the internal ear apparatus itself as a special sense apparatus.

The obvious difference between the writer who states with authority that this or that mechanism is a sense mechanism and the writer who states, with equal authority, that it is not, lies in terminology. One writer may confine the use of the term "sense mechanism" to such mechanisms as *solely* subserve the sensing of some specific thing, as odor is sensed by the olfactory sense organ. Another may use the term to indicate a mechanism whose stimulation causes the emission of afferent nerve impulses.

The first authority may object to referring to the vestibular apparatus as the ear "motion-sensing" mechanism on the grounds that other totally different mechanisms such as the visual or the deep sensibility mechanisms *can sense motion*; and that, therefore, the term is misleading in that it infers that the ear mechanism is the *only* one sensing motion. However, it is true that this ear mechanism is unique in that it is stimulated *only by motion*—and this *under any and all conditions*—whereas the others are normally stimulated, specifically (deep sensibility) by the pull of gravity and (vision) by light, and they are able to sense motion *only incidentally and only under certain conditions*. It is true that stimulation of the vestibular end-organs produces not only sensory, but motor and other reflex results—vertigo, nystagmus, past-pointing, falling, sprawling, pallor, sweat, nausea, vomiting, and faintness. In this respect this mechanism differs at first thought from the usual conception of a special sense mechanism.

It is further true that secondary *sense* stimuli concerned somewhat in effecting a sense of movement are initiated by the motor results of vestibular end-organ stimulation. These secondary sense stimuli are the muscle-and-joint-sense stimuli incidental to extra-ocular and skeletal muscle reactions to vestibular stimulation. Primary vestibular sensations of motion are thus normally amplified by secondary (additional) deep sensibility sensations of motion when these vestibular end-organs are stimulated. That incidental motor expressions of stimulation, or that concomitant additional motion-sense impulses result from the emission of afferent impulses from the end-organs, in no way seems to alter the fact that stimulation of these end-organs causes them to send forth afferent impulses of a purely special-sense character. Equally direct motor expressions of afferent impulses are seen in the operation of the primitive reflex arc, concerning which no question is raised with reference to the sensory character of the initial impulse emitted by the end-organ. With the evolution of more complex nerve connections required by the higher elaborations of the organism, differences in threshold values come to play important parts in distribution of afferent impulses. Afferent-efferent nerve circuits undergo adaptation changes of infinite variety, changes determined by special requirements of component systems going to make up the organism. What was a simple reflex arc in a lower order of organism develops into an integral part of a definitely specialized component system in a higher order of organism. In this manner certain primitive characteristics of reaction may be lost, other acquired secondary characteristics of reaction may become prominent. From the psychological point of view it seems sound, as it is from the physiological point of view demonstrably safe, to say that this vestibular apparatus is an organ of special sense.

habitants are supplied with food, drink, heat, light, power, and information. In order to effect these transmutations of energy, for instance from coal to power, the intermediations of certain physical mechanisms are necessary. In the physiological community of the living body the same intermediation of (living) physical mechanisms is necessary to the transmutation of any form of energy, such as food into strength, light into vision, sound into hearing. Every sense of the body is dependent upon the operation of some living *physical* mechanism.

While motion is the "adequate stimulus" of this ear apparatus, it must be added that other forces than simple motion do enter into its stimulus under normal conditions. Centrifugal force becomes a component of its stimulus during motion in other than a right line; the force of gravity acting upon the cupula and otolith structures also constitutes a component factor in its stimulus. These two forces always operate to modify the force of a simple motion applied to the ear apparatus; it is therefore necessary to qualify a direct statement that motion constitutes the sole force which operates as the stimulus. Strictly speaking the force operating as the stimulus is the resultant of these three forces. *For the sake of brevity in the ensuing pages of this chapter, this qualification will be understood as included in the terms "motion" or "motion-sensing."* Before proceeding to consider motion-sensing it is necessary to discuss some details concerning varieties of motion.

The term "motion" is used to indicate a change of spatial relations with respect to environment.\*

\* Wells of Washington, D. C., has called attention in a classic way to the natural evolutionary processes relating to the ear. Briefly, he points out that lower forms of life, such as corals, came into being, lived their allotted span, and died under normal conditions of immobility. Their requirements for living included very little beside the ability to select from their environment what was necessary materially. They were endowed by Nature with such sensing as was required for their primitive needs—a sort of selective appropriation of materials necessary for their metabolic processes. As evolution proceeded, higher forms of polycellular groups came into being; a group or community of cells separated from the parent bed of lower evolutionary level; this group found itself tossed about on the ocean bed by the movement of the water; it encountered its own (community) problems of survival, under its own natural environmental conditions, which differed from those of the parent bed *only in regard to mobility*. Motion thus first came to be included among these natural environmental conditions. Nature responded to a new natural need by beginning to develop in this higher form of organized cellular life a higher form of natural endowment; motion-sensing was the new need and the rudimentary otic cup developed as the higher form of natural endowment. With increasingly higher evolution the development of the motion-sensing equipment kept pace, so that when involuntary motion came to be supplemented by voluntary motion in higher species, the mechanism necessary for sensory guidance of the organism was developed to the degree adequate to its needs. Eventually amphibian forms evolved and, with the environmental change from water to air, developed new mechanisms adapted to the new needs. Among other changes the organism found itself confronted with the necessity for sensing a newly encountered form of motion—air-borne vibrating waves—which we call "sound," heralding among other things the approach of natural enemies. To meet this new need an accessory mechanism developed, adapted to the conversion of this new form of motion into sense information. An excrescence appeared upon the otic vesicle, housing the beginnings of the cochlea and tympanum. With the final evolution of animal forms living entirely in air came the final evolution of the two ear mechanisms complete; and the "vestibular (motion-sense) mechanism" and the "cochlear (sound-sense) mechanism" reached their highest development. The semicircular canals progressed through successive elaborations, from a simple pit containing a few hair-cells surmounted by casual water and sand from the ocean bed to a closed receptacle with the hairs ensconced in the ampulla of first one, later two, and finally three semicircular canals, bathed in a specially secreted fluid, and surmounted by a gelatinous cap structure, the cupula. The cochlea progressed similarly from a dome-like simple excrescence, through gradual elaborative stages showing spiral tendencies, to a final two and one-half turn snail-shell structure with the hair-cells distributed along the basilar mem-

For example, if a balanced object is standing in a non-moving railroad train it is said to be "still"; when the train begins to move, the object begins to be subjected to a series of accelerations changing the environmental relations of itself and the railroad car in which it is standing. The accelerations may be very sudden changes, in which case they cause the object to fall backward on the floor of the car; or so gradual as to cause it to sway backward only slightly. In either case the motion applied to the car during acceleration is transmitted from the car to the object and this motion represents momentarily the spatial change occurring with reference to environment of both car and object.

When the railroad car has attained its maximum speed—say forty miles an hour—on a perfect track with no directional changes, this speed may be sustained indefinitely with no more effect upon the balanced object than was apparent before the car started moving. On striking a curve, however, its balance is disturbed, causing it to fall over toward the side. If the train suddenly slows down the object falls forward; if slowing is very gradual it may only cause the object to sway forward without falling.

Immediate and remote environmental relations are not necessarily maintained similarly under all conditions of motion, as witnessed by the maintenance of unchanging relations between the object and the car during steady progression as contrasted with the changing relations between the object and the car during acceleration, progression on curves, and deceleration.

Thus it is apparent to the observer that a variety of effects with respect to immediate environment are produced as varying motions are brought to bear upon objects: acceleration, or incremental motion, produces a certain physical effect; sustained speed in a straight direction produces another physical effect, and sustained speed with changing direction produces another physical effect, while deceleration, or decremental motion, produces still another effect.

The ear motion-sensing apparatus takes particular cognizance of incremental or decremental motions, and of directional changes, which in effect fall into the same category. This must be borne in mind constantly in dealing with the problem of the vestibular functions. The ear motion-sensing apparatus does not take cognizance of motion of unvarying direction or speed.

In addition to motion changes, posture plays an important rôle in connection with motion influences. Under certain motion conditions an inclined posture of an object serves to maintain desired spatial relations with its environment, as in the "banking" posture assumed in order to maintain stance during progression on curves, or a forward inclination of an object during acceleration in a straight direction.

Under conditions of motion of sustained speed and unchanging direction the upright posture serves to maintain desired spatial relations, while during deceleration a backward inclination is necessary.

All such postural changes are requisites of the living body under similar conditions of motion, and these postural changes tend to occur auto-

brane floor of the cochlear duct, bathed in a specially secreted fluid and surmounted by a gelatinous tectorial membrane. One cannot but be impressed with the obvious significance of these evolutionary morphological changes in considering the problems of physics and physiology confronting the student of internal ear functions.

matically as the effect of reflexes originating in the vestibular apparatus. Equilibrium is the result of these body adjustments, which are conceived to represent inherited instinctive co-ordinations of the nature of "pattern reactions" to motion stimuli, acquired through the experience of countless generations.

That part of the ear which is normally stimulated by motion was the first to develop.\* This is called, for lack of a better name, the "vestibular portion" of the ear; it is the "motion-sensing portion." It consists of two main elements, the physical mechanism and the physiological apparatus; the latter is composed of end-organ cells and nervous system connections.

This physical mechanism acts in such a manner that, when motion† is brought to bear upon the ear, certain physical changes occur, as a result of which the hairs of the end-organ cells of the semicircular canals are directly affected. So accurately does the physiological apparatus co-operate with the physical mechanism that the resulting nerve impulses emitted from the end-organ cells annunciate in the sensorium the direction, plane, and intensity of these motions.

What is thus annunciated in the sensorium is normally consistent with the information concerning motion reaching the sensorium simultaneously

\* Motion is entirely relative; all things exist under conditions of constant motion. Mundane creatures which have been bred, born, and raised under the multifold motion conditions of movement through space along the earth's orbit and of the daily revolutions of the earth upon its axis, take no cognizance of these tremendous movements. If either of these were to vary, decrement or increment would be sensed by living creatures as disturbance of their "customary perfect quiet." Balanced objects would be thrown down and in general the effect of the incremental or decremental change in previously constant movement would become manifest.

In this connection allusion should be made to the ingenious experiments of Griffith at the University of Illinois. He has some generations of white rats and mice which were bred, born, and raised on tables which have been rotating constantly at from 60 to 80 revolutions per minute for several years. These animals eat, sleep, propagate and die under these rotational conditions just as others do under conditions of "normal quiet." When their rotating tables are stopped they manifest uncontrollable rolling, nystagmus, and evidences of vertiginous disturbances similar to the effects manifested by normal animals when they are subjected to rotation.

† It is a physical impossibility to apply motion of any constant speed to a quiescent mass. For instance, a mass can be set into motion at ten miles per hour, but only by constantly changing its rate of speed from zero at the beginning up to ten miles an hour. Whether the mass attain this desired velocity suddenly, as does a projectile fired from a gun, or slowly, by imperceptibly increasing gradations, the fact remains that its rate of motion is varying between its start and the attainment of the desired speed. This inexorable physical fact makes it impossible to apply motion of any definite rate as a stimulus to test the ear motion-sensing apparatus. For reasons of practicability the standard tests of the ear-motion sense have been developed according to a technic which uses rotation instead of motion in a right line, and which observes the effects of decremental motion instead of incremental motion. Whereas it would be thoroughly impractical to attempt to observe the reactions of a subject stimulated by the sudden application of rotation, at the rate of ten turns in ten seconds, for example, it is quite easy to study in detail the subject's reactions on suddenly ceasing to rotate at the rate of ten turns in ten seconds. The physical change produced within the vestibule by bringing to a sudden stop a body moving at a certain speed is for all practical purposes the same as the physical change produced by setting that body into motion in the opposite direction at the same rate of speed. Many experimental observations have been made by the writer, using the motion in a right line. (*Studies of the Ear as a Motion-sensing Organ*, Ann. Otol., March, 1919.) The apparatus required is such as to make this kind of testing prohibitive. Rotational motion, on the other hand, requires only simple apparatus, and is entirely practical for testing. Having set into motion by turning in a certain direction and speed the fluid within the internal ears the body is brought to a sudden stop and one observes the effects of the motion of the fluid which continues of its own inertia for a certain time. Providing the direction and speed are standardized, motion so applied constitutes a constant stimulus and its physiological effects can be observed more or less accurately.

from other sources of motion-sensing, namely, "motions seen" (vision) and "motions felt" (deep sensibility and tactile sense).\*

**The Physical Mechanism.**—The essentials of the physical mechanism of the vestibular apparatus include two oppositely placed halves, one in the right, one in the left temporal bone, each containing:

- I. A common cistern (utricle) communicating with both ends of
- II. Three semicircular canals, each lying in a plane at right angles to the planes of the other two canals, representing the cardinal planes of space; each canal having at its contralateral junction with the cistern
- III. A bulbous dilatation (ampulla) containing hair-cells surmounting a crista; the cistern and canals filled with
- IV. Fluid (the endo- and peri-lymph).

In order to facilitate the study of its physical operation, the rigid intervening bone between the two halves of this mechanism may be ignored and the two halves juxtaposed as shown in Fig. 364. This shows in reconstruction one common utricle and three curved canals, each at right angles to the other two, and each with an ampulla at each end as it opens into the utricle.

When such a mechanism is subjected to motion, it is apparent that some of the contained fluid will change its relations with respect to the container; and it is further apparent that these changes of relations will be governed by the laws of inertia.

\* If ear function is totally lacking, motion can still be sensed by vision, by deep sensibility, by tactile sense. Vestibularly dead deaf-mutes are able to sense motion fairly accurately in the light; but in the dark these deaf-mutes are surprisingly incapacitated by having to depend totally upon their one remaining motion-sensing, namely, deep sensibility (pressure sense, joint sense, muscle sense, visceral sense, those senses which are normally stimulated during body quiescence by the pull of gravity upon the individual portions of the body mass—the head, the extremities, the abdomen and its contents, and the chest and its contents). When, in addition to the pull of gravity, the various parts of the body-mass are individually and collectively subjected to the pull of motion—whether in the same direction as that of the pull of gravity or in any other direction—the physical effect upon them is obviously the resultant of two forces—the force of gravity and the force of the motion. Under all the conditions set forth in the foregoing, a "pull" is definitely sensed, but vaguely rather than with normal accuracy. Such an individual awaking in the dark during a smooth airship journey at a constant rate of speed of 100 miles per hour, would experience almost no sensation of movement and should the rate of speed (northward) quickly decrease to 80 miles per hour, he would feel *during the decrease* exactly as he would feel if he had as quickly begun to move at a rate of 20 miles per hour in the reverse direction (southward). In this manner normal sensing becomes responsible for sensory illusion—misinformation of the sensorium or misinterpretation by the sensorium of normal sense impulses. Sensory illusions of this sort are not confined to deep sensibility; under certain circumstances similar illusions respecting motion may be caused by a normally functioning ear motion-sensing apparatus, or by normally functioning vision. During a railroad journey it is a frequent experience to mistake the motion of another train for motion of our own train in the opposite direction when the other train alongside ours begins to pull out slowly.

It is not improbable that illusions are experienced more frequently concerning motion than any other form of energy the body is called upon to sense ("illusions" being understood as "normal sensations misinterpreted"). It seems true that accurate sensing of motion requires the simultaneous arrival *in the sensorium* of more than one quantum of sense information concerning this motion. By this statement is meant that, of the three sense (end-organ) sources capable of sending into the sensorium information (afferent impulses) concerning motion, that from at least two sources must be consistent (mutually confirmatory) if illusion is to be avoided. Under some conditions sense information coming in to the sensorium from all three functioning motion sense end-organs does not suffice to prevent illusions (see example—railroad experience of a normal person alluded to in previous paragraph); under other conditions that coming in from only two of the three may suffice for sensorial impressions free of illusion (see ordinary daylight experiences of vestibularly dead deaf-mutes). Ordinarily, *but not always*, if any two of the three motion senses agree in their sensing as to the direction, intensity, and plane of a motion, the sensorial impression concerning that motion is in accord with fact; but the sensing of a motion by only one of the three is not to be relied upon as accurate.

Occasionally during the past two decades it has been alleged by commentators on the physics of the vestibular mechanism that fluid within the canals cannot move in response to inertia or convection influence because of "capillary action"\* in a tube of such small caliber. Reasoning from this premise a German commentator proposed that the caloric test depended not upon fluid movement, but upon "exaltation of function" by heat and "depression of function" by cold. It was also proposed that, instead of actual mass movement of the fluid in the canals, "molecular movement" of the fluid probably accounted for the observed results of rotation or caloric influence. It has also been proposed that "pressure changes" within the canals as the result of rotation or cooling may account for the phenomena observed. It does not require "molecular movement" or "pressure changes" theories to account for mass movement of fluid within blood and lymph capillaries, the caliber of which is far smaller than that of the semicircular canals.

Dr. S. D. Ingham in 1921 † constructed capillary tubes the size and shape

\* "Capillary action" is a term applied to a phenomenon observed under certain physical conditions; a fluid is observed to run upward in a capillary tube, or conversely, it does not run downward as fluid ordinarily does under the influence of gravity, because "capillary attraction" outweighs the influence of gravity. The fact is that the capillary tube itself does not necessarily exert any attraction upon fluid whatsoever. Surface tension determines the behavior of the fluid; if conditions are such that a minus (negative) meniscus forms, the fluid runs up the capillary tube, if a plus (positive) meniscus forms, the fluid does not enter the capillary tube. Meniscus formation depends upon *cohesion* and *adhesion*. Cohesion is the force by virtue of which like particles of matter attract each other; adhesion is the force by virtue of which unlike particles of matter attract each other. Water forms drops because its particles *cohere* to each other; a drop of water remains on the window-pane because it *adheres* to the pane, which it does not do to any oily surface. A drop of water spreads out thin upon a dry glass surface, because the *adhesion* between the glass and the water particles is relatively great as compared to the *cohesion* holding these water particles to each other; upon a surface of oil a drop of water assumes a globular shape because the adhesion between the oil and the water particle is relatively small as compared with the cohesion holding these water particles together.

Thus the particles of water are subjected to *two attractions*—that of adhesion to the surface they are in contact with, and that of *cohesion* to each other; naturally only the particles at the surface of the water-drop can feel the pull of both these attractions. This *tension* caused by the opposing pulls is called "surface tension." If a  $\frac{1}{2}$ -inch glass tube be plunged into water, a negative meniscus forms because of the relatively great *adhesion* between glass and water as compared with the *cohesion* of water; if the same tube be plunged into mercury a positive meniscus forms because of the relatively small *adhesion* between glass and mercury as compared with the *cohesion* of mercury. *Adhesion* between glass and water tends to draw the water and the glass together; if the tube be held horizontally *adhesion* results in the water filling the tube; if the tube be held vertical, *adhesion* continues to draw the water into contact with the glass until further contact is prevented by some other influence; in this instance the mass of the water is sufficient for the pull of gravity upon it to limit the further contact of water and glass. If, however, a glass tube of very small caliber be held vertically in water, the pull of gravity upon the very small mass of water included within the lumen of the glass tube is not sufficient to counteract the force of adhesion between the tube-wall and the water particles. Glass and water particles attract each other with relatively greater force than that of the pull of gravity upon these water particles. In this combination of forces adhesion is greater than the pull of gravity upon the very small mass of water within the tube-mouth. This allows the water to creep up the tube-walls, by adhesion to the glass; the force of *cohesion* suffices to hold sufficient water particles to those adhering to the glass walls, which completes the filling of the tube lumen by water. This is the phenomenon of "capillary action"; the predominance of *adhesion* and *cohesion* over gravity under such circumstances is spoken of as "capillary attraction." This is in no way a specific force, but is the resultant of two variable forces; once surface tension is eliminated, equilibrium is established between adhesive and cohesive forces, and the fluid mass finds itself under physical conditions which permit it to respond to the influence of inertia or gravity, subject only to modification by friction. The actual mass movement of a fluid confined within such a canal shows greatest retardation at the periphery and greatest fluid movement at the center.

† In 1922 the Bureau of Standards at Washington, D. C., constructed a canal the size and shape of the membranous semicircular canal by countersinking the canal in a glass

of the membranous semicircular canals, filled these with fluid containing microscopically visible particles—blood-cells, “mother of vinegar” cellules, and fine particles of India ink—plugged the ends of the tubes, and under the microscope demonstrated movement of the fluid in convection currents under caloric influence. There were demonstrated not only longitudinal currents but *cross currents* in the lumen of this small container. Dr. Ingham’s original notes are as follows:

“Capillary glass tube with lumen of 3 mm. and of the approximate size and shape of the human semicircular canal was sealed to a glass slide. Each end of the tube communicated with a small reservoir and the entire apparatus, tube and reservoir, was filled with fluid. With the tube curvature in the vertical plane the application of heat to either vertical segment caused convection currents, visible microscopically, manifesting the phenomena of the thermosyphon. While making one observation the flow

slide. This could then be overfilled with fluid and the canal completed by pressure-sliding a cover glass over the fluid-filled countersinking. Using this preparation the author has repeatedly demonstrated fluid movement under the microscope—mass movements in accord with inertia and with caloric influence—convection currents. Thus without recourse to teleology it can be stated definitely that the physical mechanism of the semicircular canals apparatus operates according to the influences of mass movement of its contained fluids. In this connection it is noteworthy that more than a decade prior to this microscopical demonstration of mass fluid movements within a canal the size of the membranous semicircular canals the hydrostatics of lymph currents within the canals had been worked out by means of physiological methods of determination.

In answer to the proposed explanation of caloric reactions on the theory that they were due to functional exaltation by heat, or conversely, functional depression by cold, it had been pointed out by students of the vestibular apparatus that if this were the case, and cold douching of the right ear with the head in the upright position elicits rotary nystagmus to the left by depressing the functional activity of the right semicircular canals apparatus, this evidence of depressed functional activity must of necessity continue to be manifest regardless of posture variations. It was shown that inversion of the head-upright position during the continuation of this cold douche changed the direction of the nystagmus from the *left* to the *right* which is the same nystagmus as is caused by hot douching the same ear, or by cold douching the opposite ear, in the original upright position. It had also been shown that similar reversal of the character of the nystagmus resulted when the position of the head was altered after cold douching of one ear; with the horizontal canal in the vertical position, the nystagmus was horizontal, with the vertical canals in the vertical position, the nystagmus was rotary. This changing of the nystagmus with the change in position of the head indicated clearly that the causative physical factor must be influenced by change in position. It was also shown at this time reversal of both the direction of, and the character of nystagmus elicited by rotation could be accomplished at will by altering the position of the head in such a manner as to bring the plane of the desired nystagmus and the plane of the canal at right angles to the axis of rotation.

With the known facts, including:

1. Fluid-filled canals disposed in the three cardinal planes of space.
2. Rotation causing nystagmus always in the plane at right angles to the axis of rotation.
3. Rotation causing nystagmus whose slow component was always in the direction of rotation.
4. Cold douching always producing nystagmus in the vertical plane.
5. Cold douching always producing nystagmus whose slow component was toward the ear douched.
6. After a single cold douche the character of the nystagmus always following an altering position of the head in conformity with the canal happening to be vertical at the time.
7. Warm douching always producing nystagmus exactly opposite in direction from that produced by cold, otherwise behaving exactly the same.
8. The nystagmus produced by heat always promptly stopped by application of cold, and its reverse always produced by further continuation of cold and vice versa, the nystagmus produced by cold always promptly stopped by heat, and its reverse always produced by further continuation of heat.

The conclusion seems unavoidable that the eight points specifically set forth establish beyond question the physical fact of mass movement of the fluid in the canals in accord with inertia and caloric influence.

in the tube was observed to stop and then two currents—one up and one down—were observed at the same time passing each other within the lumen of the tube. Investigation showed that these phenomena were caused by obstruction of one end of the glass tube.”

If the physical mechanism of the vestibule be rotated clock-wise in the plane of the horizontal canals, the containing horizontal canals will begin to move in the direction of rotation while inertia will cause the contained fluid within the horizontal canals to lag. As the motion continues, friction eventually transfers the force of the motion from the container to the fluid within the horizontal canals, overcoming inertia, and the containing horizontal canals and the contained fluid within them come to be moving together clock-wise. If now the mechanism be stopped rotating, inertia causes the fluid to continue to flow clock-wise through the canals until friction gradually brings it to rest. Upon starting this motion the hairs of the right ampulla are inclined away from the canal (toward the utricle), while the hairs of the left ampulla are inclined toward the canal (away from the utricle). As the inertia of the fluid is overcome during the rotation, the hairs of both ampullæ assume the upright position again. After stopping this motion, the inertia of the fluid inclines the hairs of the right ampulla toward the canal, those of the left ampulla toward the utricle. As the fluid gradually comes to rest, the hairs gradually resume their upright position. During this rotation no fluid current is set up in the vertical canals for obvious physical reasons. If this mechanism is rotated about an axis vertical to either of the other canals, similar fluid changes are brought about in these canals; if the rotation be about an axis midway between these vertical axes, fluid changes occur in both canals.

It is thus apparent that this physical mechanism is so devised that its contained fluid in some or all canals comes to participate in motion in any direction, and the effect of this participation is brought to bear upon the hair contained in the ampullæ. These hairs are thus subjected to selective disturbance by a specific form of energy-motion, and this selectivity has elements of both direction and intensity. The surmounting cupula enhances the delicacy with which any motion may be brought to bear as a physical disturbance of quiescence of the hairs.

**The Physiological Apparatus.**—The sensory cells of the end-organs are situated in the ampullæ—so placed upon the crista of each ampulla as to take the greatest advantage of the disposition of the hairs just referred to. They constitute the base cells upon which these hairs stand, and the hairs are integral parts of these end-organ sense-cells. This hair-cell emplacement subserves the highest conceivable utility, with respect to the operating of the intermediary physical mechanism.

The end-organ cells each send neuronal prolongations along the bony spiral lamina, which meet to form the peripheral portion of the vestibular division of the eighth nerve. It is the common pathway along which all impulses arising in the end-organs of the vestibule pass on their way to their various distributions. Its proximal contact with the central nervous system takes place at the upper lateral aspect of the medulla oblongata.

**Reaction Details.**—Stimulation of these end-organs results in certain specific sensations of movement and in manifestations of other than sensory nature.

**Vertigo.**—These sensations of movement are termed “vertigo” (*vertigo* meaning “subjective sensation of turning movement”). This, naturally, entails the subject’s assuming fixation of his environment, because without contrast of fixed objects it would be difficult to sense subjective movement. Reversal of this assumption (which is purely arbitrary on the part of the subject) may result in a “sensation of movement of fixed objects”—with respect to the subject.

*Vertigo* means “subjective sensation of rotation” with respect to fixed objects or vice versa—“sensation of rotation of fixed objects”—with respect to the subject. The essence of vertigo is rotational sensation of disturbance of relations with one’s environment. This sensation may take the form of turning movement of the individual to the right with respect to a *vertical* fixed object—such as a post—or of this fixed object to the left with respect to the individual.

It may take the form of a turning movement of the individual’s body—head rotating forward and downward, feet rotating backward and upward—or of the (horizontal) floor in front of the individual rotating out of the horizontal upward toward his head, the floor back of him rotating out of the horizontal downward away from his head, or vice versa. It may take the form of an oblique rotational disturbance somewhere between the vertical and horizontal. But regardless of detail, vertigo involves a subjective sensation of disturbed relations with environment in some definite plane—vertical, horizontal or oblique.

The term “vertigo” is not infrequently used to allude to unusual or abnormal sensations which are not at all this sort of disturbance of the sense of normal spatial relations.\*

\* Hubby says: “Vertigo is a complex made up of at least three elements: (1) The vestibular nerve reaction movement reflex; (2) the sensation of turning, and (3) the resulting opinion of the sensation followed by psychic and somatic reactions.

“1. *The Vestibular Nerve Reaction Movement Reflex.*—The vestibular nerve reaction movement reflex passes from the labyrinth to the vestibular nerve centers (Deiters’ nucleus, ventrocaudal nucleus of Deiters, nucleus triangularis, Bechterew’s nucleus, nucleus embolobosis), then to the dentate nucleus, the cerebellar cortex, back to the dentate nucleus, then to the red nucleus and last, by the rubrospinal tract to the motor cells in the anterior horn of the cord and to the muscles. This reflex contains at least seven neurons. Involvement of any one of them may produce vertigo.

“Other neurons also enter into the reaction reflex; the corticospinal from the motor cerebral cortex to the motor cells in the anterior horns of the cord and thence to the muscles; the deitersospinal from the vestibular nuclei to the anterior horn cells and muscles; the corticopontile tract to the cerebellar cortex; the tracts from the vestibular nuclei to the cortex of the worm; then to the nuclei tecti (fastigii) and back to the vestibular nuclei and undoubtedly many others not yet traced.

“2. *The Sensation of Turning.*—The sensation of turning is brought about by the passage of these vestibular impulses of changed relative intensity from the two sides, to the centers situated in the parietal cortex, subcortex, or the optic thalamus. Physiologists are still in doubt as to the exact locality.

“There may be a slight feeling of turning in the cerebellum and the brain-stem, but it is, in the main, probably subconscious and does not become well defined until reaching the higher centers. On the other hand, the sensation may be a resultant from all these sources. At any rate it is beyond the arcs of vestibular somatic muscular reaction.

“The muscular reaction movements of the eyes, limbs, and body are also followed by sensory impulses to the sensorium, which contribute to form the vertigo complex.

“It would seem probable that the nearer the labyrinth the alteration of the afferent impulses from the labyrinth occurred, the clearer the sensation would approach that of real turning and the greater the vertigo.

“3. *The Resultant Opinion of the Sensation Followed by Psychic and Somatic Reactions.*—Every projection center is connected with every other one by associatory neurons and is also thus connected with the associatory areas, in which the different concepts are supposed to arise together with the motivating feeling to act.

“The sensation of turning, clearly felt in the parietal cortex, is further analyzed in the

Patients not infrequently use the term "vertigo" to describe feelings of uncertainty, of vague confusion, indefinite head noises, or flashes of light or heat, faintness, and the like; these should not be mistaken for vertigo. "Giddiness" or "dizziness" may better be used to describe these.

When a semicircular canal end-organ is stimulated there normally result certain movements of the nature of adjustment; inasmuch as end-organ stimulation of the typical simple reflex arc causes movement of its effector, at least so far these movements may be considered of the general nature of reflexes. These movements involve the eyeballs, the extremities, and the entire body.\*

**Drift reactions** during stimulation of the end-organs of the semicircular canals are co-ordinative motor reflexes. The motor impulses reflected along the distribution of the vestibulospinal connections effect muscle tonus alterations, causing "drifting" of the trunk and of the extremities, including the head, in the direction of the flow of labyrinthine fluid—to the right or to the left, forward or backward. The following are examples: During rotation of the horizontal canals to the right, if the arms are held in position of extension forward or to the sides, they "drift" involuntarily to the left in the horizontal plane; the upright head also "drifts" toward the left, rotating about the vertical axis of the neck; after stopping this rotation, these "drifts" are in the reverse direction. After rotating the vertical canals to the right with the head 120 degrees forward,

association areas; it is compared in quality and intensity with past experiences together with their sequelæ, and a real concept is developed. This results in anxiety or fear and produces all the concomitant somatic reactions depending on the temperament and physical constitution of the subject.

"This may bring about changes in the circulation of the blood, feelings of flushing of the face, faintness, syncope, etc.; respiratory changes; gastro-intestinal reactions such as nausea, vomiting, and diarrhea; changes in the kidneys, such as increased micturition and production of urine.

"Otic vertiges and those caused by diseases of the vestibular nerve tracts up to the parietal cerebral cortex involve all three of the foregoing components of the complex called vertigo; hence the result, marked sensation of turning, etc.

"When the two other components outside of the ear and the vestibular tracts only are affected, such as the multitude of associatory fibers, not only in the cerebral cortex but also in the rest of the brain, then the sensation of vertigo is much more confused and may amount merely to a feeling of uncertainty. This is true of ocular and psychic vertiges.

"Of course vertigo may be of a turning type, when the nerve currents are sufficiently strong to spill over from neighboring neurons, such as the oculomotor to the vestibular neurons, but I believe this is rare."

\* Thus it comes to pass that certain group reactions, "pattern co-ordinations," reflex and sensory reactions, are seen to be elicited as the direct result of impulses originating in the vestibular end-organs. These are properly termed "vestibular reactions," yet careful qualification of the use of this term is necessary if erroneous impressions are to be avoided. The "sprawl-pattern" is touched in upon by the vestibular impulses and a sprawl results; the "conjugate movement of the eyes to the right in the horizontal plane-pattern" is touched in upon by the vestibular impulses, and a perfectly co-ordinated direction of the gaze to the right results; the "emesis pattern" is touched in upon by the vestibular impulses and pallor, sweat, nausea, vomiting, and faintness result. Even the purely sensory result of the sensation of movement caused by vestibular stimulation has its counterpart (if not identical, yet similar) as the result of the touching in upon this sensory impression by impulses from other sense end-organs, those of deep sensibility, of tactile sense, and of visual sense. Yet the fact remains that *solely as a result of vestibular stimulation* these various sensory and "pattern" reactions result; and so resulting, *they are the vestibular reactions*.

The ophthalmologist, the neurologist, the physiologist may object to calling any or all of these reactions "vestibular reactions" on the grounds that they are elicited by other mechanisms—even in the total absence of functioning vestibular end-organs. The fact remains, however, that when these group reactions are elicited solely as the result of vestibular stimulation they are just as definitely "vestibular reactions" as are the pupillary reflexes "visual reactions."

on assuming the upright position the trunk drifts to the right in the vertical plane, rotating about the anteroposterior horizontal axis.

After rotating the vertical canals to the right with the head tilted 90 degrees over the right shoulder, on assuming the upright position the trunk drifts backward in the vertical plane, rotating about the transverse horizontal axis.

With respect to the planes of the head, "drifting" reactions may be produced at will in any plane or direction, by rotating the head in such a position as to produce flow of labyrinthine fluid in the selected plane and direction of drift.

**Past-pointing** during stimulation of the end-organs of the semicircular canals is not a simple reflex, in that it combines with the "drifting" tendency the effect of voluntary motor mandate influenced by the conscious sensation of vertigo. For example, after rotating the horizontal canals to the right, the vestibulospinal reflex produces a drift tendency of the extended arm to the right; the vertigo produces a sensation of rotating about the vertical axis to the left. If a voluntary effort is made at this juncture to point to a certain predetermined fixed object such as the examiner's finger, the sensation of rotating away from this fixed object to the left would (consciously and subconsciously) direct the voluntary pointing toward the right. As has already been mentioned, the "drift tendency" is also toward the right. The resulting past-pointing is, obviously, to the right as the result of both of these influences. With respect to the planes of the head, "past-pointing" may be produced at will in any plane or direction by rotating the head in such a position as to produce flow of labyrinthine fluid in the selected plane and direction.

**Falling** or fall-tendency is similarly a mixture of reflex and voluntary effort, the latter in accord with subjective conscious or subconscious influence of the coincidental sensation of vertigo. After rotation to the right of the vertical canals with the head tilted forward 120 degrees, on assuming the upright position the "drift tendency" produces involuntary drawing of the head and trunk to the right about the anteroposterior horizontal axis; the conscious or subconscious adjustment to the subjective vertigo sensation of rotating about this same anteroposterior horizontal axis to the left produces rotation of the head and trunk to the right; in this manner both tendencies find expression in a fall-tendency to the right. With respect to the planes of the head, falling may be produced in any plane or direction by rotating the head in such a position as to produce flow of the labyrinthine fluid in the selected plane and direction.

**Other Motor Reactions.**—In standing upright, or walking, or pointing, when the normal co-ordination "pattern" is touched in upon by vestibular impulses resulting from artificial or pathological stimulation, the resulting modification of standing upright or walking or pointing is evidenced by falling, drifting, or past-pointing to the side. These *modifications* of normal "pattern" co-ordinations (*not the acts themselves*, such as walking or pointing) are vestibular reactions. With respect to varying intensity, the effect of vestibular stimulation may become so great as to convert the maintenance of a normal sitting posture into an active sprawl. The vestibular reaction thereupon assumes the character of so-called "forced movements." The phenomena which Magnus and de Kleijn have emphasized and studied particularly are of this nature. Forced movements of head and neck,

forced body movements, and forced movements of extremities are included in Magnus-de Kleijn phenomena.

In studying the effects resulting from stimulating these end-organs still another variety of expression is seen—the group of so-called “sympathetic responses”—pallor, sweat, nausea, vomiting, and faintness. These must be classed as “physiological vestibular reactions,” despite the fact that they are elicited only by a relative “overdosage” of stimulus. From the standpoint of the effects of stimulation, however, they must be included among the manifestations to be noted as normal reactions. Pallor, sweat, nausea, vomiting, and faintness are the sympathetic system manifestations of vestibular stimulation; these constitute expressions of ear stimulation in a wide range of ultimate distributions, including cardiovascular, glandular, visceral, sensory, vasomotor, and general systemic.

**Maculæ Sacculi et Utriculi.**—In addition to the end-organs of the semicircular canals, the vestibular apparatus includes end-organ hair-cells distributed to the macula sacculi and to the macula utriculi. The hairs of these cells are surmounted by semisolid structures suspended in the endolymph—the otolith membranes. The macular organs are so disposed and constructed as to be physically affected by motion applied to the head; their hair-cells send forth neuraxons which join with those from the hair-cells of the semicircular canals to make up the vestibular portion of the eighth nerve. Physically and anatomically there are definite reasons for regarding these organs as integral portions of the vestibular apparatus. The physiology of the maculæ is gradually being built up from the studies which are being made in many laboratories. Theoretically it has been proposed that they are especially concerned with movements in the horizontal plane; and that they are especially concerned with maintenance of posture; they have been referred to, hypothetically, as the “static” portion of the labyrinth in contradistinction to the “kinetic” (semicircular canals) portion. At present, however, little is known definitely concerning the function of the maculæ sacculi and utriculi.

On undertaking to study either normal or abnormal vestibular reactions the following statement of relations must be clearly understood because, for the sake of brevity, qualifying explanations with respect to these relations must be omitted from all subsequent discussion of the details of vestibular reactions in this chapter.

After reaching the medulla oblongata the fibers from the vestibular end-organs find themselves in contact with large numbers of nerve connections; these contacts are established successively, beginning with connections at Deiters', von Bechterew's, and triangular nuclei, and involve mechanisms of various kinds, such as skeletal, neuromuscular, sympathetic, vagotonic, viscerosplanchnic, oculomotor, and deep sensibility mechanisms. Just in so far as portions of these nerve mechanisms are actuated or influenced by nerve currents originating in the vestibular end-organs do the effectors of these various mechanisms express vestibular impulses. (See footnote on p. 679.) And the sum total of all these expressions—sensory, motor, reflex, visceral, vasomotor, ocular, etc.—constitutes the *vestibular reactions*.

This paragraph is so fundamentally important in conjunction with the study of the vestibular apparatus that it should be read with especial attention.

It is this mechanism which must be put to functional proof when one undertakes to "test the vestibular apparatus." The foregoing allusion to its complexity may serve to point out the general nature of understanding which must form the basis of any serious attempt to interpret results of tests.

The eye movements resulting from stimulation of the vestibular end-organs are termed "nystagmus." Prior to referring more particularly to the physiology involved in a consideration of the exact nature of nystagmus, it is desirable to attempt to fix in mind certain fundamental relations between the physics of the vestibular mechanism and the physiology of the vestibular apparatus involved in a clear conception of why and how it comes about that so many variations in nystagmus may be encountered. In order to take up this phase of the interrelation between the physics and the physiology of the *physical mechanism itself* and *physiological apparatus* connected with it, it is necessary to depart from the solid ground of scientific accuracy and make an excursion into the realm of speculation. With the full understanding that some of the following considerations *cannot be proved*, but are nevertheless consistent with certain known facts which *can be proved*, the following relations between physical changes and physiological effects should be considered.

#### WORKING BALANCE BETWEEN RIGHT AND LEFT HALVES OF THE VESTIBULAR MECHANISM

**Physical and Functional.**—The vestibular end-organs lie in the two petrous bones, so disposed that the end-organs contained within each canal are paired, physically and functionally, with those of the opposite side (see Fig. 364). The right horizontal canal end-organs are in the plane of, and oppositely placed from the left horizontal canal end-organs. By virtue of this relative disposition, rotation in the plane of these canals produces equal and opposite physical effects upon each half of this pair of end-organs; after rotation to the right, for example, the rate of the labyrinthine fluid flowing away from the utricle on the right side is exactly equal to, and opposite, the rate of the labyrinthine fluid flowing toward the utricle on the left side. Thus there is a balance anatomically established, physically maintained, and physiologically expressed in the co-operative action of these end-organs. Movement of fluid toward the left utricle causes reflex slow pull of the eyes *toward the right*; movement of fluid away from the right utricle causes reflex slow pull of the eyes *away from the left*; similarly fluid movement *away from the right* utricle produces a sensation of vertigo *toward the left*, fluid movement *toward the left* utricle produces a sensation of vertigo *away from the right*. The same co-operative effects obtain with respect to nystagmus and other reflexes resulting from labyrinthine fluid movements in this pair of canals.

Just as the horizontal canals are so paired, the vertical canals are paired; the right anterior vertical with the left posterior vertical, and the left anterior vertical with the right posterior vertical canals. Each pair co-operates in the same manner with respect to lymph movements, sensations, and reflexes in the plane of its canals. This co-operative conjugation serves to maintain a state of functional balance between the two halves of the apparatus.

While it is true that the physical effects of motion in actuating fluid movement within the canals are equal and opposite with respect to utricle-

ward movement of the fluid, it is not true that the physiological effects from the end-organs of a conjugate pair of canals are *equal*. Comparisons of the results of stimulations of equal intensity, as shown by vertigo and nystagmus, prove conclusively that the same ampulla emits impulses which produce greater vertigo and greater nystagmus when the fluid is made to flow in one direction than when it is made to flow in the opposite direction. And it has further been determined that the relation between the unequal physiological effects is approximately two to one. Thus, in a vestibular

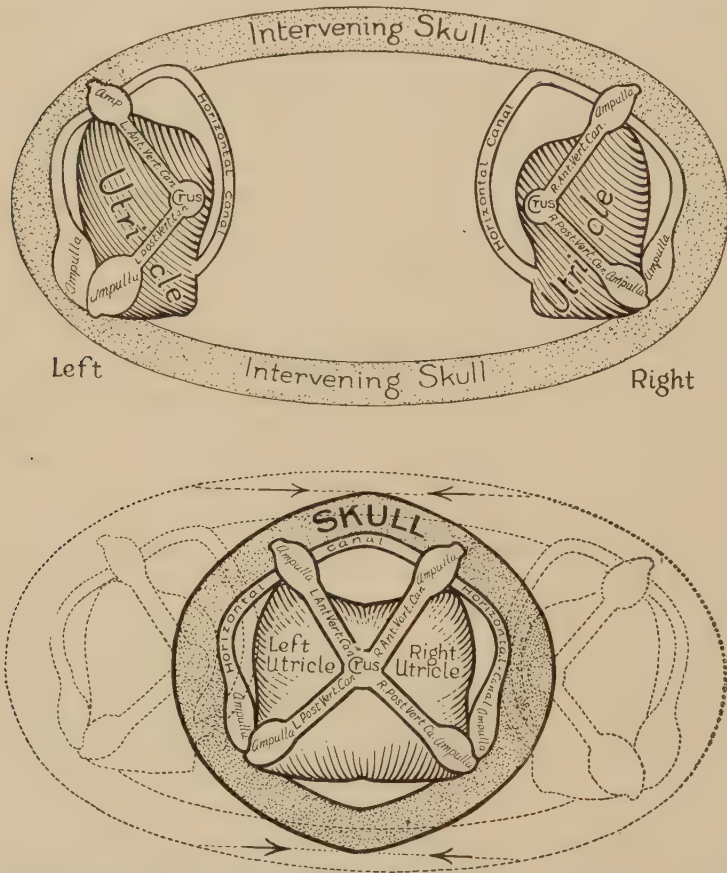


Fig. 364.—Schematic representation of the physical mechanism of the vestibular apparatus. The upper illustration presents the anatomic separation of the right and left halves; the lower illustration represents the two halves juxtaposed functionally.

apparatus only the right half of which is functioning, the horizontal nystagmus to the left produced by rotation to right endures for about eight or nine seconds, whereas the horizontal nystagmus to the right produced by rotation to the left endures for about sixteen or eighteen seconds. Similar differences are seen in the physiologic effects of the same caloric stimulation of an end-organ, first at 120 degrees forward tilt, then at 60 degrees backward tilt. The greater effect of the same stimulus is produced when the fluid movement is away from the utricle in the vertical canals, when it is toward the utricle in the horizontal canals. Normally rotation

produces a physiological effect such as vertigo or nystagmus which embodies the contributions from each half of a pair of functionally conjugated end-organs. It is theoretically probable that horizontal nystagmus to the left caused by rotation to the right is contributed to in the proportion of two-thirds from the left ear and one-third from the right. The same relative proportion obtains respecting the right and left contributions to vertigo and other reactions resulting from normal end-organ stimulation simultaneously in both ears.

When anything occurs to upset the functional balance normally existing between the right and the left halves of the apparatus this upset results in certain definite manifestations, as follows: Vertigo, nystagmus, fall-tendency, past-pointing, and systemic signs of sympathetic disturbance—pallor, sweat, nausea, vomiting, and faintness.

This functional balance may be upset in two ways: By monolateral increase in afferent impulses, such as results from local stimulation of one side only; or by monolateral decrease in afferent impulses, such as results from functional depression of one side only. With respect to preponderance it matters not whether the impulses from the left half are decreased or the impulses from the right half are increased; in either case the right side preponderates in the emission of afferent impulses. This particular preponderance results in vertigo (sensation of rotation) toward the right, rotatory and horizontal nystagmus to the right, falling to the left, and past-pointing to the left, pallor, sweat, nausea, vomiting, and faintness. With gradual lessening of this preponderance these results tend to disappear in the reverse of the order named. Preponderance of the right horizontal canal tends to produce vertigo sensation to the right in the horizontal plane; of the right vertical canals, vertigo sensation to the right in the transverse vertical plane, the composite vertigo sensation resulting from the mixture of these two is a general sensation of rotating to the right. Preponderance of the right horizontal canal tends to produce horizontal nystagmus to the right; of the right vertical canals, rotatory nystagmus to the right; the composite of eye movements resulting from these individual nystagmus tendencies is mixed horizontal and rotatory to the right. The composite fall-tendency caused by preponderance of the right horizontal and the right vertical canals is to the left, as is the past-pointing.

Bilateral equal depression or exaltation of function in the two halves of the vestibular apparatus does not cause vertigo, nystagmus, falling, or past-pointing; it produces pallor, sweat, nausea, vomiting, and faintness, depending upon its degree of intensity.

Monolateral sudden depression or abrogation of function does cause onset of vertigo, nystagmus, falling, past-pointing, pallor, sweat, nausea, vomiting, and faintness; if monolateral depression of function takes place very gradually, it may proceed to the degree of total abrogation without producing any of these reactions.

Similarly monolateral exaltation of function causes vertigo, nystagmus, falling, past-pointing, pallor, sweat, nausea, vomiting, and faintness. Some or all of these reactions to vestibular irritation are present, their degree being proportionate to the intensity of the irritation responsible for them. It is obvious that, with respect to stimulation, its effects become manifest as the result of upsetting, whether by monolateral exaltation or depression,

this balance existing between the two halves of the physical apparatus of the vestibule.

**Varieties of Nystagmus.**—The term “nystagmus” is used to describe more or less regular involuntary eye movements of several varieties.

“Miner’s nystagmus” is an occupation neurosis motor phenomenon in which the eyeballs rotate in the vertical plane irregularly upward and downward; it is not intensified by any particular direction of gaze and may disappear during mental abstraction. It is not believed to be of vestibular origin, but its nature is not fully understood.

“Optical nystagmus” is a pendulum-like, to and fro, irregular rotation of the eyeballs, always in or near the horizontal plane. It is found associated with congenital anomalies of the eyeball such as albinism, persistent hyaloid, polar cataract; and with other congenital and developmental defects of the oculocerebral apparatus associated with low visual acuity and high refractive error. It is characteristic of *miner’s* and *optical* nystagmus that the eye movements are not necessarily associated with any subjective sensation of movement, either of fixed objects or of the subject of the nystagmus.

“Vestibular nystagmus” is a rhythmic rotation of the eyeballs, a slow pull in one direction followed by a quick jerk in the opposite direction. It may occur in any plane or in any direction depending upon the nature of the cause. It is a physiological oculomotor response consisting of two components, the first of which, the slow pull, represents the physiological visual fixation of gaze upon a moving object; this slow pull is a pure reflex of vestibular origin. The quick return jerk is of a more complex nature. In that it has to do with voluntary direction of gaze, the quick return jerk has a cerebral volitional element; in that it represents a tendency of the muscles to return to a pre-existent state of quiescence—a restoration of the one muscle (or set of muscles) from a state of contractility or of antagonist (or antagonists) from a state of proportionate stretching—it is of a reactive nature. In this reactivity there is a certain element of reflex response to the deep sensibility, afferent impulses arising in the muscles and tendons as a result of the initial slow pull; there is also an element of innate elasticity of the extra-ocular muscles and tendons. In all writings on the subject the direction of vestibular nystagmus has been indicated by the direction of the quick return jerk; for example, if the slow pull of the eyes is toward the right and the quick jerk pulls them back to the left, the nystagmus is spoken of as being “to the left.”

The plane of rotation of the nystagmus has been indicated by the terms “rotatory,” “horizontal,” “vertical,” “oblique,” and “mixed.” The term “rotatory” is used to describe the nystagmus which would occur if each eyeball rotated about a needle transfixing it horizontally through the pupil; the term “horizontal” is used to describe the nystagmus which would occur if each eyeball rotated about a needle transfixing it vertically; the term “vertical” is used to describe the nystagmus which would occur if each eyeball rotated about a needle transfixing it horizontally from side to side; the term “oblique” is used to describe the nystagmus which would occur if each eyeball rotated about a needle transfixing it midway between the transfixions described for “rotatory” and for “vertical” nystagmus; the term “mixed” is used to describe any such mixture as “mixed horizontal and rotatory to the right” for example. Rotatory nystagmus is further

described according to the direction of its quick component as "rotatory to the right" or "to the left"; horizontal nystagmus is thus further described as "horizontal to the right" or "to the left"; vertical nystagmus is thus further described as "vertical nystagmus upward" or "downward"; oblique nystagmus is further described according to its individual plane of rotation as well as its direction, *e. g.*, "obliquely upward to the right," or "obliquely downward to the left."

When the fluid within the semicircular canals is set into motion, the oculomotor expression of that motion is very definite with respect to the plane of the movement which has excited the flow of intralabyrinthine fluid. The eyeballs rotate about an axis exactly vertical to the plane of the exciting movement, and the slow pull of the resulting rhythmic nystagmus is always in the direction of the exciting movement.

*Posture 1.*—For example, starting with the head upright so the horizontal canals lie in the horizontal plane; if now the head is rotated about the vertical axis to the right, the resulting nystagmus is horizontal (each eyeball rotating about a vertical axis), and the slow pull is toward the right. In this instance the eyeballs rotate in the exact plane of the stimulated canals (the horizontals) and the slow pull of the nystagmus is in exactly the same direction as the flow of the fluid within the horizontal canals.

*Posture 2.*—If the head be turned 45 degrees to the right and inclined 90 degrees over the right shoulder, the planes of the left anterior vertical and the right posterior vertical canals are brought into the position of being at right angles to the (vertical) axis of rotation. After being rotated about the vertical axis in this posture the labyrinthine fluid is flowing in the left anterior vertical from the utricle upward, in the right posterior vertical toward the utricle, downward. With the head upright the quick jerk of the resulting nystagmus is "obliquely downward to the left"; in other words it is in the oblique, anteriorly-to-the-left, posteriorly-to-the-right plane of the head, and the direction of the slow pull is obliquely upward, to the right.

*Posture 3.*—If the head be turned 45 degrees to the left and inclined 90 degrees over the right shoulder, the planes of the right anterior vertical and of the left posterior vertical canals are brought into position of being at right angles to the (vertical) axis of rotation. After being rotated about the vertical axis in this posture the labyrinthine fluid is flowing in the right anterior vertical canal from the utricle upward, in the left posterior vertical canal toward the utricle, downward. With the head upright the quick jerk of the resulting nystagmus is "obliquely downward to the right," in other words it is in the oblique anteriorly-to-the-right, posteriorly-to-the-left plane of the head, and the direction of the slow pull is obliquely upward to the left.

*Posture 4.*—If the head be turned neither 45 degrees to the right nor 45 degrees to the left, but faced straight ahead (half way between these two directions) and inclined 90 degrees over the right shoulder, the planes of the right anterior vertical and the left posterior vertical canals will be brought into position of being at an angle of 45 degrees to the (vertical) axis of rotation; at the same time the planes of the left anterior vertical and the right posterior vertical canals will also be brought into position of being at an angle of 45 degrees to the (vertical) axis of rotation. After

being rotated to the right about the vertical axis in this posture, the labyrinthine fluid is flowing in all four of the vertical canals; in the left anterior vertical and the right posterior vertical it is flowing in the same direction as in Posture 2; in the right anterior vertical and the left posterior vertical it is flowing in the same direction as in Posture 3. The eyeballs therefore are subjected simultaneously to two different pulls, one tending to produce nystagmus whose quick jerk would be obliquely downward to the left, the other to produce nystagmus whose quick jerk would be obliquely downward to the right. This of course is a physical impossibility and the obvious effect is the resultant of these two forces, namely, a rotation in the plane midway between the two different planes—which is the anteroposterior vertical plane of the skull; inasmuch as the directional tendency of each of these forces is the same, the slow pull of the resultant nystagmus is upward, and there is produced “vertical nystagmus downward.”

*Posture 5.*—If the head be turned neither to the right nor the left, but faced straight ahead (as in Posture 4) and inclined 60 degrees backward, the planes of the right anterior vertical and the left posterior vertical canals will be brought into position at 45 degrees to the vertical axis; at the same time the planes of the left anterior vertical and the right posterior vertical canals will also be brought in position at 45 degrees to the vertical axis. After being rotated to the left about the vertical axis in this posture, the labyrinthine fluid is again flowing in all four of the vertical canals; in both anterior and posterior left vertical canals it is flowing from the utricle upward and toward the right, in both anterior and posterior right vertical canals it is flowing toward the utricle, downward and toward the right. Under these conditions the eyeballs are again subjected simultaneously to two different pulls, one tending to produce nystagmus obliquely forward to the right, the other to produce nystagmus obliquely backward to the right. The resultant of these two forces falls in the vertical transverse plane of the skull, which produces “rotatory nystagmus”; in both component forces the directional tendency was to the right, so the slow pull of the resulting rotatory nystagmus is to the left—so-called “rotatory nystagmus to the left.”

Thus it is apparent that horizontal nystagmus is the expression of oculomotor influence from *one pair* of semicircular canals—the horizontals; that oblique nystagmus (either oblique forward to right or oblique forward to left) is also the expression of oculomotor influence from *one pair* of semicircular canals. In this sense, from the standpoint of canals, there are two “simple nystagmus” types, horizontal and oblique, and two “compound nystagmus” types, vertical and rotatory; in addition there are “mixed nystagmus” varieties—“horizontal and rotatory,” “horizontal with oblique,” “rotatory with vertical,” etc. From the standpoint of plane of rotation it is possible to produce at will nystagmus in any plane or in any direction, simply by rotating about an axis at right angles to the plane of the nystagmus to be produced.

*From the physiological standpoint* it should be especially emphasized that there exists no direct connection between certain of the end-organs of the canals and certain extra-ocular muscles. It has been schematically represented in certain writings on this subject that the horizontal canals are directly connected with the external and internal rectus muscles, the posterior vertical canals with the superior and inferior obliques, the an-

terior vertical canals with the superior and inferior rectus. In this manner it has been further explained that horizontal nystagmus results from horizontal canals stimulation, rotatory nystagmus from posterior vertical canals stimulation, and vertical nystagmus from anterior vertical canals stimulation. This conception has no known basis of fact, and is contrary to accepted conceptions of the manner of operation of the oculomotor mechanism. The "oculomotor brain" in the region of the geniculate bodies, corpora quadrigemina, III, IV, and VI nuclei, and their association fibers, operates as an independent mechanism controlling all eye movements, receiving mandatory impulses from all sources and emitting co-ordinated motor impulses which control conjugate or convergent eye movements.

Certain lesions interfering with the functional operation of the "oculomotor brain" are manifested by lack of normal co-ordination of conjugate ocular movements, yet by no impairment of any of the individual ocular movements involved in executing these convergent or *conjugate co-ordinations*. The oculomotor nuclei are essential integral parts of the "oculomotor brain," and are, as such, subject to its operative influence and control; the incoming impulses resulting from stimulation of the vestibular end-organs are so co-ordinated among all other similarly incoming impulses in the "oculomotor brain"; their oculomotor result upon the eyes is conjugate rotation in the plane and the direction of the flow of labyrinthine fluid. But this is not proof of direct neuronal connections between individual vestibular end-organs and individual nuclei of the third, fourth, and sixth nerves. The vestibular impulses touch in upon a definite oculomotor "pattern," and certain definite eye movements result.

**The Nature of Nystagmus.**—Ordinary eye movements under practically all conditions consist of two main phases, the fixation of gaze upon some object and the quick movement of directing the gaze to the next object of fixation. If the objects fixed upon are still, the eyes are still during the first phase; if the objects fixed upon are moving, the eyes move with the moving objects. The movement of directing the gaze upon the next chosen object of fixation is always a quick jerk.

Bright objects, brilliant colors, or lights, against less striking background, attract the fixation of the gaze either consciously or subconsciously. If a cylinder of solid color be rotated before the eyes, no special eye movements will result; but if the cylinder be striped in striking colors, the eyes will execute a series of movements as long as the rotation continues, and the movements will be specifically: (1) A slow pull in the direction of the rotating stripes; (2) a quick return jerk in the opposite direction. This is normal physiological nystagmus—the result of fixing the gaze upon a stripe attracting visual attention as long as it is in sight, and successively fixing the gaze upon other stripes as they rotate into and out of view. During a railroad journey gazing out of the window causes the same series of eye movements, the gaze being consciously or subconsciously fixed upon landscape objects as they come into view successively. This is called "railroad nystagmus"; its slow pull is always in the direction of the passing of objects, the quick jerk in the direction of the train's progression; the plane of the eye movements is always that of the train movement responsible for it.

While nystagmus is an ocular phenomenon, its nature is such as to be understood only when studied from an approach based upon broad general

physiological considerations. The "pattern" concept suggested by Jendrasssek differs radically from any previously advanced in dissertations upon the subject of nystagmus; it takes no cognizance of the relations conceived to exist between certain ampullar end-organs and certain extra-ocular muscles, but recognizes much broader and more fundamental biological principles and physiological facts.

The evolution of species attests the truth of certain biological principles, among the first of which is that natural adaptations trend toward higher efficiencies in inherited characteristics. Complicated motor co-ordinations, varied by individually acquired modifications in generation after generation, come gradually to be laid down in the germ-plasm as more or less definitely predetermined "reaction patterns." This conception of "reaction patterns," as elaborated by Ingham, constitutes a fundamental explanation of practically all instinctive acts and generic automatisms. Once the "pattern" comes into circuit, the neuromuscular apparatus performs in more or less exact accordance with the pattern requirements. The patterns represent co-ordinations acquired through age-long lineage adaptations to environmental requirements. The swimming movements of baby fish, the flying movements of baby birds, or the sucking movements of newborn mammals are examples of spontaneous motor performances fundamentally similar regardless of familial relations. In this simple biophysical concept of inherited tendencies toward the maturing of certain cell groups according to definite generic or species "patterns" is found an explanation of the similarity of inherited motor reactions. Just as the "anlage" of the gonads are predestined to attain maturity only at the age of puberty, so are the "anlage" of other tissues predestined to attain definite functional stages at various developmental periods. In the case of the sucking apparatus of mammalian infants, the actual cellular neuromuscular elements are adequately developed at birth; in the case of the flying mechanism of baby birds, the actual cellular neuromuscular elements are not completely developed until the fourth or fifth week after hatching. In both instances further developmental maturity brings with it additional technical facility and physical capabilities, constituting acquisitions on the part of the individual. As a result of such acquisition, the four weeks old baby is able to nurse better and longer than it could at birth; the ten weeks old fledgling is able to fly more accurately and longer than it could at its first flight. But the "pattern" of each of these co-ordinations was laid down at the moment of beginning of embryonic existence of the individual, with a certain definite predestination of its attaining workability at a certain stage in the development of the individual. With the higher and higher development of visual efficiency of each of the two eyes comes the necessity for binocular fixation of the gaze upon the same object at the same moment, in order to avoid the confusion of simultaneous distinct, but dissimilar, visual impressions. A marked degree of conjugation of eye movements exists at birth by virtue of an inherited co-ordination pattern; but the individual acquisition of higher and higher degrees of conjugate ocular co-ordinations proceeds hand in hand with other mental developments until, during the period of infancy, fully established binocular fusion is evolved. Eye movements actuated by the requirements of visual efficiency demand certain anticipatory direction of gaze of each eye simultaneously, prior to, or in process of, fixation of gaze of sufficient duration

to permit the cerebral registration of a clear visual image. During sustained movement of an individual in one direction, this results in an arrest of the gaze as it fixes upon a certain object which is thereupon clearly sensed visually before falling behind out of view as the body progresses; followed by a quick jerk of the eyeballs forward in the direction of the body movement, in fixing the gaze afresh upon some newly chosen object; and so on during the continuation of the sustained forward movement of the individual. This succession of slow reverse pullings of the eyeballs and of quick return jerks, resulting from such sustained forward movement of the body, is physiological or "railroad nystagmus." This type of "pattern" has developed, obviously in answer to the demands of *visual efficiency* for the body in motion. Rotation to the right causes "railroad nystagmus" whose quick jerk is to the right; its plane is always that of the body movement responsible for it. With eyes closed, or with unseeing eyes, a normal individual on being subjected to the same sustained movement develops certain *involuntary* or *reflex* eye movements which are *not actuated by vision*, but depend upon nerve impulses originating independently in the motion-sensing apparatus of the ears. These eye movements consist of a slow pulling followed by a quick jerk in the opposite direction; they continue as long as the impulses continue to emanate from the vestibular end-organs; they occur in certain definite direction and plane, the direction of the quick jerk always in that of the rotation, and the plane of the eye movements always in that of the body movements responsible for it. This nystagmus is called "vestibular nystagmus during rotation."

Thus two special senses, each functioning individually, each concerned with adjustments relating to movement, each having its special reaction to every form of movement, are seen to be so intimately related as to have a "common reaction pattern," which acts as an interlocking director. In other words, a certain motion will produce *visually* in the absence of vestibular function, or *vestibularly* in the absence of visual function, the *identical series of eye movements* called nystagmus.

Certain commentators have expressed doubts as to the vestibular apparatus subserving a special sense function, despite the functional characteristics which seem to fulfil the requirements of a special sense; *the identity* of the vestibular eye movement reflex with physiological visual nystagmus constitutes a pertinent additional indication of the special sense nature of this *motion-sensing apparatus*.

This "vestibular nystagmus during rotation" is identical with the "railroad nystagmus" caused by this rotation. As has been explained under "The Physical Mechanism" (p. 675), the physical stimulation of the end-organ endures only until the fluid within the canals has taken up the rotational movement; as the vestibular nystagmus during rotation only continues as long as the impulses continue to emanate from the end-organs, it is apparent that with continued rotation at a constant speed this nystagmus gradually ceases. On ceasing to rotate, with closed eyes or unseeing eyes, the normal individual develops certain involuntary or reflex eye movements *which are not actuated with respect to visual considerations*, but depend upon nerve impulses originating in the vestibular portions of the ears. These eye movements consist of a slow pull followed by a quick jerk in the opposite direction; they continue as long as impulses continue to emanate from the vestibular end-organs; they occur in certain definite

direction and plane, namely, the slow pull always in the direction of the rotation, the plane always in the plane of the head coinciding with the plane of the rotation. This nystagmus is called "vestibular nystagmus after rotation"; it is, in direction, the reverse of the railroad nystagmus occurring during the rotation responsible for it.

#### ANATOMICOPHYSIOLOGICAL SCHEME OF VESTIBULAR APPARATUS

The vestibular system comprises an intricate number of nerve pathways; some of these are definitely known, others are purely hypothetical. Beginning with the end-organ in the ampulla, the peripheral neuron includes the ganglion vestibulare and extends to the medulla oblongata. The vestibular nerve fibers run to Deiters', von Bechterew's, and triangular nuclei. Here the horizontal canal tract separates from the vertical canals tract. This separation is of microscopical proportions. In this region are the beginnings of various nerve connections, including the vestibulospinal, the vestibul sympathetic, the vestibulo-ocular, and the vestibulocerebral. These various connections and the separation of the vertical canals tract from the horizontal canal tract form the beginnings of intermediate nerve paths of the vestibular system.

**From Horizontal Canal.**—The nerve tract from the horizontal canal, having thus separated from the vertical canals tract, is presumed to undergo division into separate pathways, one leading ultimately to the sensory cerebral cortex, the so-called "sensory path"; the other leading to the oculomotor nuclei, the so-called "oculomotor path," from the horizontal canal. The "sensory pathway" from the horizontal canal is presumed to enter the cerebellar hemisphere of the same side (hypothetically *via* the inferior cerebellar peduncle) and there to connect with the cerebellar nuclei, globosus, emboliformis, and tecti. The "oculomotor pathway" from the horizontal is presumed to proceed to the oculomotor nuclei more or less directly near the midline (hypothetically *via* the posterior longitudinal bundle).

**From Vertical Canals.**—The nerve tract from the vertical canals after separating from the horizontal canal tract, is presumed to extend upward, lateral to the midline, to about the level of the middle of the pons varolii. Here this tract is presumed to undergo division into two separate pathways, one leading ultimately to the cerebral cortex, the so-called "sensory path," from the vertical canals, the other pathway leading to the oculomotor nuclei, the so-called "oculomotor path" from the vertical canals. The "sensory pathway" from the vertical canals is presumed to enter the cerebellar hemisphere of the same side (hypothetically *via* the middle cerebellar peduncle) and there to connect with the previously mentioned cerebellar nuclei. The "oculomotor pathway" from the vertical canals is presumed to proceed more or less directly to the oculomotor nuclei (hypothetically *via* the posterior longitudinal bundle). Further presumptions, as yet not fully proved, are the following: From the cerebellar nuclei a nerve pathway connects with the sensory cortex of the cerebrum (hypothetically *via* the superior cerebellar peduncle and crura cerebri; also, hypothetically, these fibers are distributed to the cerebral cortex of both sides, the greater portion of the fibers decussating to the opposite cortical distribution). The sensory goal of these fibers is presumed to be at or near the second temporal convolution, the supposititious cerebral center for the sense of

motion. Among other connections beginning in the region of the medulla oblongata is the vestibulospinal, which distributes motor impulses of general reflex character to the skeletal musculature. Past-pointing and falling responses to stimulation are expressions of these motor impulses, as are the forced movements described by Magnus and de Kleijn.

It is definitely established that neurovisceral impulses producing pallor, sweat, nausea, vomiting, and faintness follow a different course, obviously communicating with the sympathetic system. This has been referred to by some writers as "the vagus distribution." These anatomical details, however, are not definitely known.

That the "sensory pathways" from these end-organs come into connection with the cerebellum is unique in that no other "sensory" path is known to have such connections, although afferent fibers are known to run in great numbers to the cerebellum in such tracts as the direct cerebellar tract. Tonogenetic impulses originating in the current of afferent impulses emitted by the vestibular end-organs (in common with tonogenetic impulses originating similarly from afferent currents of all sorts) are distributed *via* vestibulospinal connections. When a destructive or nerve-blocking process interrupts the arrival of afferent impulses from the vestibular end-organs of either one or both sides, one of the immediate results is hypotonus in the skeletal muscles, of temporary duration. Conversely, hypertonus results from stimulation of these end-organs, as it does from stimulation of the end-organs of other sensory apparatus. It is not improbable that these tonogenetic impulses are reflected subsequent to contact with the nuclei of the medulla oblongata, but their exact point (or points) of reflection from the afferent tracts is not definitely known.

Cajal and other histologists have identified so-called "acoustic striæ" in the floor of the fourth ventricle, using degeneration methods for identifying fibers. While these histological studies have been sufficient to establish definitely that these "striæ" are of eighth nerve origin, until 1925 it has remained undetermined whether they are of cochlear or vestibular origin. This seems to have been settled definitely by Spiller in a series of sections of the brain and medulla of a case known to have no functional impairment of hearing but definite complete lack of vestibular reactions on stimulation. The finding of pathological lesions involving these "striæ" would seem to suggest their vestibular character.

#### CONCERNING TESTING

In applying the stimulus to test the ear mechanism, accuracy is of great importance. Special devices, such as the modern turning-chair and douche apparatus, make it possible to attain standardization of stimulus which goes far to enable the examiner to obtain consistent results. With a known constant stimulus, comparisons of results are possible and certain deductions can be made. Considering the many variables in the living mechanisms and physiological apparatus involved in ear tests, it is obviously all the more imperative to use all possible accuracy in the *physical mechanisms* producing the stimulus. Even with the most accurately measured and applied standard stimulus there is a certain latitude of physiological responses which fall within normal limits. Fatigue, inattention, uncertainty, all cause the individual subject to vary more or less in indicating accurately the sensory responses to motion stimulus; less variation exists in the motor

responses to stimulation—nystagmus, past-pointing, falling—and in the systemic responses—pallor, sweat, nausea, vomiting, and faintness.

**Galvanic Method.**—Galvanic stimulation measured by rheostatic means has been used in testing this ear mechanism. The writer has had so little experience with this method that his discussion of it would be valueless. Galvanism is not widely used.

**Caloric Method.**—The fluid within the vestibule may be set into motion by changing its temperature, which sets up convection currents resulting in stimulation similar to that produced by turning. This is called “caloric stimulation.” It is obviously susceptible of less precise application than rotation, but is measurable by using constant factors of rate of temperature change, and its effects may be observed with some accuracy. For the purpose of bringing about this change of temperature, cold water at 68° F. is flushed through the external auditory meatus up against the tympanic membrane at a definite rate and volume of flow, and the number of seconds noted until the beginning of the first observable effects of setting up the convection currents within the ear. This first observable effect is nystagmus—a slow *pulling* of both eyes toward one side followed by a quick *jerking* of the eyes in the opposite direction. The use of caloric stimulation has one great advantage over the use of rotation for some tests in that it permits the stimulating of one side only; for instance, douching the right side with head 120 degrees forward or 60 degrees backward stimulates the right horizontal canal only or, with the head 30 degrees forward, the right vertical canals only, according to the choice of the observer, a selective stimulation of canals of one side obviously impossible when rotation is used as the stimulus.

**Rotation Method.**—Motion applied to the body is communicated to the fluid contained within the semicircular canals; when this fluid moves there is set up a disturbance of the physical relations existing during quiescence between the hairs and the gelatinous cap structure (cupula) surmounting the end-organ hairs of the ampullæ; the resulting interaction of cupula and hairs constitutes the actual irritation of the end-organ cells, as the result of which afferent impulses are emitted.

When a subject is rotated (at approximately ten turns in twenty seconds, for example) the starting motion disturbs the relations previously existing between the cellular structures and the fluid within the semicircular canals. If the rotation be to the right the cellular structures are moved to the right, whereas the fluid lags, failing to keep pace with the cellular structures. As long as the fluid continues to lag this disturbance of relations may be expressed in one of two ways—the cellular structures are moving to the right with respect to the lagging fluid, or the fluid is lagging to the left with respect to the cellular structures. After the rotation has continued at the desired speed for a sufficient length of time it comes to pass that the fluid has taken up the movement completely—it no longer lags—and the cellular structures and fluid are moving at the same rate of speed. Then, and not until then, the previously existent relations between cellular structures and fluid are restored and, as long as the rotation continues at the same rate of speed, these relations remain exactly as they were before the start of rotation. If now the rotation is suddenly stopped the relations between cellular structures and fluid are again disturbed, and in the same degree but in the opposite direction. Whereas at the moment of starting

this rotation the cellular structures were moving to the right with respect to the lagging fluid, at the moment of stopping the fluid flows (continuing) to the right with respect to the (stopped) cellular structures. Observations made at this moment of stopping will, therefore, reveal the reactions which would be revealed at the moment of *starting* a similar rotation to the left. Inasmuch as it is practically impossible to make careful observations at the beginning of rotation, those made at the instant of stopping after rotation to the right will suffice as evidence of what would be observable at the beginning of rotation to the left. Conversely, after rotation to the left, the phenomena observed at the moment of sudden stopping will show what would be observable at the instant of beginning rotation to the right. The keynote to a fundamental understanding of rotation tests is the realization that the turning continues long enough to permit the re-establishing of the quiescence relations between the cellular structures and fluid within the canals; so that, at the instant before stopping the rotation, the fluid is not moving with respect to the cellular structures within the vestibules. Then, with the sudden stop (of the cellular structures), the fluid continues flowing in the direction of rotation; during the continuation of this flowing it causes stimulation of the end-organ, allowing the observations of the subject's responses to this stimulation. When it finally comes to rest stimulation has ceased and the quiescence relations between cellular structures and fluid are again established.

It is obvious that a column of fluid within a canal which lies in a plane parallel to the axis of rotation cannot be set into motion by rotation, and that a column of fluid within a canal which lies in a plane perpendicular to the axis of rotation can be set into motion by rotation.

It is therefore necessary in making rotation tests to see that the plane of the canals to be tested lies perpendicular to the axis of rotation. The plane of the horizontal canals is brought into position at right angles to a vertical axis of rotation by tilting the head forward sufficiently to make horizontal a plane intersecting the external canthus of each eye and the roof of each external auditory meatus—a tilt forward of about 30 degrees from the ordinary head-upright posture. The plane of the vertical canals is brought into position at right angles to a vertical axis of rotation by tilting the head 90 degrees forward or backward from this just-described posture—120 degrees forward or 60 degrees backward from the ordinary head-upright posture. It has been determined that ten rotations suffice to overcome the inertia (lagging) of the fluid within the canals, and that rotation at the rate of one turn in two seconds is not unpleasant to the average person. For these reasons standard rotation technic rotates the subject for ten turns at the rate of one turn in two seconds. For purposes of attempting accentuation of stimulus for certain tests (vertigo measurement and past-pointing) rotation is made at a more rapid rate—ten turns in ten seconds; but for nystagmus measurement the standard rate of rotation is ten turns in twenty seconds.

**Results of Stimulation.**—The chief responses of a normal subject resulting from stimulation of the end-organs of the ear motion-sensing apparatus are, as has been set forth, of three varieties: *Sensory*—a subjective sensation of motion; *motor*—nystagmus, falling, and past-pointing; and *systemic*—pallor, sweat, nausea, vomiting, and faintness.

**Vertigo.**—On suddenly stopping rotation to the right with the head

tilted 30 degrees forward (stimulating the end-organs of the horizontal canals) there is produced the sensation of rotating about a vertical axis toward the left ear; rotation in this position to the left produces similar sensation but in the opposite direction. On sitting up after rotation to the right with the head tilted forward 120 degrees (stimulating the end-organs of the vertical canals) the sensation is one of rotation about a horizontal anteroposterior axis toward the left ear; rotation in this position to the left produces similar sensation but in the opposite direction. Suddenly stopping rotation to the right with the head tilted 60 degrees backward (stimulating the end-organs of the vertical canals but exactly reversely from that just described) produces the sensation (on sitting up) of rotating about a horizontal anteroposterior axis toward the right ear; rotation to the left in this posture produces similar sensation but in the opposite direction.

**Nystagmus.**—Suddenly stopping rotation to the right with the head tilted 30 degrees forward (stimulating horizontal canals end-organs) produces nystagmus in the horizontal plane whose slow pull is to the right, whose quick return jerk is to the left—so-called “horizontal nystagmus to the left.” Similar rotation in the opposite direction produces “horizontal nystagmus to the right,” as if the eyeball were rotating around a needle transfixing the eyeball at the points of intersection of its vertical anteroposterior and vertical transverse equators. Rotation to the right with head 120 degrees forward (stimulating vertical canals end-organs) produces nystagmus in the transverse vertical plane whose slow pull is to the right, whose quick return jerk is to the left—so-called “rotatory nystagmus to the left” (as if the eyeball were rotating about a needle transfixing the eyeball anteroposteriorly through the pupil). Conversely, rotation in this posture to the left produces “rotatory nystagmus to the right.” Rotation to the right with the head tilted 60 degrees backward produces “rotatory nystagmus to the right” and conversely, in this same posture, rotation to the left produces “rotatory nystagmus to the left.”

**Falling.**—Rotation to the right with the head 120 degrees forward produces, on sitting up, falling of the body toward the right; rotation to the left, in the same posture, falling to the left. Rotation to the right with the head 60 degrees backward produces falling to the left; rotation to the left in this posture, falling to the right; rotation to right, head 30 degrees forward produces neither right nor left falling, but a tendency toward rotation of the body about a vertical axis toward the left; and conversely, rotation in the same posture to the left produces a similar tendency to the right.

**Past-pointing.**—Rotation to the right, head 30 degrees forward produces past-pointing of each hand (or of both simultaneously) toward the right; rotation in the same posture to the left produces past-pointing to the left. Rotation for the purpose of testing pointing is usually not done in any other position of the head for reasons of impracticability; it is possible to test past-pointing elicited by the vertical canals by rotating the subject in a horizontal position; the past-pointing is normally to the left after rotating clockwise in this position, to the right after rotation counterclockwise.

**Sympathetic Reactions.**—Systemic responses to stimulation of the ear motion-sensing apparatus—pallor, sweat, nausea, vomiting, and faintness—are usually proportionate to two factors, the intensity of the stimulation and the sensitiveness of the subject. They appear in the order named in

direct proportion to these two factors. With respect to the canals, stimulation of the verticals is followed by more pronounced systemic responses than is a similar intensity of stimulation of the horizontals.

#### STANDARD TECHNIC OF TESTING

**Spontaneous Phenomena.**—Standard technic of testing a subject includes first of all the careful observation of the presence or absence of spontaneous phenomena traceable to the ear motion-sensing apparatus. Note is made as to the presence or absence of nystagmus: (1) On gaze straight ahead; (2) on gaze upward; (3) on gaze downward; (4) on gaze to the right; (5) on gaze to the left. Next, as to presence or absence of past-pointing to the right or to the left with head in upright position. Next, as to presence or absence of falling tendency: (1) On gaze straight ahead; (2) on gaze to right; (3) on gaze to left, while standing with heels and toes together, head upright, eyes closed. Any departure from normal is carefully noted, and is registered as "spontaneous rotatory nystagmus to right," or as "spontaneous fall-tendency toward right ear on gaze straight ahead—on gaze toward right—on gaze toward left." Accurate detection of spontaneous phenomena of this nature is of great importance, as they indicate definitely certain abnormalities of the vestibular apparatus.

For the purpose of accurate determination of the presence or absence, as well as of the character and direction of finer degrees of nystagmus, two methods have proved especially valuable in the author's experience. The binocular loupé serves admirably as an instrument of precision in studying nystagmus; and the ophthalmoscope is of even greater value in determining definitely the finer details of nystagmus. With the ophthalmoscope the smallest movements of vessels of the fundus are accurately recognized.

Next in order of routine examination is rotation, first to right at the rate of ten turns in twenty seconds with eyes closed, head tilted 30 degrees forward. On stopping after this rotation the subject is told to attempt to gaze straight ahead at some previously indicated mark, and the exact description and duration of the nystagmus is noted, such as "horizontal nystagmus to the left, small amplitude, twenty seconds duration." This rotation is then repeated to the left, and similar observations are made and recorded. In registering such a finding use may be made of a convenient system of abbreviation, based upon the understanding that the registration sheet is in place of the patient's eyes—*facing the observer*—the arrow indicating the plane and direction and quick jerk of the nystagmus. The above described nystagmus would be set forth as follows: " $\rightarrow$  small, twenty seconds"; rotatory nystagmus to the left, large amplitude, lasting twenty-six seconds would be expressed as " $\curvearrowright$  large, twenty-six seconds"; vertical nystagmus upward, medium amplitude, lasting twenty-two seconds, as " $\uparrow$  med., twenty-two seconds."

Next in order is testing the past-pointing reactions. With the head tilted 30 degrees forward, eyes closed, the subject is next rotated at the rate of ten turns in ten seconds, and the past-pointing is tested, first after rotation to the right and then after rotation to the left. This is done by executing a previously practised act, the object of which is to demonstrate how accurately the subject is able, with eyes closed, to point to or touch the observer's index-finger, after having located this index-finger

with closed eyes by feeling for its position. Once this index-finger has been located definitely by touch, the subject raises his finger to a point higher than his head without bending his elbow, so raising his whole arm straight from the shoulder, after which he attempts to point to, and touch again, the observer's index-finger which has been meanwhile kept in exactly the same position during this entire test.

For several years the writer has made it a routine practice in testing past-pointing to register three observations of the subject's ability previous to rotation; to execute this act of touching the examiner's finger with closed eyes, raising the arm as described, and coming back to again touch the examiner's finger at its original location; the three observations are made first with the examiner's finger held directly in the midposition in front of the subject's finger, second, with the examiner's finger held 8 to 10 inches to the outer side of the first position, third, with the examiner's finger held 8 to 10 inches to the inner side of the first or midposition. These are registered as "spontaneous pointing"—for instance, if the subject, after having the examiner's finger placed in contact with his finger (underneath it) in the midposition, raises his arm above his head as described and succeeds in bringing his finger again into contact with the testing finger of the examiner, such a result is registered as "right arm, midposition—touches"; if in reaching for the examiner's finger the subject's finger reaches 1 inch too far to the right it is registered as "right arm—mid-position—past-pointed 1 inch to the right." Abbreviating these registrations one may conveniently express the results, in the space allotted to "spontaneous pointing," under the caption "Right Arm" as "1 to R, T, 2 to L," which would mean in the outposition the right arm pointed 1 inch too far to right, in the midposition it succeeded in touching accurately, in the inposition it pointed 2 inches too far to left. After such practice in touching, rotation at the described rate is performed and, on stopping, the pointing is tested, first testing the right arm, then the left arm, then the right arm, then the left arm, until the subject succeeds in again touching the examiner's finger with each—right and left. Normally, after rotation to the right, some such reaction as the following occurs:

Right arm	Left arm
12 to R	8 to R
6 to R	4 to R
2 to R	1 to R
T	T

which would mean that the right arm, in attempting to touch the examiner's finger (which is always first brought by the examiner into contact underneath the subject's finger in the position to be maintained by the examiner's finger) past-pointed 12 inches to the right, then the left arm past-pointed 8 inches to the right, then the right arm past-pointed 6 inches to the right, then the left arm past-pointed 4 inches to the right, then the right arm past-pointed 2 inches to the right, then the left arm past-pointed 1 inch to the right, then the right arm finally succeeded in touching the examiner's finger, then the left arm finally succeeded in doing the same.

The fall reaction is next tested as follows: With the eyes closed, the subject bends forward in the turning chair so that his forehead rests upon his fists which in turn rest upon his knees. When a line connecting the external canthus and the roof of the auditory canal is vertical the head

is 120 degrees forward, which brings the plane of the vertical canals at right angles to the vertical axis of rotation. In this position the subject is rotated very slowly (at the rate of one turn in four seconds) for five revolutions. He is then told to sit up without opening his eyes. Normally after rotation to the right, he manifests a decided fall to the right; after rotation to the left, a decided fall to the left. At times this fall tendency reaches the intensity of a violent sprawl, the head and body falling in the normal direction, the opposite leg sprawling outward toward the reverse direction. Such a phenomenon has a component of the nature of a genuine "forced movement."

The next step in standard technic of rotation tests is to measure the type and duration of the subjective sensation of motion (vertigo) caused by rotation with head tilted 30 degrees forward, eyes closed, at the rate of ten turns in ten seconds. The subject is carefully instructed to maintain a continuous current of words describing the direction he feels he is moving—by saying "right, right, right, right, right, still, still, still, still, still (when he feels he is still), left, left, left, left," etc.—*never* ceasing to report by some word every second until the examiner says: "That is enough." During the continuation of the rotation to the right, he will normally continue to report: "Right, right, right." At the moment of stopping he should report: "Left, left, left" for between sixteen and twenty-five seconds at which time he will report: "Still, still, still," as indicating that he no longer feels any sensation of movement.

Inasmuch as his buttocks touch, sole touch, and general deep sensibility may enable him to realize that he is actually not moving even while his ear motion-sense makes him feel as if he were still moving, to the right, for example—it is necessary to explain fully that he is to report his *sensation of moving* to the right as long as he feels that sensation, *even though his common sense* tells him he is not actually moving—for it is this sensation of movement that is being measured by this test.

Not all the foregoing standard tests can be carried out consecutively at one sitting—for the reason that the successive stimulations add up an intolerable degree of disturbance, which induces pallor, sweat, nausea, vomiting, and faintness. These normal systemic reactions to stimulation should be watched for carefully and registered on the chart at the moment of their observation, as "pallor, sweat after two turnings" or "pallor, sweat, nausea after one turning," or "pallor, sweat, nausea, vomiting after two turnings," etc. With the exercise of care it is possible to avoid inducing vomiting by interrupting the testing as soon as marked pallor and sweat are observed. The testing can be resumed at a later hour or day, and thus can be completed piecemeal without more than mildly unpleasant experiences on the part of the subject.

The next step in the standard technic of testing is the caloric test of the right ear, using 2 quarts of water at 68° F., flowing through a nozzle against the drum-head at the rate of about 21 ounces per minute; with the head tilted 30 degrees forward, a suitable basin must be held so as to catch the douche-water as it runs from the ear canal. Normally rotatory nystagmus to the left  $\curvearrowright$  begins in between thirty-six and seventy-six seconds; the number of seconds and character of nystagmus are registered, and the douching is continued for about thirty to fifty seconds longer in order to set up convection current conditions *very positively* before proceeding to complete

the caloric test. With eyes closed the past-pointing is examined for each arm with the head in the upright position (30 degrees forward). This is normally from 3 to 8 inches to the right with each arm, the extent depending upon the rapidity with which the arm is lifted and brought back in search of the testing finger. The head is then tilted backward 60 degrees in order to bring the horizontal canal into the vertical plane so that convection currents can be set up within it. When this has occurred the nystagmus changes to horizontal to the left; then in this position the past-pointing with closed eyes is examined first with the right then with the left arm. Normally this is from 6 to 12 inches to the right with each arm, again depending upon the rapidity with which the pointing is executed. After the character, direction, and intensity of nystagmus and the past-pointing are registered, the subject is brought to standing position with toes and heels together, eyes closed, and head upright for the purpose of testing fall tendency. Normally this is marked sway or even actual falling toward the ear which was douched. This is observed with the head facing straight forward, then facing to the right, then to the left; in each position the fall tendency should be manifest toward the douched ear. This is registered and the ear promptly douched with 8 to 12 ounces of water at 102° F. to bring to an end the caloric stimulation. At another sitting the same procedure is gone through with the left ear, the findings of which are normally as just described except that left must be substituted for right and right for left throughout.

Following the completion of the foregoing tests of the semicircular canals apparatus, the findings are carefully studied in comparison with the normal; it is frequently advisable to revise certain findings which stand forth as questionable or inconsistent. The effect of revision is either to verify or to alter details of the original test findings, with the result that the examiner feels moderately sure of the reliability of the items comprising his tests of the ear motion-sensing apparatus. The findings are then set aside for future use in consideration with a careful history, the findings on physical examination, including general physical and general neurological as well as upper respiratory, eye examinations, and cochlear examination.

#### **PRACTICAL APPLICATION OF WHAT IS KNOWN CONCERNING THE VESTIBULAR APPARATUS**

The known facts concerning the ear motion-sensing apparatus are to be made of all possible use in attempting to solve the problems presented by the sick. Unfortunately anatomy and physiology are not yet sufficiently developed to answer many questions concerning the centers and tracts involved in this study. Pioneers in this field of work have been forced to postulate centers and tracts whose existence seems certain despite the fact that they have not yet been identified anatomically.

The first step in attempting to render knowledge concerning the ear motion-sensing apparatus of practical value to the sick should be to formulate the best possible working concept, incorporating in it all known anatomical and physiological facts.\*

\* In 1910 the study of the vestibular apparatus had reached a point at which further progress could not take place upon known anatomical facts. Declining to brook indefinite delay awaiting the establishment of new anatomical facts, the very nature of which certainly promised to require years, decades perhaps, Bárány postulated centers in the cerebellum in more or less direct connection with the vestibular nerve and proceeded to

Such a concept must necessarily be completed by the tentative adoption of the best available hypotheses to fill in the gaps. It is worthy of particular emphasis to remember at all times that this working scheme is at best hypothetical and must be held subject to change whenever further facts become available or whenever more plausible hypotheses are put forth. On the other hand it should be emphasized that progress must be seriously interfered with if scientific men are to refrain from applying any of the known facts because there is available less than a full complement of scientifically proved details concerned in formulating a working concept. Such a concept has been built up gradually during the past two decades. In it have been incorporated the contributions of many careful students, beginning with Flourens in 1828. This has been utilized clinically by otologists and neurologists in attempting to solve problems in diagnosis—particularly in intracranial diagnosis.

That it has failed to enable those who have attempted to use it to solve the entire problem in every case is not surprising, nor is this necessarily a proof that the concept itself is fundamentally fallacious. Obviously much must depend upon the individual accuracy and judgment with which it is applied. In the hands of a capable otologist and a painstaking neurologist it may confidently be expected to approximate an accurate diagnosis more nearly than any alternative method of study. It is probably best formulated in general terms, these terms to be supplemented in every case by the individual findings. There should be first considered the neurons originating in the end-organ and completing the peripheral link to the nuclei, three of which lie in the medulla oblongata—Deiters', triangularis, and von Bechterew's; three of which lie in the cerebellum—the globosus, emboliformis, and fastigii (or tecti). That actually more than one neuron exists in this link should not operate to nullify the conception of this distinctly "*peripheral link*." The third or central link should next be considered comprising the neurons extending roughly from the basal ganglia to the supposititious cortical center of the cerebrum.

Even the certain knowledge that this supposititious cortical center does not exist as a definitely circumscribed anatomical entity, and that more than one neuron is included in this "*central link*," again should not operate to nullify its conception as a central link *per se*. The second link, or the spinocerebellocerebral link intermediating between the first and the third links, is by far the least understood and the most involved of

elaborate theories respecting the operation of this complex ear mechanism. He followed the precedent of similar postulates in cerebral and cerebellar localizations by André Thomas, and in sensory-motor lesions of cortical epilepsy by Hughlings Jackson. Further, Bárány made perfectly clear that the gaps thus bridged were only tentatively bridged, and only by postulates because nothing else was available. Many years later Jones followed these precedents by postulating certain tracts to bridge an impasse in studies of the vestibular intracranial connections. These tracts were suggested by neurologists of authority, and were only postulated because absence of definite knowledge concerning tracts compelled a choice between accepting something hypothetical or nothing. In using these postulated tracts Jones reiterated in his writings that they were purely hypothetical.

Despite their most careful disclaimers, much criticism has been made of Bárány and Jones because of their having assumed the existence of certain centers and tracts which have never been proved.

The writer believes that the practical value of their constructive theorizing has fully justified itself. Even though otology is still compelled to do without scientific confirmation of the existence of the centers and tracts which have been postulated, these working theories must be used until such time as substitution of more plausible hypotheses or of additional known anatomical facts becomes possible.

the constituents going to make up the working concept of the vestibular tracts.

This link, extending as it does *from the nuclei* where occurs beginning ramification of the afferent pathways leading from the vestibular end-organs *to the central neurons* ending in the cerebral cortex, must include connections with nerve mechanisms of many orders and natures. Associations with other sense mechanisms—notably the spinal sensory connections subserving deep sensibility and tactile sense, and the ocular connections subserving vision; other collaterals involving motor mechanisms—notably the skeletal muscular system, and the oculomotor system; and still other collaterals involving the splanchnic and sympathetic systems, are definitely known to be integral with this second or intermediary link between the periphery and the center. Phylogenetic “pattern” automatisms, and racial, familial, and individual “pattern” co-ordination mechanisms must be included in this second link, as must be those of experience and acquisition, of grace or awkwardness, of agility or slowness of bodily movements. It is definitely established anatomically that the end-organ cell sends an axis-cylinder to the ganglion vestibulare in the apex of the petrous pyramid, from whence a second axis-cylinder extends to the medulla oblongata nuclei. Just what neuraxial connections exist between the afferent neuron and the nuclei is not fully known, but it is anatomically established that these medulla oblongata nuclei have direct axis-cylinder connections with the cerebellar nuclei—globosus, emboliformis, and tecti. Careful analysis of the symptoms, test findings, and operative or autopsy findings in many cases seems to justify the following statements: That a more or less direct tract goes from the cerebellar nuclei to a cerebral center for motion-sensing; that this center lies at least partly in or near the second temporal convolution; that a more or less direct tract goes from the nuclei of the medulla oblongata to the oculomotor nuclei. The first of these two tracts is indicated by the demonstrated absence of usual sensory signs of stimulation of the end-organ in cases of proved lesion involving cerebellar hemispheres and in cases of proved lesion in or near the second temporal convolution. The second of these tracts is indicated by the demonstrated absence of rotatory nystagmus on stimulation of the vertical canals end-organ in many cases of proved lesion at or near the midline on the level of the pons varolii.

That the motor tract from the vestibular end-organs of either side is more or less directly concerned with the oculomotor co-ordinations of both sides is indicated by the production of normal nystagmus movements of the two eyes similar in direction, extent, and rotation plane by stimulation of the end-organs of either side; furthermore, by the ability to change at will the direction or the plane of the nystagmus by altering the position of either right or left, or of both end-organs during the continuation of stimulus.

That dissociation of ocular movements due to disturbed oculomotor co-ordinations is not due to lesion along the motor tract but is attributable to elision in the oculomotor centers themselves is indicated by: (1) Failure to develop oculomotor incoordinations in cases of proved lesions at various points along the vestibulo-ocular tracts; (2) development of inverted and perverted and incoördinate nystagmus in cases of proved lesion at or near the midline in the region of the oculomotor centers.

## VESTIBULAR RESPONSES TO CHEMICAL IRRITATIONS

The eighth nerve structures are affected especially by certain agents of a chemical nature; of these agents inorganic compounds such as those of the salicylic group, organic compounds such as those of the quinine group, toxins of mumps, syphilis, and typhoid deserve special mention. The end-organs and their neuraxes show evidence of special affinity for these agents, both as irritants and as destructants. Considerable evidence exists indicating that nicotine and the toxins produced by many common bacteria also produce low-grade toxic effects upon these eighth nerve sensory elements, resulting in primary stimulation or irritation, and subsequent impairment of sensory acuity. Acute intoxication due to absorption from the gastro-intestinal tract, so-called "bilious" attacks, are notoriously associated with vertigo, pallor, sweat, nausea, vomiting, and faintness. The writer has had opportunity to observe acute "bilious" attacks during which nystagmus was observed in addition to the symptoms already mentioned. Such acute vestibular irritations, with or without secondary functional impairment, are completely recoverable in many instances; chronic intoxications, drawing little or no attention to themselves as such, may be responsible for marked impairment of cochlear or vestibular functions. Gingival and alveolar infections are also concerned in impairments of this nature, especially those by spirilliform organisms of the Vincent's angina order.

## PERVERTED NYSTAGMUS

Stimulation of the vestibular end-organs normally results in certain definite eye movements, which have been described. Under abnormal conditions stimulation results in eye movements which differ from normal eye movements in plane of eyeball rotation, in direction of the movements, or in both. This kind of abnormal nystagmus is called "perverted nystagmus." It may be spontaneous, or on stimulation, or both.

The only difference between spontaneous nystagmus and nystagmus on vestibular stimulation is that the latter is produced by *artificial application of vestibular stimulus*; in either case the nystagmus is an expression of stimulation, either relative or absolute, as has been set forth.

The nature of the stimulus responsible for spontaneous nystagmus is either general or local. A general stimulus acting either upon the end-organs of one or both sides, or on the end-organs and central nervous connections, if it results in nystagmus, theoretically must produce mixed horizontal and rotatory nystagmus. A local stimulus, acting only upon circumscribed portions of the vestibulo-ocular tract, may produce vertical or rotatory nystagmus, mixed or pure; but theoretically does not produce pure oblique or pure horizontal nystagmus, for reasons which have been set forth under the "Mechanics of Nystagmus." Spontaneous nystagmus, therefore, which expresses a general stimulus, theoretically, must be mixed horizontal and rotatory; expressing a local stimulus acting on the end-organs of one side, must be mixed horizontal and rotatory. If, therefore, a spontaneous nystagmus is found on examination to be pure rotatory or pure horizontal it may be said to be "perverted" in that it expresses an oculomotor reaction which must be the effect of less than the full oculomotor current from *one* half of the vestibular end-organs (in case of peripheral origin); or (of impulses originating at some point along the vestibulo-

ocular pathways) of less than the full oculomotor current from *both* halves of the vestibular end-organs.

Stimulation of the vestibular end-organs normally results in certain definite eye movements—the varieties of normal nystagmus already described. Under abnormal conditions stimulation results in conjugate eye movements which differ from the normal in plane of eyeball rotation. This is called “perverted nystagmus.” It is due to intracranial lesion of such a nature as permits the passage of the vestibulo-ocular impulse from the end-organ to the eye muscles, but not along normal pathways. For instance, if the right vertical canals, douched with water at 68° F., produce horizontal nystagmus or vertical nystagmus, perversion of the normal is evident.

What is known at present concerning the workings of the vestibular mechanism points to infratentorial location of lesions responsible for perverted nystagmus.

#### INVERTED NYSTAGMUS

The term “inverted nystagmus,” is used to describe abnormal nystagmus whose plane is identical with, but whose direction is opposite to the normal. For instance, rotating to the right normally produces horizontal nystagmus to the left; if, instead of this, horizontal nystagmus to the right occurs, this constitutes inversion of nystagmus. Inverted nystagmus suggests infratentorial location of lesion responsible for it.

#### INVERTED FALLING AND PAST-POINTING

Normally, falling and past-pointing produced by stimulation of the vestibular end-organs occurs in the plane and direction of the motion producing the stimulation. Under certain abnormal conditions they occur in the direction opposite the normal. This is termed “inverted” falling and past-pointing.

Inverted falling and past-pointing suggest abnormality, the nature of which may be hysteria, or intracranial or intraspinal pathological cellular lesion affecting the vestibulospinal distribution.

#### EVIDENCE OF VESTIBULAR RESPONSE TO STIMULATION

If *any* of the reactions which have been described become manifest upon stimulation of the end-organs of this apparatus they constitute proof that the vestibular function is not lost. Any combination of partial loss of vestibular reactions may be encountered; stimulation may fail to elicit nystagmus, vertigo, falling or past-pointing, but the subject may manifest pallor and sweat. Another subject may manifest no nystagmus, no sympathetic symptoms, but active fall tendency or past-pointing. Regardless of details, positive reactions of any kind prove functional activity of peripheral nerve elements, eighth nerve, and portions of the central connections of the vestibular apparatus. These manifestations constitute definite proof:

1. That the end-organs are discharging afferent impulses, and
2. That the eighth nerve is conducting these afferent impulses to the central nervous system where
3. They are conducted to their nervous system distribution.

#### VESTIBULAR CHARACTERISTICS OF PERIPHERAL LESION

While it is impossible to state completely the characteristics of “peripheral” lesions, there are certain recognized characteristics suggesting

peripheral location of lesions responsible for certain abnormalities. In general, irritative lesion of the end-organs or eighth nerve of one side produces mixed horizontal and rotatory nystagmus to the side of the lesion, fall tendency toward the side opposite the lesion, past-pointing to the side opposite the lesion, vertigo sensation of rotating toward the side of the lesion, and sympathetic responses—pallor, sweat, nausea, vomiting, and faintness—depending upon the intensity of the irritation.

Coincidental signs of cochlear irritation are frequently present, such as hyperacusis and tinnitus.

There seems to be no sign or reaction differentiating between lesion of the end-organs and lesion of the eighth nerve. A lesion depressing or blocking function of the end-organs or eighth nerve of one side, if of sudden onset, produces the reverse of the above mentioned reactions; if of sufficiently gradual onset, may produce no spontaneous reactions. There may be coincidental depression of cochlear function with hypo-acusis and tinnitus. Responses to test stimulation are exaggerated in the presence of irritative lesion and are subnormal in the presence of a depressing or blocking lesion proportionate to the extent of the irritation or depression, but are otherwise not different from the normal responses.

#### VESTIBULAR CHARACTERISTICS OF INFRATENTORIAL LOCATION OF LESION

Spontaneous nystagmus observed in association with infratentorial lesions is characteristically rapid and of small amplitude; and in contradistinction to that of peripheral origin, is not commonly mixed horizontal and rotatory, but either vertical, oblique, horizontal, rotatory, or dissociated. Infratentorial lesion may fail to produce spontaneous nystagmus, but may cause perversion of nystagmus elicited by stimulation or dissociation of ocular movements. It may also be responsible for total absence of nystagmus on stimulation.

Exaggerated spontaneous vertigo, especially if the vertigo be markedly systematized (sharply defined with respect to plane and direction) suggests infratentorial or peripheral location of lesion. Exaggerated spontaneous fall tendency and past-pointing suggest infratentorial or peripheral location of lesion.

Spontaneous pallor, sweat, nausea, vomiting, and faintness are frequently associated with infratentorial or peripheral lesions, but in infratentorial lesions it is usually impossible to cause any great increase in these symptoms by moderately intense stimulation.

#### VESTIBULAR CHARACTERISTICS OF SUPRATENTORIAL LOCATION OF LESION

Spontaneous ocular movements tending toward conjugate deviation are frequently observed in association with supratentorial lesions. Even if these are oscillatory they should not be regarded as true nystagmus because they are of the nature of *cerebral pattern reactions* and are not expressions of the vestibular nystagmus pattern. Under these conditions nystagmus from vestibular stimulation tends to resolve itself into deviation conjugué in the direction of the slow component. Myers has called attention to the fact that a series of supratentorial lesions showed that stimulation of the vestibular end-organs on the side of the lesion produced deviation conjugué to that side; the same stimulation applied to end-organs of the opposite side produced vestibular nystagmus. The reason for this

difference becomes obvious if one considers the result of superimposing the vestibular "pattern" upon the cerebral "pattern." On vestibular stimulation, nystagmus of slow rate and wide amplitude suggests supratentorial location of lesion.

Vertigo associated with supratentorial lesion is characteristically slight or absent—either spontaneous or on stimulation. Past-pointing and falling reactions are also less than normal in association with supratentorial lesions.

#### CEREBELLOPONTILE ANGLE LESION SYNDROME

A single lesion at or near the junction of the eighth nerve and brainstem frequently causes total abrogation of function of the cochlea, the horizontal and the vertical canals of the same side and of the vertical canals of the opposite side. This is called the "cerebellopons angle lesion syndrome," and establishes diagnosis of some lesion—whether exudative, infiltrative, or neoplastic—of such a nature as to block afferent impulses of the entire homolateral eighth nerve and afferent impulses of the opposite vertical canals fibers only.

The angle of pons and cerebellum is the anatomical location of a single lesion capable of causing such nerve blocking.

#### DISSOCIATION OF OCULAR MOVEMENTS

Normally all ocular movements are identical in extent, direction, and plane except those movements requiring convergence; in all binocular movements the element of so-called "conjugation" is effected by the coordinating "oculomotor brain" (posterior geniculate, corpora quadrigemina, third, fourth, and sixth nuclei, and the associated nerve connections). Nystagmus eye-movements, like all normal eye movements, are normally *conjugate*. Under certain abnormal conditions ocular movements are normally conjugated on cerebral and visual mandates (voluntary direction of gaze), whereas stimulation of the vestibular end-organ produces nystagmus characterized by dissimilar movements of the eyes, those of one eye differing from those of the other either in extent, direction or plane; for instance, the right eye shows horizontal nystagmus while the left eye shows vertical nystagmus, or the right eye shows rotatory nystagmus while the left eye shows oblique nystagmus; any such combination may occur.

This kind of dissociation of ocular movements suggests location of lesion in the region of the so-called "oculomotor brain."

The problems of construing the findings in certain cases showing departure from normal reactions are various and difficult. While it has been assumed that the entire end-organ apparatus of one side is known to be functioning if any part of it can be shown to be functioning, this is not proved, and is open to question. For example it is not impossible that the left horizontal end-organ may be functioning while the left anterior vertical end-organ may not be functioning.

The author made a study of R. H., twenty-eight years, who showed the following findings: Spontaneous vertical oblique nystagmus downward to the right, spontaneous past-pointing to the left, spontaneous fall toward the left ear in all positions of the head, no pallor, sweat, nausea, vomiting, or faintness; visual acuity 6/IV in each eye, visual fields normal for form and color, normal ocular fundi, other cranial nerve functions normal; general motor and sensory findings normal; general physical and blood

examinations negative; history negative except for bilateral purulent otitis media since age three following severe upper respiratory infection; present illness four weeks, beginning with general malaise, no fever, sudden violent vertigo on waking two weeks ago, accompanied by pallor, sweat, and nausea lasting four days.

Pain in left ear developed a week ago, when she was found to have large furuncle in left external auditory meatus which was incised. Following this pain disappeared; Roentgen rays of both mastoids failed to reveal evidence of active bone destruction, showing only general vague clouding usual in such long-standing tympanic suppurations; leukocytosis moderate, and differential count normal. Ultrarapid rotation to right, head 30 degrees forward failed to produce nystagmus, vertigo, past-pointing, pallor, sweat, or nausea; ultrarapid rotation to right, head 120 degrees forward failed to produce nystagmus, vertigo, pallor, falling, sweat, or nausea; rotation to right, head 90 degrees over right shoulder, gaze straight ahead produced marked vertical nystagmus downward and faint vague vertigo; ultrarapid rotation to left, head 30 degrees forward failed to produce any nystagmus, vertigo, past-pointing, pallor, sweat, or nausea; ultrarapid rotation to left, head 120 degrees forward produced slight falling to left, but no nystagmus, no pallor, sweat, or nausea; rotation to left, head 90 degrees over left shoulder produced marked vertical nystagmus downward, and faint vague vertigo. Twenty-six consecutive ultrarapid rotations of ten turns each in all positions produced no definite vertigo, pallor, sweat, nausea, vomiting, or faintness. Cochlear function is unimpaired, right or left, high tone limits full normal, Rinné negative each ear with only moderate conduction impairment; all conjugate eye movements are normal, as are pupillary reactions and deep and superficial reflexes.

From these findings one must deduce that cellular pathological changes exist intracranially or peripherally in the nerves or end-organ cells of both right and left sides, to the extent of abolishing the usual nystagmus, vertigo, and sympathetic reactions from the horizontal end-organs even on intense stimulation; not sufficient in degree to abolish the marked vertical nystagmus downward on stimulating the vertical canals, yet sufficient to abolish rotatory nystagmus to the right or to the left, even on repeated ultrarapid rotation in supine position, and to abolish vertigo and sympathetic reactions from these same vertical canals. Conceiving these cellular changes to be in the end-organ cells it is further necessary to conceive certain cells of one ampulla being affected to the point of not causing normal reactions, while others of the same ampulla remain able to participate in causing normal reactions. The intracranial alternative conception of location of cellular pathological change must be distal to the oculomotor brain—in other words, symmetrically located in both sides of the brain-stem region. Against the latter theory of location is the absence of any neurological evidence of motor, sensory, or other abnormality. A lesion in this region, sufficient to be responsible for such extensive blocking of vestibular afferent impulses from both sides, might confidently be expected to reveal some evidence on most searching neurological study. Extra-ocular muscle co-ordinations elicited by visual mandate (railroad nystagmus) and by voluntary mandate (direction of gaze in all directions) are normal: it is conceivable that some lesion of medulla or brain-stem might interfere with normal nerve connections between the oculomotor brain

and its special contacts with the fibers from the vestibular tracts, with the resulting oculomotor manifestations noted in this case. But this pathology has not yet been demonstrated and it is difficult to conceive of a lesion sufficient to be responsible for the blocking here apparent in the oculomotor pathway, the vertigo pathway, and the sympathetic and vestibulospinal pathways, without causing some demonstrable departure from normal in other nerve mechanisms.

In the presence of such evidence one is compelled to choose between two theories, one involving lesions difficult to conceive in the periphery (end-organs and eighth nerve), the other involving lesions even more difficult to conceive in the central nervous system. Until definitely established by pathological findings it is necessary to maintain an open-minded attitude toward either or both of these possibilities in attempting to attain a working diagnosis regarding such a case.

The foregoing furnishes an example of the cases presenting problems of diagnosis. Such problems are fraught with difficulties as yet not solved. There are, however, many examples in the average run of cases whose diagnosis may be established with satisfactory exactness by otological and neurological collaboration in applying what is known today concerning the vestibular apparatus.

EUGENE R. LEWIS.

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### SYPHILIS OF THE EAR

Syphilis may manifest itself in any portion of the auditory apparatus and the simplest method of classification for its study is according to its location in the external, middle, or internal ear. In the primary form it only occurs in the external ear. Secondary syphilis may affect both the external and middle ear, whereas the inner ear is only directly affected by tertiary syphilis although serious disturbances may be caused in the labyrinth as the result of secondary syphilitic disease of the middle ear, causing tissue changes. By far the greater proportion of cases of aural syphilis are hereditary, as compared with acquired.

Knapp<sup>1</sup> quotes a remarkable statement by Hinton that, "Hereditary syphilis furnishes more than one-twentieth of all the aural patients at Guy's Hospital." Knapp comments that this statement is certainly not in accord with the statistics regarding such cases from other institutions. Twenty-five years ago the author<sup>2</sup> studied 2500 consecutive cases treated in the Out-patient Department for Diseases of the Ear of the Pennsylvania Hospital. Out of this number there were but 7 cases which could be considered as of distinctly syphilitic origin. This was, of course, before we had the serological aides in diagnosis which we now possess. Doubtless many patients suffering from syphilis were not suspected, but at all events nothing in the aural condition of the other 2493 patients seems to have aroused a suspicion that it could be associated with syphilis.

## SYPHILIS OF THE EXTERNAL EAR

1. Primary lesions on the external ear are extremely rare. Politzer<sup>3</sup> had apparently never seen one. He refers to 4 cases. One was reported by C. Pellizzari, in which infection followed the use of a handkerchief which had been previously used by a syphilitic. The second was reported by J. Zucker as resulting "from the too fervent love caresses of a publican." The third case was reported by Hermet as occurring in a woman who was caressed by her syphilitic husband. Hermet related the fourth case to Politzer as a phagedenic ulcer of the auricle and lobule following the bite of a syphilitic. Dépres<sup>4</sup> reports a case of chancre of the auricle. L. Duncan Bulkley<sup>5</sup> in an analysis of 9058 cases of extragenital chancre found 27 cases in which the chancre was located on the external ear. He reports a case occurring in a man who was bitten on the ear by another who had mucous patches in his mouth. Mracek<sup>6</sup> in a study of 400 extragenital chancres found but three located on the external ear. In this connection it is interesting to recall that one of the charges brought against Cardinal Wolsey was that he had given Henry VIII syphilis by whispering in his ear. There is no doubt Henry had syphilis but there were many more simple ways by which his infection might have been explained.

2. Secondary syphilis of the auricle and external auditory canal is most common as the accompaniment of cutaneous lesions involving the face or scalp. One of the cases which I observed at the Pennsylvania Hospital was that of a man in whom a syphilitic skin eruption of squamous nature had extended to the auricle. Syphilitic perichondritis of the auricle has been quite frequently reported. The author<sup>7</sup> reported a case of this character, occurring in a negro twenty-five years of age. H. Mendel<sup>8</sup> reports a similar case in a woman. M. A. Goldstein<sup>9</sup> and G. W. Linthicum<sup>10</sup> each report a case of bilateral syphilitic ulceration of the auricle occurring in negroes. C. D. Roy<sup>11</sup> reports a case of condyloma of the external auditory meatus in a colored man who had secondary syphilis. Knapp<sup>12</sup> reports condylomata of both external auditory canals in a woman who had contracted syphilis six months before, and refers to a case reported by Wilde in his "Practical Observations on Aural Surgery," and to the fact mentioned by Stohr that he had seen 14 cases of flat condylomata of the canals, 11 of which were in females. Noquet<sup>13</sup> also observed bilateral condylomata of the external auditory meati. Dépres<sup>14</sup> in 1200 syphilitics observed condylomata of the external auditory in 5 cases. Politzer reports several cases in which he observed syphilitic ulcerations within the external auditory canal.

Most of the cases of syphilis of the external ear are acquired, but the author observed one case in which the disease was undoubtedly inherited. The patient was a white boy, aged nine months. The mother said that the child's father was syphilitic. When seen at the out-patient department there was a large broken down gumma forming an abscess in the left auricle, involving the tissues above and below it. The pus was evacuated and under antisyphilitic treatment the child improved rapidly.

## SYPHILIS OF THE MIDDLE EAR AND EUSTACHIAN TUBE

Syphilitic manifestations involving the eustachian tube should be classed in the group with those occurring in the middle ear because in most instances syphilis of the middle ear results from infection from the nose or

throat through the eustachian tubes, except in the cases in which the middle ear is involved as part of a constitutional syphilis. Suppuration in the middle ear is frequently observed in patients suffering from syphilitic lesions in the neighborhood of the eustachian-tube orifices in the nasopharynx, and may be considered as the result of direct infection through the tube. In some case the infection of the middle ear has been ascribed to the use of the catheter. The author has observed a primary sore in the throat involving the tissues immediately surrounding the pharyngeal eustachian orifice on one side, and many similar cases have been reported. The use of the Politzer bag or catheter would certainly be strictly contraindicated in such cases.

Poltizer in his "Text-book of Diseases of the Ear" states that Baratoux is supposed to have observed in a case of syphilis a small opalescent gumma on the membrana tympani, and that Kuschner also saw a syphilitic ulcer on the membrana tympani. Such manifestations must be very rare as a careful search of the literature has failed to reveal any similar cases.

Acute or chronic suppuration in the middle ear is no more frequent in association with syphilis than it is in non-syphilitics. Woakes, many years ago, made the assertion that chronic purulent otitis media in strumous children was the result in many cases of syphilitic caries of bone in the middle ear. This statement is quite incapable of direct proof. In a negative way the evidence at hand would indicate that syphilis must play a very indirect rôle, if any, in the maintenance of chronic purulent otitis media, because of the thousands of such cases which every aurist sees, of which practically none ever show improvement or cure as the result of antisymphilitic treatment. Poltizer states that the only characteristic symptoms pointing to the syphilitic origin of a middle-ear suppuration are rapid destruction of the membrana tympani and loss of sound perception through the bones of the skull. The occurrence of multiple perforations in the membrana tympani in purulent otitis media has been advanced as absolutely characteristic of a syphilitic origin. Every experienced aurist has observed such multiple perforations in cases where syphilis could be absolutely excluded.

#### SYPHILIS OF THE INNER EAR

By far the most frequent and most serious manifestations of aural syphilis are those which occur in the inner ear. They usually occur late in the disease, although Poltizer saw a case in which the inner ear was involved seven days after the appearance of the primary lesion. Syphilis of the inner ear may occur in either acquired or congenital syphilis. In the congenital form it rarely manifests itself before the seventh or eighth year, for which reason it does not seem as though hereditary syphilis was as large a factor in the production of deaf-mutism as it is generally considered. The pathological changes observed are chiefly a serous exudation into the labyrinth, with deposits of connective tissue and new bone, especially in and about the round and oval windows. The walls of the labyrinthine arteries are said to show signs of endarteritis and the ganglion cells degeneration. Hemorrhagic extravasation in the labyrinth is a not uncommon finding and probably is often accountable for the usually sudden onset of symptoms. If labyrinthine suppuration occurs it is probably associated with syphilitic necrosis of the temporal bone.

Many of the cases of deafness due to diseases of the inner ear in syph-

ilis are undoubtedly the result of a syphilitic meningitis affecting the auditory nerve-sheaths. Knick and Zaloziecki<sup>15</sup> think this is by far the commonest cause of syphilitic deafness. This view is supported by the great improvement in the hearing power which is seen in many of these cases following lumbar puncture, causing relief of any intracranial pressure. Willcutt<sup>16</sup> thinks that the disturbance of the acoustic nerves in the early stages of syphilitic labyrinthine involvement is due to a toxic syphilitic irritation of the nerve-sheaths and also of the peripheral end-organs, these being most sensitive to the toxin, the acoustic nerve being in fact the most sensitive of all the cranial nerves to such toxic influence.

Wintermute<sup>17</sup> believes that syphilitic neuritis of the auditory nerves is the most common form of luetic disease of the inner ear. Wintermute, while stating that neuritis of the auditory nerve may be due to other causes, such as drug poisoning, alcohol, points out that the majority of cases are due to syphilis. The cochlear branch is usually affected first, and may be solely involved. The symptoms vary according to the amount of infiltration of the nerve, in proportion to the number of the fibers incapacitated by pressure. The vestibular symptoms—vertigo, nystagmus, nausea—usually appear later than the cochlear. This serves as a diagnostic guide to distinguish auditory neuritis from labyrinthine disease, such as serous labyrinthitis, because in the latter the symptoms occur simultaneously.

The so-called “fistula symptom,” nystagmus to the opposite side on using the Siegle otoscope, has been regarded as significant of labyrinthine syphilis. Hill Hastings<sup>18</sup> reports a case of hereditary labyrinthine syphilis in which it occurred.

Jonathan Hutchinson<sup>19</sup> first noted the occurrence of facial palsy in syphilis of the inner ear. He recorded a case in 1879, attributing the facial palsy to involvement of the sheaths of the facial nerve from its proximity to the labyrinth.

There are several very characteristic features attending the onset and progress of syphilis of the inner ear. One of the most striking of these is the suddenness of its onset. It is not infrequent for the patient to have had no premonition of any aural disturbance until he suddenly became deaf. This deafness is usually accompanied by loud tinnitus and vertigo, and is in the majority of cases bilateral. Although both labyrinths are usually simultaneously involved, one may be more profoundly affected than the other. The deafness is sometimes total but usually not so at first. It progresses rapidly so that in a few days the hearing may be entirely lost. There is from the outset marked diminution in the perception of sound through the cranial bones and a positive Rinné test.

In a most interesting article on disturbances of the acoustic nerve in the early stages of syphilis Willcutt<sup>20</sup> considers the occurrence of a very characteristic symptom, namely, shortened bone conduction. Crockett,<sup>21</sup> as early as 1897, noted the condition but did not stress its importance, but, as Willcutt states, Wanner in 1903 and Oscar Beck in 1911 first directed attention to the great diagnostic importance of this shortened bone conduction in syphilis of the inner ear.

On examination there are no characteristic objective symptoms to be found in the affected organ.

With the above facts in mind whenever a patient presents himself with

the history of deafness of sudden origin in one or both ears, accompanied by tinnitus and vertigo, with loss or diminution of bone conduction, particularly if the condition is bilateral, no time should be lost in having a Wassermann test performed. The time element is a most important factor in the successful treatment of these cases. As the condition is a late manifestation in many patients, the history of syphilitic infection can be elicited either by voluntary admission on the part of the patient or by the observation of other symptoms. Finally in any suspicious case the Wassermann test should be tried at once.

#### TREATMENT OF AURAL SYPHILIS

With the discovery of salvarsan the treatment of syphilis of any kind underwent an entire change. Some years after it had been in use reports were made of a number of cases of auditory neuritis following its application. A number of otologists reported cases in which aural symptoms had been apparently aggravated or even provoked by salvarsan therapy. Ehrlich said that these cases were neurorecidives or syphilitic focal infections, occurring because salvarsan sterilizes the body by destroying the spirochetæ, except in instances such as are seen in the auditory nerve, which lies in a narrow canal with a poor blood-supply. In such a locality the spirochetæ multiply rapidly, producing a local reaction, with consequent pressure on the nerve. G. E. Davis<sup>22</sup> after a careful summing up of the evidence in the case concludes that most of these early unfortunate cases were due to faulty technic in the administration of the salvarsan. They are certainly much less frequent now than they formerly were, and no one would be justified at the present time in withholding salvarsan or some similar preparation from a patient for fear of producing any reaction in the auditory nerve.

Although the antisyphilitic treatment of a patient suffering from aural syphilis should be placed in the hands of a serologist, the observation of the aural condition and local treatment of the patient are of the greatest importance.

The local lesions of the auricle and external auditory canal require great care in order to avoid unsightly deformity or cicatricial contractions. Ulcers, condylomata, or broken down gummata should be kept clean, and treated by dusting with calomel, iodoform, or bismuth powders. Mercurial ointments are of service. Exuberant granulations should be destroyed by nitrate of silver, chromic acid or, if necessary, curettement.

It is most important that the condition of the inner ear should be carefully noted at brief intervals. Symptoms may arise at any time showing disturbance of the cochlear or vestibular labyrinth, or the auditory nerve, and may have a great bearing on the patient's welfare. At one time subcutaneous injections of a 2 per cent. solution of pilocarpine hydrochlorate pushed to the physiological limit were supposed to have a beneficial effect in syphilis of the internal ear by lessening the tendency to the formation of exudate. At present there is a tendency to abandon all efforts to affect the internal ear except by means of constitutional treatment. Vertigo, nausea, or intense tinnitus are to be treated symptomatically by rest, ice-bags, and sedatives such as the bromides. The patient's general condition must be kept as good as possible and the hygiene of his life carefully regulated.

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CONGENITAL DEAFNESS

The term by which congenital deafness has been most generally recognized and classified is *deaf-mutism*. In the light of scientific and pedagogic progress this term should be relegated to the past together with obsolete methods of education and the earlier theories of the pathology of the labyrinth. Deaf-mutism, if regarded as a congenital factor, would imply congenital deafness and congenital mutism. Congenital deafness is an established entity; congenital mutism *per se* is of such rare occurrence that it need not be considered as a separate classification. Mutism, when occurring in conjunction with deafness, is usually a circumstantial and not a congenital factor.

Speech, normally considered, is a voluntary act and function dependent on the accurate imitation of the sounds we hear. If, then, a child is born deaf and does not hear speech and is, therefore, not in a position to imitate those sounds, it consequently and eventually becomes mute or dumb. If this status exists until the child is ten to twelve years old there will be a resultant atrophy of the intrinsic muscles of the larynx and an ankylosis of the mechanical organs of articulation that will preclude the development of flexible speech.

Where the older methods of training the deaf child were used—methods by which the deaf child was taught by signs and finger spelling—the opportunities for acquiring speech were meager and such pupils consequently became dumb or mute and there originated the expressions “The Deaf-mute” and “The Deaf and Dumb.”

Today the opportunities for the congenitally deaf child to acquire speech, as manifested by Oral Methods of Training, have been so generally recognized and disseminated that no congenitally deaf child need now remain

mute or dumb. It may be stated as a general axiom that *every congenitally deaf child has the potentialities of speech and, if properly trained at an early age, may acquire fluent, flexible, understandable speech.*

The training of the deaf child has undergone an important evolution. Early in the history of Schools for the Deaf in this country the majority of deaf children were trained by the "Sign Method" and the "Manual Method." Trained by these methods the opportunity for the development of speech in this group of pupils was an unsatisfactory one. Efficiency in scholarship and social contact was concentrated on the acquisition of the Sign Language and the Manual Alphabet. Good scholarship was acquired, but these pupils were socially ostracized and compelled to communicate only with those familiar with their Signs or the Manual System.

The steady growth and improvement in the Oral Method has brought about a very advantageous and economic uplift for the deaf child. Where he is given the benefit of training by the Oral Method he is taught Speech as his method of contact with his normal fellows and is taught Lip-reading as a substitute for his handicap of deafness. Progress by Speech Methods has been so consistent and so gratifying within the past few years that the American Medical Association, through its Special Committee on the Deaf Child, has adopted a general resolution, as follows: Resolved: That we recommend exclusively Oral Instruction of deaf children and that the Oral Teaching of the congenitally deaf child and the acquired deaf child be made a part of a department of the public school system in all larger cities.

This resolution was also adopted by "The American Association to Promote Speech to the Deaf" and by the "Society of Progressive Oral Advocates," both national associations of teachers of the deaf.

Another interesting development in the education of the deaf child is the introduction of the Acoustic Method. We define the Acoustic Method as follows: *Stimulation or education of the hearing mechanism and its associated sense-organs by sound vibration as applied either by voice or any sonorous instrument.*

This definition is comprehensive enough to include: (a) Voice and musical sounds directed through the physiological tract of the ear to the peripheral or central auditory areas. (b) Sound vibration as sensed by tactile impression to interpret pitch, rhythm, accent, volume. (c) Analysis of speech-sounds by tactile differentiation. (d) Synthesis and speech construction by tactile impression. (e) Sound-waves and their significance as appreciated by optical perception.

It is a confirmed observation that the majority of congenitally deaf children retain some remnant of their hearing. If this residual hearing can be stimulated at an early period by various measures of the Acoustic Method, a working-factor of hearing may be recovered in a considerable percentage of cases. This factor may serve to secure for the deaf child the hearing of his own voice by sound-amplification and thus help to regulate and modify his own speech production.

Recent surveys in Schools for the Deaf throughout the Nation have placed the estimate of pupils with some residuum of hearing at over 30 per cent., and it is in this large group that the application of the Acoustic Method in conjunction with other modern methods of training affords the most gratifying results.

In the conclusion of this brief résumé we may safely prognosticate that there has been a great impetus developed in the education of the deaf child, and not least among the stimulating factors in this movement is the awakening interest and co-operation of the members of the American medical profession in general and of the otologists in particular.

MAX A. GOLDSTEIN.

## ANESTHESIA IN OPERATIONS ON THE NOSE, THROAT, AND EAR

**Definition.**—The term “anesthesia” as herein used is defined to be a state of general or local insensibility produced by the inhalation, introduction or application of an anesthetic.

**Shall the Anesthesia be General or Local?**—Practically all operations upon the nose and throat are performed under anesthesia and the operator must decide whether there shall be complete insensibility known as general anesthesia, or insensibility solely about the operative area, known as local anesthesia.

In rendering his decision he will be influenced by the fact that local anesthesia is the anesthesia of choice by a large majority of operators in throat conditions, and is the anesthesia of choice by practically all operators upon the nose in this country.\*

General anesthesia is given most frequently in operations upon the ear. To decide as to which form of anesthesia to apply, the operator will be guided by the nature of the operation and its duration, the age of the patient, and diseased conditions present.

Extensive operations, involving much loss of blood—as in removal of the superior maxilla, laryngectomy, often a tedious operation, and extensive operations on the tongue—are usually best performed under general anesthesia. Tonsil operations in adults, where the tonsil has become fibrous and hemorrhage during operation is feared or the operation is apt to be unduly prolonged, are also best performed under general anesthesia.

Patients with advanced pulmonary disease, with marked valvular disease of the heart, and those with pronounced disease of the kidneys often react unfavorably to the general anesthetic, while the local anesthetic is borne well.

The age of the patient must be taken into consideration. It is practically useless to attempt any operation under local anesthesia on a child under fourteen years of age, except in those instances where the child can be reasoned with and the procedure explained. The writer has seen instances of remarkable fortitude in the very young while being operated upon under local anesthesia. But these are the exceptions. Children dread the knife, the sight of blood, or the thought of pain, and cannot, as a rule, be persuaded that there is to be no pain. Far better to give no

\* The writer has met but one operator among many hundred interviewed who stated that he invariably used general anesthesia in throat and nose operations.

anesthetic than attempt the use of local anesthesia in these latter instances.

Next must be considered the condition of the nerves of the patient. The adult who dreads the thought of operation, who cannot bear the sight of an instrument or the sight of blood, should not be forced to have the slightest operation performed while conscious. It must be borne in mind that the surgeon, from great familiarity, considers an operation with a much more detached frame of mind than does the patient to whom it often assumes terrifying proportions.

As a rule, all operations are best performed in properly managed institutions.

#### GENERAL ANESTHESIA

Produced either by the inhalation of chloroform, ether, nitrous oxide gas, or ethyl chloride, or per rectum by means of tubes. As to the latter, its use is too infrequent for general comment, but it has in its favor, as far as operations in the upper air-passages are concerned, that the anesthesia may be continued without interfering with the operator when a "few more whiffs" may be needed.

Anesthesia is best administered by a trained specialist, whose sole attention is directed to the patient's respiration and condition; and whose judgment is often of great value in the selection of the anesthetic to be used.

Nitrous oxide gas is too evanescent except for the shortest of operations nor is it entirely free from danger. The same is true of the chloride of ethyl. Both of these are, however, frequently used to induce primary anesthesia before the general anesthetic is given. Chloroform owing to its toxicity has given place, in this country, to the far safer sulphuric ether. A few make use of scopolamin, but its use is not only limited but has a large element of danger. The following is the—

**Mode of Procedure in Inducing General Anesthesia.**—Bowels cleared beforehand. Stomach kept empty for not less than six hours before the use of the anesthetic. One hour before the beginning of the operation, adults may receive a hypodermic injection of  $\frac{1}{4}$  grain morphine with 1/150 grain atropine. The anesthetist induces primary anesthesia by the use of nitrous oxide gas or inhalation of ethyl chloride. This is followed by the inhalation of the vapor of sulphuric ether, using a specially constructed apparatus for this purpose.

The narcosis in tonsil operations is profound and complete, and both tonsils are resected and adenoid tissue removed without recourse to further anesthesia, before the patient regains consciousness.

**Anoci Association.**—The rule of anoci association, consideration of the minutest detail of everything conducive to the comfort of the patient, soothing and allaying his nervous dread, should be strictly adhered to. There must be absence of noise, of conversation, and comments on the patient's condition in his presence in the operating or anesthesia room. The presence of the patient's own physician most frequently aids materially to the restful feeling of content and confidence so essential in inducing perfect anesthesia.

**Synergistic Local Anesthesia.**—The use of this method in head surgery is advocated by King<sup>1</sup> with special reference to novocaine, magnesium-sulphate, and morphine.

It has been established by Gwathmey that the effect of morphine is

increased when chemically pure magnesium sulphate is added. King had previously used as preliminary to novocaine injections,  $\frac{1}{8}$  grain morphine in 2 c.c. of a solution of magnesium sulphate; two such injections were given at half-hour intervals.

Hooper and Gwathmey found that there was a synergism between novocaine, magnesium sulphate, and morphine, increasing the analgesic and anesthetic potency of the solution, without increasing the toxicity, and that novocaine allays any irritation from the use of magnesium sulphate. The nausea and vomiting following the previous use of morphine alone is said not to occur.

King gives two or three intramuscular injections, at one-half hour intervals, of 2 c.c. of sterile 50 per cent. solution of magnesium sulphate combined with  $2\frac{1}{2}$  per cent. novocaine and  $\frac{1}{8}$  grain morphine sulphate as a preliminary medication to local anesthesia, three hours before operation.

This preliminary he has used in 100 tonsillectomies in which 0.5 per cent. solution of novocaine was infiltrated around the tonsils. In about 20 other cases, only the preliminary intramuscular injections were given. In 6 cases only one tonsil received local injection after preliminary intramuscular injections.

The analgesia produced by the three intramuscular injections alone was sufficient and satisfactory in only about one-third of the selected cases, one of them a woman of seventy.

He believes that the preliminary injections and the local infiltration will be found to be successful.

He states that the average adult will require three intramuscular injections. No food should be permitted for at least six hours prior to the operation. Three hours before the operation is to take place injection into the thigh, deltoid or subscapular region of 2 c.c. of sterile 50 per cent. solution of magnesium sulphate with novocaine and morphine in quantities stated is administered. The site of injection is thoroughly cleaned and painted with iodine and alcohol; needle and syringe must be sterile. The patient must be quiet, and the intramuscular injections repeated until two or three doses have been given as may be required by the individual patient. The patient at the end of three hours from the first injection should be carried to the operating room. He should not walk after the first injection. The patient must be carefully observed according to King's statement and considerable judgment exercised in regard to the dosage for different persons. Idiosyncrasies as to morphine must be recognized and dosage regulated accordingly. Toxic symptoms from the morphine should be promptly combated.

The writer of this article has no experience with this method, and counsels reserve in the general use of King's method until it has been subjected to independent observations. He would also call attention to the danger of the tendency to increase the dose originally proposed. He would further state that the amount required cannot be judged by any nurse or house officer, and this is even difficult for the operator. It would, therefore, be necessary for the operator to be present three hours before operation to control the necessary dosage and watch its effect. As the effect of morphine is increased by the magnesium sulphate, the single dose of  $\frac{1}{8}$  grain of morphine is none too small.

Experience has shown that drugs which affect the system so powerfully

as to blunt all sensibilities are not of permanent value preliminary to local anesthesia, and sooner or later all methods involving their use in this way will have been discarded.

**The Previous Administration of Morphine.**—The use of a hypnotic of this kind before the use of a general or local anesthetic is never intended as an analgesic. It is given solely as a nerve sedative, making the patient less acutely sensitive to his surroundings and inducing cheerfulness, calmness, and content. It is of value too in that its sedative action prevents distressing nausea after ether and affords relaxation and rest if quiet is maintained.

**The Combination of Local and General Anesthesia.**—It is not the custom in America to combine the two, although it is apparently the custom in Great Britain.<sup>2</sup> The general feeling here is that if two operations are required they should be done separately. In addition to this we have called attention to the experiments of Macht<sup>3</sup> who found that the lethal and toxic doses of local anesthetics are very much smaller during general anesthesia than in non-anesthetized animals. He says: "One could not help drawing the deduction from these experiments, that to add a general anesthetic on top of a local anesthetic would be a very dangerous procedure and this, of course, is pharmacologically plausible on account of the depressing effect on the medulla and heart or both."

#### LOCAL ANESTHESIA

It was not until nearly forty years had passed in which general anesthesia held sway (ether in 1846, chloroform in 1847) that Karl Koller introduced cocaine (1884) as a local anesthetic in affections of the eye. Its use became general for all manner of operations.

The use of local anesthetics was, however, limited and remained so until the use of the suprarenal extract was advocated by Bates,<sup>4</sup> so that to the painlessness of the cocaine, there was added the bloodlessness from the suprarenal extract. This extract had to be prepared freshly each day by making a decoction, as it did not keep and was a culture-bearing medium. Search for the active principle of the suprarenal glands was then actively conducted by chemists and in 1900, Takamine in his New York laboratory succeeded in isolating needle-shaped crystals which he believed to be the active principle of the adrenals. He called this preparation adrenaline and brought it to the writer in the form of a solution 1 : 1000 for clinical investigation. The results were published,<sup>5</sup> this being the introduction of adrenaline to the medical profession.

Experiments made by the writer<sup>6</sup> showed that tissues were blanched instantly, the solutions could retain their activity for a protracted time, did not decompose and that the 1 : 1000 strength often produced a lymph-exudate soon after its use. A solution of 1 : 5000 or even 1 : 10,000 was found to be sufficient for all general purposes. In any event operations can now be performed bloodlessly. This gave an enormous impetus to the use of local anesthetics. Loss of consciousness, nausea, and pulmonary complications could now be avoided as well as those instances of nervous affection following the use of general anesthetics where it often required years for patients to recover.

**Synthetic Preparations.**—It was soon noted that toxic effects followed the use of cocaine and that it was a habit forming drug, and new prepara-

tions of a synthetic character were offered with the statement that the new preparations had all the good effects of cocaine and were either non-toxic or much less toxic than cocaine. These statements were often disproved on investigation. None has thus far equalled cocaine in every particular, and some are many times more toxic. These preparations had some temporary popularity and enthusiastic adherents. These devotees became silent when their lauded preparations failed to stand the test of time, but they did not retract their previous endorsement.

The local anesthetics now in common use aside from cocaine are: Procaine (an unfortunate name, as it may be mistaken for cocaine) or novocaine, apothesine, butyn, and benzyl benzoate. Alypin, quinine-urea, holocaine, and eucaine are now very rarely used.

Pick and Meyer, replying to the query as to the advances in local anesthesia,<sup>7</sup> state that they have been enabled to produce synthetic cocaine from well-known chemicals presenting a number of cocaines. Among these we have psicaine which is in every way a substitute for cocaine. It is much less poisonous, decomposes more rapidly, and produces surface anesthesia. It seems to them to be the cocaine of the future. There is another preparation called ekcaine, which, however, is not on the market. By combining other preparations with the synthetic we can increase the effect of the latter. Novocaine (procaine) is principally used because it contracts the blood-vessels.

The effect of the novocaine may be increased by adding an alkaline solution. Epinephrine has the same effect. Anemia follows with the resultant increase of the anesthesia.<sup>8</sup> Reference is made to the disagreeable effect after lumbar anesthesia. In these cases headaches occur and these have disappeared after intravenous injection of a physiological salt solution.

**Dosage.**—The dosages of all these drugs have been either those suggested by the manufacturers or those proposed by the teachers originally, and we have ascertained that it has been quite the custom for the individual operator to change the instructions given, frequently increasing the dose, and some fatalities have been apparently due to disregarding the conservative recommendations concerning dosage.

Under the auspices of the Therapeutic Research Committee of the Council on Pharmacy and Chemistry of the American Medical Association, a committee was appointed to investigate and report on the advantages and disadvantages of local anesthesia.<sup>9, 10</sup> After a careful study of this subject during the past ten years, certain definite recommendations as to dosage as well as other suggestions tending toward the prevention of toxic effects have been presented. It is believed that adherence to these recommendations will lessen toxic effects and prevent most of the fatalities. These recommendations are endorsed by the writer as chairman of the committee, and form the basis of this article.

**Toxic Effects.**—These range from faintness to loss of consciousness, long lasting numbness and tingling at the site of the application, to nausea, vomiting, hysterical manifestations, and death. As a general rule the toxic effects of the milder sort disappear after rest of a day or two in the hospital.

The fatalities that occur in the human being bear a remarkable similarity to those occurring in animals and also to each other, so much so that the Committee refused to accept the statement that a local anesthetic

caused death unless the symptoms were similar to those now known as classical, and accepted by all American pharmacologists.

**Fatalities.**—A fatality due to a local anesthetic is one where the symptoms of poisoning occur within a few minutes of the application of the drug, and life is pronounced extinct within three hours of its use.

No single local anesthetic is absolutely non-toxic. Fatalities may occur from any of them, but the danger of such accidents is remarkably small in some of them when we consider the millions of times they are used and the infrequency of fatalities after their application. No fatality should be ascribed to a single local anesthetic where another local anesthetic had been used at the same operation. (Thus, deaths have been ascribed to procaine, when cocaine had been used first. These should be called cocaine-procaine deaths.)

The injection of a local anesthetic is the most frequent cause of deaths. It has been repeatedly shown that deaths may follow the use of the local anesthetic when packed or merely applied to the nasal mucosa. Fatalities are occasioned by the substitution of one drug for another, by the use of a greater concentration than directed, by evaporation, too rapid injection, or entering a vein.

**Symptoms of Fatalities.**—Within a few minutes of the injection or the application of the anesthetic, the patient becomes suddenly pale, utters an exclamation of some serious apprehension, convulsions occur, accompanied by cessation of respiration and feeble pulse. Despite the hasty application of restoratives, unconsciousness remains, further convulsions appear, the pulse is no longer felt and life is pronounced extinct within a short time.

In a study of nearly 100 deaths following the use of a local anesthetic, 95 per cent. had these classical symptoms. A very few lived a day or two after the operation, and the Committee concluded that these did not constitute a true anesthesia death and that some other cause existed in those instances of delayed death. The striking similarity of the symptoms in fatalities to each other, as well as to those occurring in animal experimentation, made a profound impression on the Committee, and it was unanimously concluded from this study that no local anesthetic should be held responsible for any death occurring a day or more after its use.

**Necropsies.**—The protocols of a number of necropsies made have been carefully studied by competent men, in which no pathological condition was found that might show susceptibility or be a causal factor in those fatalities. A large number have been diagnosed as "status lymphaticus," without any evidence that such conditions existed.

**Causal Factors.**—The greatest single cause of fatalities was from the substitution of cocaine for procaine; next the use of a far greater concentration than prescribed. (In one instance 10 per cent. solution of cocaine was made up and injected where 1/10 per cent. was ordered.) One death followed the use of a solution that had been standing for some time, had evaporated, and become concentrated.

Pollack<sup>11</sup> records an instance where death followed a novocaine injection. Fortunately the fluid remaining was subjected to chemical analysis and found to have become far too concentrated for safe use. Without this chemical analysis a death from a small percentage of novocaine would have been recorded.

**Cocaine Paste.**—In all rules of therapeutics, it is stated that only such quantities of drugs should be given as will produce the required effect.

Cocaine in small doses produces local anesthesia. A small but influential percentage of fellow specialists use a paste made of cocaine crystals and adrenaline for nose operations; practically 100 per cent. on the theory that the larger dose produces such profound anesthesia and ischemia that absorption cannot occur. If this theory is correct, there should be no fatality following the use of this concentration. We know, however, of four deaths following the use of this concentration. One of these, carefully studied by the writer, in which he had the full written report as well as the protocol of the autopsy and repeated interviews with the physician in whose office the accident occurred, is instructive and refutes absolutely the theory of safety so relied upon. With this careful study, endorsed by all American pharmacologists, the writer feels that those using this concentration are in duty bound, at least, to call the attention of their pupils to the risk they are running in using this "paste."

The history is here briefly recorded<sup>9</sup>: Reported to Dr. Norris, 1923. Female, aet. 52. For puncture of antrum of Highmore. At the office of the physician, cocaine-epinephrine paste applied. The nose-trocar plunged into right antrum and irrigated.

Three days later, again at the office of the same physician, a 4 per cent. solution of cocaine (total amount unknown) was applied to the left side of the nose. Cocaine-epinephrine paste applied to the same side, after twenty minutes trocar introduced into the left antrum; no pus found. Stertorous breathing at once; frothing at mouth; pulseless; heart stopped before respiration, and death occurred within a few minutes. Necropsy showed acute pulmonary congestion and edema, suppurative frontal sinusitis. There could be no doubt that the death was occasioned by cocaine and that the theory of immunity from this powerful concentration is untenable. (The physician—a careful, conscientious observer—offered every facility for investigation.)

*Prevention of Accidents.*—The concentration in which these drugs may be used with a reasonable degree of safety is stated in connection with the paragraphs relating to them. Safety lies in conservatism and *not* in increasing the concentrations recommended. Cocaine solutions should be colored. The possibility of substitution should be borne in mind and rigid rules laid down for its prevention. The recumbent position during the entire procedure is recommended. If an accident occurs, the remainder of the preparation used should be sealed and submitted for chemical examination. The reporting either to medical journals or to a properly constituted committee, of all accidents in detail is strongly urged as being the means of preventing similar accidents.

*Treatment of Accidents.*—Efforts should be made to sustain the heart by artificial respiration, so that the drug may reach the liver where it is destroyed. Intracardiac injections of a solution of epinephrine and methods used to sustain the heart such as the injection of strophanthin and digitalis should be employed; morphine, so frequently used as an antidote, is of no avail and, in one instance, the symptoms indicated that death was due to the morphine so used. Ether has not been found of value.

Tatum,<sup>8</sup> Atkinson, and Collins have suggested the use of barbital as a preventive of cocaine toxicosis. They conclude that decerebrate animals are more tolerant to cocaine than normal animals, and it seems probable that the brain poisoned by cocaine influences unfavorably the respiratory

center. Cocainized animals which are allowed to pass into a state of clonic convulsions do not recover as readily as those whose convulsions have been prevented by the administration of barbital and paraldehyde. The higher the type of animal, the lower is the lethal dose of cocaine. Leshure<sup>12</sup> concludes that barbituric acid compounds are safe and efficient antidotes to cocaine, especially the sodium barbital compounds. The drug should be given one-half hour before using cocaine. Cocaine should never be injected in any amount under any circumstances. The amount of cocaine should not exceed  $\frac{5}{6}$  of a grain. A sterile solution of sodium barbital should be on hand for intravenous use, if required. C. B. Williams<sup>13</sup> warmly endorses this method.

**Cocaine.**—Until the present time, no local anesthetic has equaled cocaine in that it paralyzes reflexes, contracts blood-vessels, thus permitting free examination even where there is intolerance or much induration; it renders the parts insensitive to pain when applied and has been of the utmost value so used in the removal of foreign bodies. Were it not for its toxicity and habit forming tendency, it would be the ideal drug. All other local anesthetics fail to produce all the effects of cocaine or else require an exceedingly long time in doing so. By this effect of cocaine, laryngoscopy can be successfully accomplished in most intolerant cases. Rhinoscopy is made possible without the delay of waiting for the intumescence to subside.

*Manner of Application.*—In the form of solution, by instillation, application, or injection. The application is made by dipping cotton-covered applicators into the solution and applying these freely to the part. A limited percentage of the solution may be sprayed over the part instead of this form of application. The amounts so used in both are indefinite; some of the solution clings to the cotton. It has, therefore, been recommended that in each of the latter concentrations the solution be measured carefully beforehand and noted, so that the quantity remaining will indicate at least that no more than a given quantity could have been used.

*Instillation and Injection.*—Instillation is rarely used in the nose and throat. Injection in varying strengths has been practised in operations on the nose and throat. After careful study of reported fatalities the injection of cocaine was condemned as was also the use of cocaine paste.

*Strength of Solution.*—This varies from  $\frac{1}{2}$  of 1 per cent. to 2 per cent. Cocaine is sufficient to paralyze reflexes and reduce congestion at examinations, and anesthetizes the parts sufficiently to render painless the introduction of the hypodermic needle prior to the use of local anesthesia of the synthetic variety. It may also be used prior to stronger solutions as a preliminary in endolaryngeal operations to paralyze reflexes. The larynx has been shown to be more tolerant than other parts when applications of greater strength are used so far as absorption is concerned; hence to produce complete anesthesia within the larynx, as in the removal of growths, etc., solutions as high as 20 per cent. may be used.

*Frequency of Deaths.*—Cocaine is much more frequently used than any of the local anesthetics except procaine, and the large number of deaths from the former must not be considered in comparing with deaths from other drugs rarely used, as alypin, etc., without reference to the infrequency of the use of the other drug. Comparisons have been made in animal experimentation notably by Hatcher and Eggleston<sup>14</sup> and the relative frequency of deaths noted.

**Procaine (Novocaine).**—This synthetic chemical, the hydrochloride of procaine, para-aminobenzoylethylamino-ethanol, has stood the test of time, is the most frequently used of all local anesthetics now in common use, and the claim that it is far less toxic than cocaine has been amply substantiated. It is safe to say that this drug is used millions of times in this country yearly and the number of deaths that can be truthfully stated to be due to it is infinitesimal. Time and again cases of deaths alleged to be due to procaine are upon investigation proved to be due to other causes. In most of these, there was a preliminary application of cocaine. This was disclosed on closer investigation. Such deaths must be classed as due to cocaine-procaine.

While deaths may occur from procaine alone, it is the general consensus of opinion that the slow injection of a solution not over 2 per cent. in strength is practically safe. While procaine induces anesthesia when applied, it is a very slow process.

The addition of sulphate of potassium to the saline solution of novocaine has a synergistic action in increasing the depth and duration of the anesthesia.

Prinz<sup>15</sup> has made use of this combination for more than four years with excellent results in the Evans Dental Institute of the University of Pennsylvania. His experience has been unusually large in that he has used over a pound of novocaine per year; he has prepared the solutions and has been enabled to watch the results, averaging 15,000 injections per year for the past five years. He prepares a standard solution as follows:

Sodium chloride C. P., 7 grams  
Potassium sulphate C. P., 4 grams  
Distilled water, 1000 c.c.

When needed,

400 c.c. standard solvent  
6 grams novocaine

are slowly boiled in an Ehrlenmeyer flask, stoppered with cotton for four to five minutes. When cool, the solution is filtered through paper (using a freshly flamed glass funnel) in a sterile glass-stoppered bottle. This quantity is made up in accordance with needs.

“When needed to every 10 c.c. of this novocaine solution (which is a 1½ per cent. solution) 4 to 5 drops of epinephrine solution should be added, mixed but *not boiled*. It is now ready for immediate use. The standard solution with novocaine should not be kept over a month; aërial infection occurs after that time. Both novocaine solution and the epinephrine solution must be alkali free. Alkali free glassware for this purpose may be readily prepared.”

There may be substituted for this, the use of ampules containing a solution of novocaine in proper strength with the synthetic preparation of epinephrine (Metz). These must not be boiled.

**Butyn.**—It is claimed for this preparation that it has all the properties of cocaine in that it induces anesthesia when applied locally. It has been used rather frequently among ophthalmologists. Four deaths in all have been recorded<sup>16</sup> following its use, two of them after packing the nose with a 5 per cent. solution of butyn. It is but fair to state that reported fatalities show that the solution was used in much greater concentration than was recommended by the manufacturers of butyn. If injected, not over 2 c.c.

of a  $\frac{1}{2}$  per cent. solution should be used and, if applied, in not over 2 per cent. solution. The other two were confidentially reported to the writer.<sup>17</sup>

**Apothesine.**—Four deaths following the use of this drug were reported to the Committee on local anesthesia. It has also been stated that sloughing frequently followed its use.<sup>18</sup> It should not be used in greater concentration than 2 per cent. and not more than 1.5 grains.

**Alypin.**—It is most rarely used; it is apparently as toxic as cocaine, if not more so.

**Quinine-urea.**—Seems to have been abandoned as far as the nose and throat are concerned because of the great amount of sloughing following its use.

**Epinephrine.**—The preparation which the writer has found to be constantly reliable is adrenaline, as also the synthetic preparation. Adrenaline, when injected, should be carefully watched and noted.

Prinz, loc. cit., states that not more than 5 minims of the 1 : 1000 solution of epinephrine should be used as a maximum quantity. One drop of this solution is added to 2 c.c. of the standard novocaine solution and only when needed. The epinephrine solution is to be kept in a dark cool place. Any solution containing epinephrine should not be boiled. Boiling for less than five seconds destroys this alkaloid.

We have already stated that further studies of epinephrine are needed. Fatalities have been recorded following its use, and indications of its use in large quantities and concentrations are not wanting. Attention is also called to the statement that intracardiac injections of epinephrine in cases of toxicity should enter the cardiac muscle, and that a long needle is required for this purpose owing to retraction of the cardia. The dose of epinephrine solution so administered should not be excessive.

**Conclusions.**—Local anesthesia has supplanted the use of general anesthesia in operations on the nose and throat. It has been clearly demonstrated that fatalities do occur more frequently than was believed, that many are avoidable, that the time of onset and the nature of the symptoms are always the same, so that we now know definitely what constitutes a death from these drugs. Accidents have occurred in the practice of men of exceptional ability and it is no reflection on their skill to have had such misfortune in the past, because of our imperfect knowledge of the dangers attending the use of local anesthetics and precautions whereby they can be avoided. The treatment of symptoms has been given. Bearing in mind the need for caution, we present the following:

**Precautionary Measures.**—Every step should be taken to prevent substitution. Sealed ampules, colored solutions, and a checking system dividing responsibility. Solutions that have been prepared a long time are not to be used, unless in ampule form.

Careful measurements made of quantities used. The amount of epinephrine should be carefully watched and noted. The concentrations that are considered safe as herein stated should be adhered to; they are the result of careful deliberation by a thoroughly competent committee of physicians selected from various fields of medicine, and are their unbiased and independent opinions.\*

\* The Therapeutic Research Committee of the Council on Pharmacy and Chemistry of the American Medical Association says of these recommendations, "This does not question the right of the surgeon to depart from these concentrations, but he may see that in exceeding them he is treading on dangerous ground."

The use of two local anesthetics are best avoided. If an accident occurs both drugs should be held responsible, not one. The fluid remaining should be placed under seal and subjected to analysis. All accidents should be reported either in medical journals or to the permanent Committee.

EMIL MAYER.

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### AN IMPROVED TECHNIC IN THE OPERATION FOR CLEFT-PALATE

The following is not a discussion of the whole subject of cleft-palate, but a brief description of the technic devised and employed by the author for the closure of the cleft in the hard and soft palate. All procedures (such as plastic bone work on infants) leading up to the final operation are omitted.

The text-book classifications of cleft-palate and the methods employed by others in this field are also not mentioned. My purpose is to present my own conclusions drawn from about 250 operations and to describe a method which, after much experimentation, I have finally evolved and which in the hands of others and in my own have enormously increased the percentage of cures.

To more intelligently present this operation I shall lead up to it by a brief summary of the mechanical principles involved.

The adverse forces present in this surgical field are so many and so constant that one wonders why failure is not the rule. These forces are septic infection and traumatism.

**Septic Infection.**—We aim to secure primary union in a septic field. This might be called a surgical paradox. It took us a long time to realize that given a septic field we could easily turn the scale against us by traumatism, inflicted either by the surgeon during the operation or by the patient during the healing period.

**Traumatism.**—Under this heading eight sub-classifications appear: (1) Injury to the tissues inflicted by rough handling during the operation. (2) Injury to the palatine arteries which are the chief sources of blood-supply to the flaps. (3) Tongue pressure, present at all times and particularly during the act of deglutition. (4) The eccentric pull on the stitches during the act of deglutition. (5) Constant tension on the line of union due to failure on the part of the surgeon to bring the flaps into easy apposition. (6) Careless asepsis. The realization that a patient may conquer the infection he carries but may succumb to an added one of the same family dawned slowly upon the surgeons. "Why be clean in a septic field?" is fortunately not heard now as often as formerly. (7) Contact of the stomach contents with the unclosed wound may ruin the best done operation. (8) The effect of seasonal conditions on infections. I long ago discovered that the percentage of healing was cut in two during the winter months and during the very hot months of summer. Hence I do all my cleft-palate work in April, May, June, September, October, and November. The technic followed in the operation is the logical application of the above principles.



Fig. 365.—Right-angle knife making incision through mucous membrane and periosteum along the edge of the cleft, from the posterior end of the hard palate on one side, around the anterior angle to the same point on the opposite side.

As the operation even in the most expert hands is not a short one, depression from excessive anesthesia should be carefully avoided. Many failures may be traced to this cause since it acts not only immediately as a tissue depressant but remotely in stopping food absorption. (A serious condition in children.) The strictest asepsis is observed. The face and the entire mouth are painted with a 2 per cent. iodine, and a 2 per cent. acriviolet is dropped into the nose. Gloves and masks are worn by all assistants and by the anesthetist.

The patient's head rests on the operator's knees over the head of the table. This position gives absolute control of the head and affords the best possible view of the operative field. One assistant stands on each side. Suction is used to take the place of sponging as it causes much less traumatism. Sponges are used for compression and for wiping the edges of the flaps before tying the stitches.

**Technic.**—Using a right-angle knife, an incision is made along the edge of the cleft on both sides. Brophy's elevators are used to lift the soft tissues, including the periosteum, away from the bone of the palatal process. This separation proceeds from the center outward toward the alveolar process. When no teeth are present the flaps may be lifted away to a point level with the apex of the alveolar ridge. In older cases, where teeth have erupted, too close an approach to the teeth may seriously damage the blood-supply. The posterior palatine artery must not be injured. The periosteal reflection around the posterior end of the palatal process is cut outward, thus allowing

the palate to drop into a horizontal position. The posterior pillars must sometimes be nicked to make this flattening of the palate complete. This is essentially the Langenbeck method (Warren-Ferguson-Langenbeck).

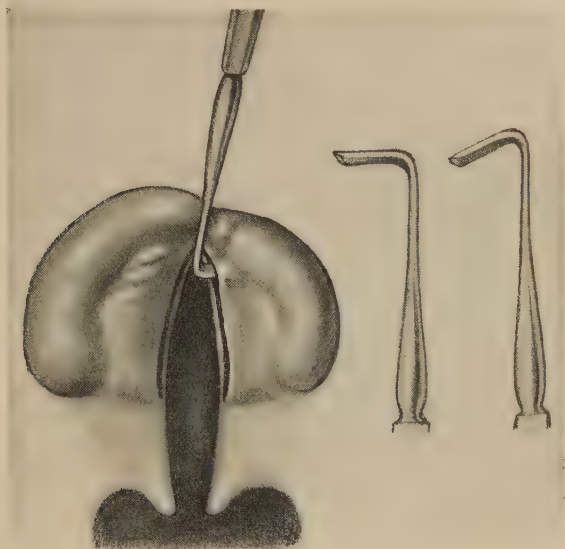


Fig. 366.—Elevators of different angles lifting the periosteum and mucous membrane away from the bone. This separation is carried outward to the alveolar process when teeth are present, and up to the apex of the process when teeth are absent.

The edges of the soft palate are denuded making a raw edge as wide as possible with the removal of the least amount of tissue. Many methods of

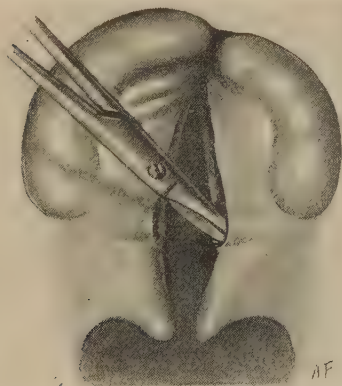


Fig. 367.—Incising the periosteum where it is reflected around the posterior end of the hard palate onto the floor of the nasal cavity. This is carried outward to the wall of the nasopharynx.



Fig. 368.—Soft palate dropped into horizontal position. Where teeth are absent a further drop and elongation of flaps may be obtained by carrying the separation to the top of the alveolar process.

placing the stitches are advocated. I prefer the straight through-and-through stitch of fine silkworm gut for the soft palate and the on-end mattress-stitch of Equisetene for the hard palate. This mattress-stitch

everts the edges of the thin flaps over the hard palate and makes union more probable by increasing the coaptated surfaces. At this point my method diverges from the others.

In 1914 I published a paper on this subject giving the finer points on technic. Here the question of tension was discussed in detail. Out of

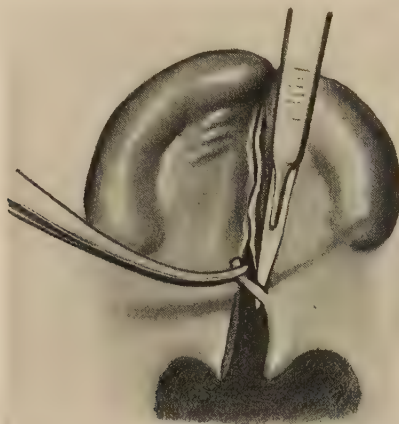


Fig. 369.—Denudation of edges of the soft palate. The knife points inward toward median line in order to widen the denuded area.

the experience gleaned from over 100 operations, done prior to that time, two principles emerged. One dealt with the relief of tongue pressure and the other with lateral flap tension. The following is quoted: "Tongue pressure as a factor in producing strain has, so far as I am aware, been over-

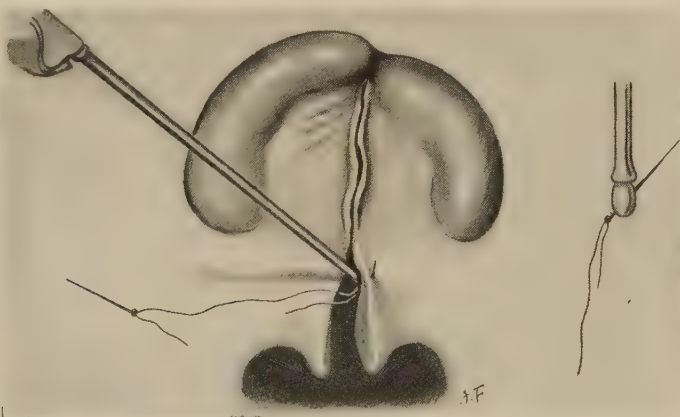


Fig. 370.—Method of using straight needle. It is hooked from above downward through the tissues, taking a liberal bite.

looked. That it is a potent cause of failure needs no proof when we consider the amount of upward force exerted by the tongue in each act of swallowing, and that this act takes place every few minutes more or less unconsciously, and quite independently of taking food."

For several years I have been using a device for the relief of this strain

and now feel justified, on account of the good service it has rendered me, in presenting it for trial by others. A plaster cast of the palate is taken and on this cast is made an obturator which fits accurately along the inner margin of the gingival ridge in infants and along the base of the teeth in older

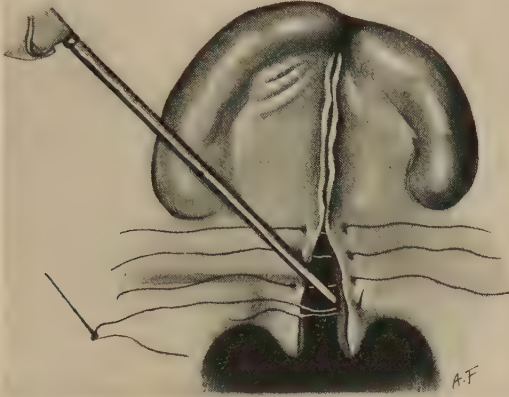


Fig. 371.—Four stitches in place. The last one must be well forward of the point where the soft palate curves on to the uvula in order not to strangle the uvula. The bite of this stitch should be a little closer than the others to the raw edge for the same reason.

children. The obturator is made of palatinoid wire, heavy at the rim and light inside. The wires are cross-barred and soldered at the points of crossing. After the completion of the operation the obturator is sewed to the gums of infants and tied to the teeth in older children. Three points

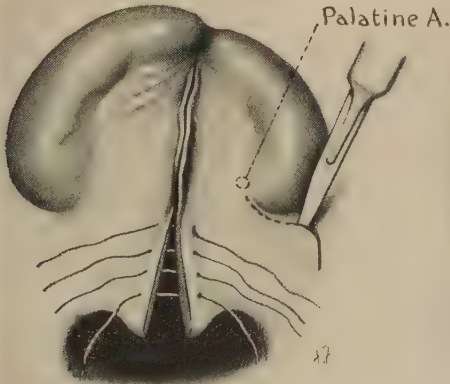


Fig. 372.—Method of making posterior lateral incision. Note that the knife enters the tissues with a distinct slant toward the center of the nasopharynx and at the palate buccal angle. The length of incision depends on amount of tension present.

of attachment are sufficient—two well back on the sides and one in front. “The obturator is left in until the stitches are removed. It in no way obscures inspection of the wound or interferes with cleanliness. I use it in all cases where the cleft extends beyond the soft palate. It should be made flat

or curved slightly upward, and should not project far enough back to impinge against the downward curve of the soft palate."

At that same period (1914) I was endeavoring to relieve lateral flap tension by inserting small hook retractors into the lateral incisions. The following

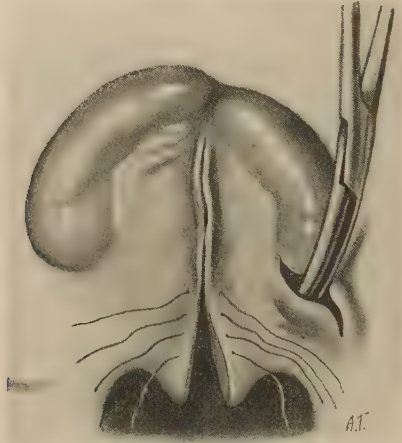


Fig. 373.—Stretching the tissues of palate toward the center line.

is extracted from the above mentioned paper: "Then I devised flat-faced small retractors of silver. These can be made any width desired. The arms are about equal in length, the object being to secure a good grasp on the inner edges of the lateral incisions. The arm on the plate of the buccal

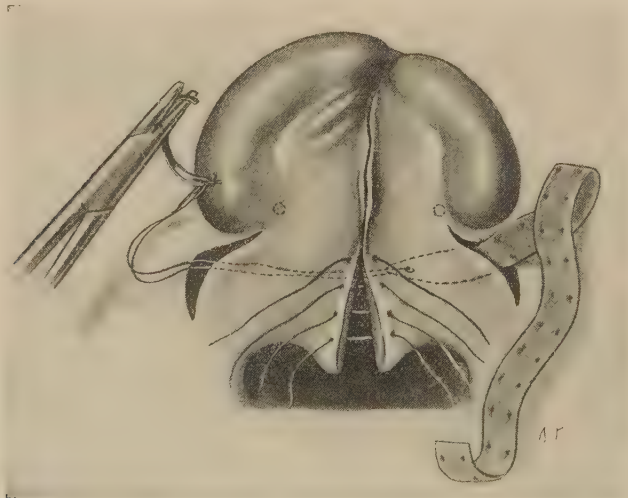


Fig. 374.—Method of conducting the band through the nasopharynx.

side has an eye in its end to which a strong linen thread is secured. The threads of the two retractors are tied and drawn together sufficiently to take all tension off the central line. The retractor can be made for each case by the surgeon from virgin silver plate rigid enough to maintain its

shape when bent. It should be of sufficient width to insure it against cutting into the edge of the lateral incision."

The obturator devised eighteen years ago, excepting in refinement of construction, has been retained in its original form and used ever since in all cases where the cleft extends anterior to the hard palate. The hook

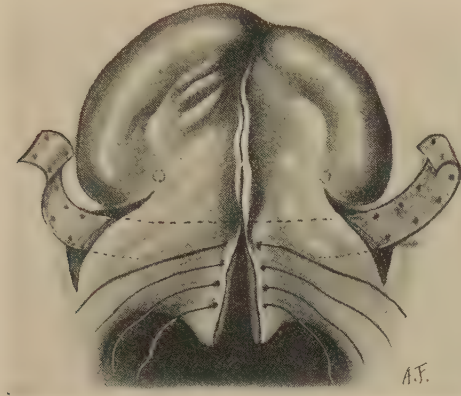


Fig. 375.—Band in position.

retractors were not entirely satisfactory and were replaced by the lead- or silver-band now used. This consists of a strip of lead or silver slightly less than 1 cm. wide and 15 cm. long. One end of this band is cut to a point. At this point a small hole is punched for the attachment of a stout

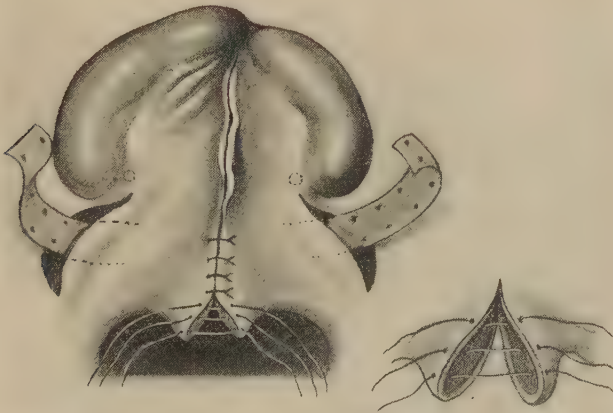


Fig. 376.—Tying of stitches and closing of uvula. Note that stitches in uvula pass only through the mucous membrane.

thread. Two rows of holes,  $\frac{1}{2}$  cm. apart in cross section and the same in longitudinal section, are punched along the extent of the middle third of the band. A thread carrying a large curved needle is tied through the hole in the pointed end of the band.

This band has been used to relieve tension in two situations, one in

front of the emergence of the palatine arteries and the other behind the posterior ends of the alveolar processes. When the stitches are all in place the gap between the flap edges can be estimated. Then the lateral incisions which are to carry the band (or bands if two are used) are made. It is evi-

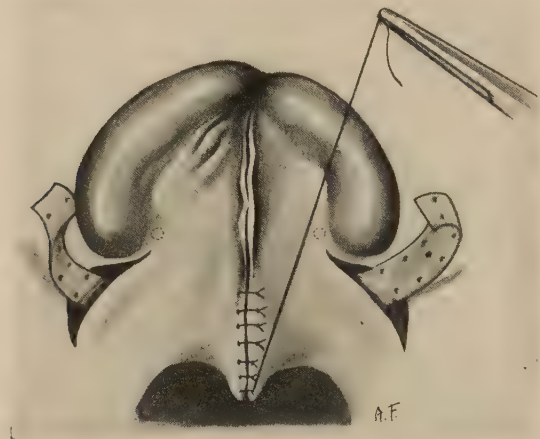


Fig. 377.—Last stitch in uvula retained as a tenaculum to evert the palate and expose the upper or posterior surface.

dent that the farther these incisions are from the line of union the better for the circulation along these edges. At a point on both sides just behind the alveolar process and well up against where the buccal surface curves into the soft palate a knife is plunged through the tissues to emerge in the

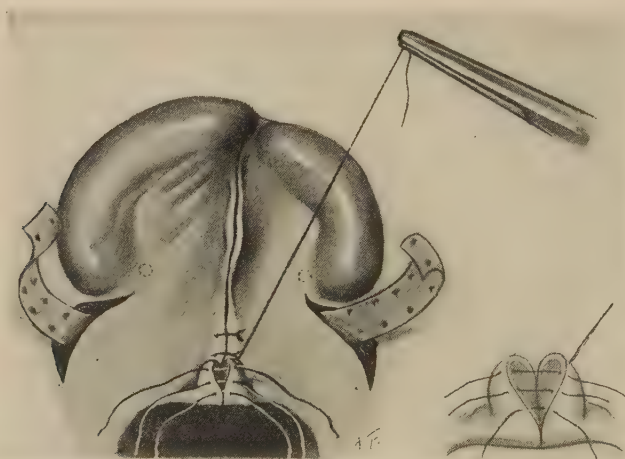


Fig. 378.—Stitching the posterior surface of the uvula and soft palate. Note that only the mucous membrane is included in the bite.

nasopharynx. The line of perforation is directed slightly to the center line. Through this hole an artery clamp is passed and gently opened, the object being to stretch the tissues. Slowly and gently the palate is worked over toward the center. It is sometimes necessary to carry an incision along

the posterior end of the alveolar process. This incision must never curve forward around this end since at this point is found the palatine artery. The same procedure is carried out on both sides until the flaps meet with ease at the center line. The curved needle holding the band is now backed, eye first, through the stretched incision, carried across the nasopharynx and

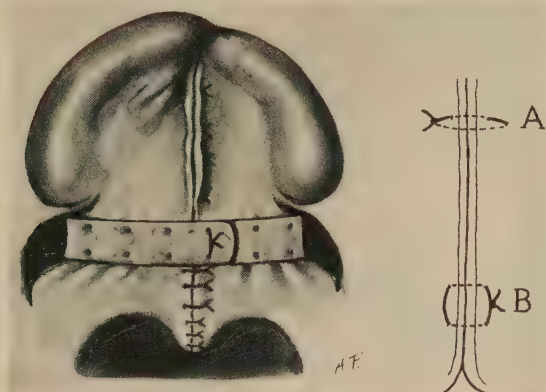


Fig. 379.—Tissues of hard palate united by mattress-stitch. *A*, Correct position of stitch. *B*, Incorrect position of stitch. It is obvious that *A* strangulates less tissue than *B*.

brought out on the buccal surface through the corresponding incision on the opposite side. The thread then leads the band through in the same way. The stitches are now tied in the soft palate. More stretching of the tissues toward the center line may be required as each stitch is tied to secure coaptation without tension. The importance of complete relaxation can-

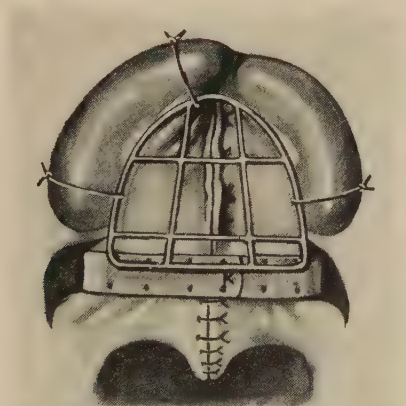


Fig. 380.—Operation completed. Tongue-pressure obturator in position. Band crossed and tied with just enough pull to cause slight puckering of tissues of soft palate.

not be too strongly stressed. In some cases the lateral incisions gape so wide on either side that the nasopharynx can be seen through the openings. If the center unites, these large lateral holes always close promptly. The band is now drawn across until all central tension is overcome and the palate lies flaccid at the line of union. It is then secured by tying through

the holes in the band which come opposite each other. If required a similar band may be placed so as to relieve tension in the hard palate region. Here the lateral incisions are made as far out as possible and the tissues stretched to the center as before described.

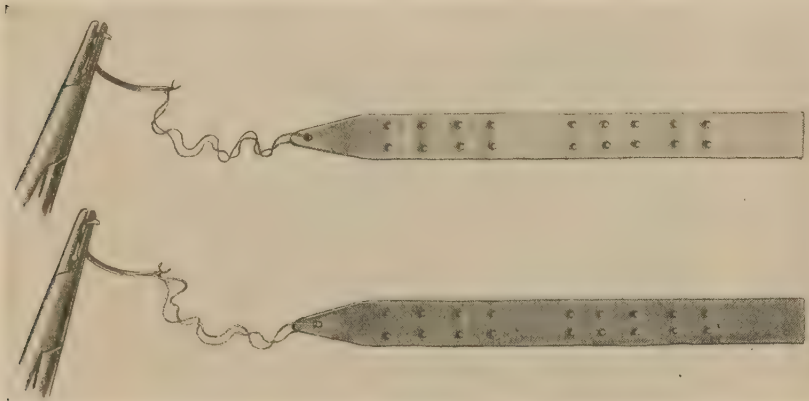


Fig. 381.—Lead and silver bands. Lead is preferable.

These bands and the obturator are removed with the stitches eight or nine days later. This should always be done under general anesthesia, to obviate injury to the palate from struggling.

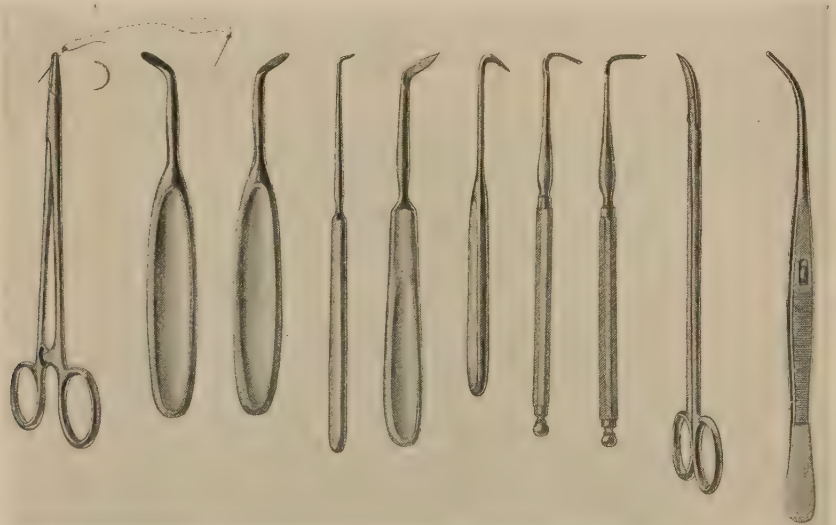


Fig. 382.—Instruments employed. The essential ones are the needles and holder, angular knife, and Brophy's elevators.

Full feeding is begun as soon as possible. The mouth is irrigated after each feeding and a few drops of a 2 per cent. acriviolet solution dropped over the line of stitches and into both sides of the nose.

This method puts the palate completely at rest during the healing period. Tongue pressure is entirely obviated. The muscular contractions

of the soft palate during crying and deglutition exert their force against the band and not against the line of union.

Our percentage of primary healing has been advanced at least 20 per cent. over those reported by me in 1914. The union in uncomplicated cases has been over 95 per cent.

JOHN EDMUND MACKENTY.

## PART IV—DISEASES OF THE LARYNX

### ANATOMY OF THE LARYNX

The anatomy of the larynx is best understood and remembered more easily if we start our study at the bottom and advance upward. This is particularly so with regard to memorizing the various muscular and ligamentous elements. We find they have all been named from their origin which is inferior to their insertion, usually in a superior locality. We will therefore take up the trachea first.



Fig. 383.—Anterior view of the larynx showing the ligamentous attachments to each other and superiorly the hyoid bone.

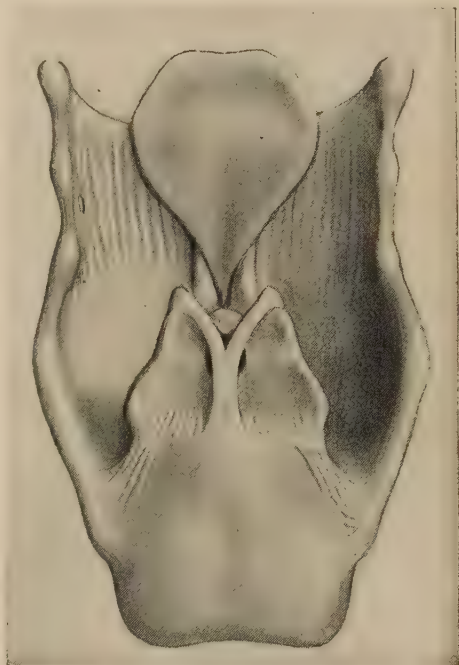


Fig. 384.—Posterior internal view of cartilages with their ligamentous attachments.

**Trachea.**—The trachea varies in length, averaging from 11 to 15 cm. in the adult, and is composed of a varying number of cartilaginous rings. These rings are not complete, but form approximately a two-third circle, the open space being situated posteriorly. The cartilaginous members are bound together at certain intervals by two structures—a membrane of connective tissue and a band of muscular fibers. We can visualize, therefore, that by this arrangement the trachea is kept on a stretch and open, as well as giving it a certain degree of elasticity and mobility. A mucous membrane with innumerable cilia, all of which wave upward, forms the lining of this tube.

**Cricoid Cartilage.**—Situated on top and acting as a means to keep the trachea open at this point, and also as the base of the larynx, we have a

cartilage which is best described as resembling a signet-ring, the wider part posteriorly on which we find two facets for the insertion and bed of the cricoid posticus. The smaller part lies in front. The lower margin or inferior edge lies in almost the same plane when the head and throat are in the upright position. The upper or superior margin of the posterior portion gradually rises from before backward. It is considerably above the narrow part, and is called the arch. The height of the cartilage at this point averages 15 mm., while the anterior portion is approximately 5 mm.

Near the junction with the posterior portion upon either side we have an elliptical articulating facet for the inferior cornua of the thyroid cartilage.

**Thyroid Cartilage.**—A large cartilage of hyaline structure, consisting of two symmetrical vertical plates having a superior and inferior elongation or horn on either side. The plates are joined anteriorly, forming an angle. Above and below their junction the two plates are notched—the superior and inferior thyroid notches. The posterior portions of the thyroid plates

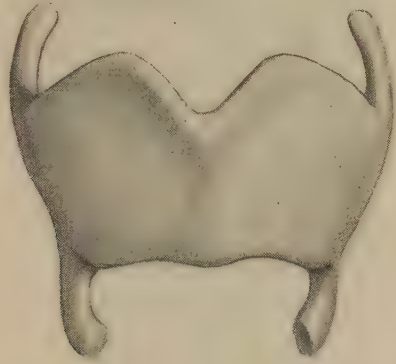
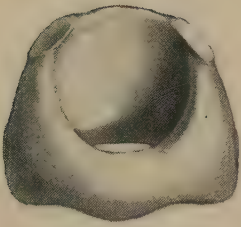


Fig. 385.—The cricoid cartilage, seen from before.

Fig. 386.—The thyroid cartilage, seen from before.

are widely separated from each other, the entire cricoid cartilage being interposed between their lower margins. The inferior horns or cornua present a slightly flattened angular articulation which sets into a corresponding articulating surface on the lateral and inferior surface of the cricoid cartilage. The thyroid cartilage, shaped in the manner of the ancient breast plate, acts in part as a shield for the delicate mechanism within the larynx. The superior horns are joined to the hyoid bone by muscular and ligamentous bindings. Situated in the median line and on the inner surface we find a small fibrous projection which serves for the insertion of the vocal cords. From this surface we also have attached the petiolus of the epiglottis. It can thus be readily seen that while attached to the cricoid cartilage below, the hyoid bone above, nevertheless the thyroid cartilage is distinctly movable forward and backward. The articulation of the lesser horns with the cricoid acts as the fulcrum; this is essentially necessary for the proper and complete approximation of the vocal cords.

**Epiglottis.**—The epiglottis is rhomboid in shape, a more flexible cartilage than any of the others, and is situated as the upper one of this series, the larger

part or more expanded portion being above. It is entirely covered with mucosa with the exception of the anterior surface, which is separated from the throat by the hyothyroid membrane. Within the mucous membrane there are numerous glands, secretory in nature. The sensory element is highly developed upon the crest and laryngeal surface, and well may we look upon the epiglottis as the watchman of the larynx.

**The Arytenoids.**—These twin cartilages, similar in form to a three-sided pyramid, articulate posteriorly by a concave inferior surface or base with the convex crest of the cricoid cartilage. A prolongation of roughened bone, named the vocal process, extends anteriorly and serves as an attaching surface for the vocal muscle and elastic fibers of the vocal cord. Posteriorly at the base there is a slight protuberance, the muscular process, which serves as an insertion for the respiratory muscles—the posterior crico-arytenoideus. This protuberance is also used for the insertion of the phonatory muscles—the lateral crico-arytenoideus and thyro-arytenoideus. The posterior surface of the arytenoids is concave and serves for the insertion of the third of the



Fig. 387.—Cartilage of the epiglottis, seen from behind.



Fig. 388.—The arytenoid cartilage, seen from the outer side.

phonatory or constrictor muscles, known as the transverse arytenoid. Having its origin from the sides of the arytenoid cartilages and with its insertion along the lateral border of the epiglottis, there is a wide, thin muscular band, the aryepiglottic folds, which form the tube-like upper portion of the larynx. Between these folds and the inner surface of the thyroid cartilage we have the pyriform sinuses.

**Cartilages of Santorini and Wrisberg.**—Four in number, pyramidal in form, small in size, and situated on the apices of the arytenoids, presenting the appearance of a part of that cartilage. On the superior and anterior margin of the aryepiglottic folds we find the flattened cartilages of Wrisberg.

The thyroid, cricoid, and arytenoid, except the vocal processes, are composed of hyaline cartilage. The epiglottis, cartilages of Santorini, Wrisberg, and vocal processes of the arytenoid consist of elastic cartilage.

**Ligaments of the Larynx.**—*Conus Elasticus.*—A ligament consisting of connective tissue interspersed with elastic fibers arising from the crest of the cricoid, the external surfaces of the vocal processes, and the anterior edges of the arytenoids. This ligament is inserted to the inferior edge of

the thyroid cartilage and thus fills the space between the thyroid cartilage and the cricoid. Some of these fibers are inserted to the inner surface of the thyroid cartilage. The superior fibers extend horizontally from the vocal processes to the junction of the plates of the thyroid cartilage.

The vocal cords which are the basis for the vocal bands are formed by the superior fibers of this ligament. We can thus visualize and see that the conus elasticus may be likened to a pyramid, the apices above thus forming the rim of the glottis.

The ventricular ligaments composed of less elastic fibers running parallel to the vocal ligaments arise from the inner surface of the thyroid cartilage at its anterior angle, and are inserted to the anterior surface of the arytenoid cartilage, superior to the vocal ligament.



Fig. 389.—Anterior half of a coronally divided larynx.



Fig. 390.—Lateral view of larynx showing attachment to the hyoid bone and articulating fossa of thyroid cartilage on lateral wall of cricoid.

The epiglottis by an elastic band is attached by its petiole to the thyroid cartilage on its inner surface at the convexity, by an elastic band about 1 cm. in length. Superiorly, this cartilage by ligamentous bands is attached to the hyoid bone, tongue, and the arytenoids. The lingual epiglottic attachments are one median and two lateral folds.

Binding the thyroid cartilage to the cricoid anteriorly we have the middle cricothyroid ligament. Surrounding the inferior cornua of the thyroid with its junction with the cricoid cartilage there is a thin lax ligament, the cricothyroid capsule. This capsule is assisted or reinforced by the lateral and posterior cerato-cricoid ligaments, these acting as a partial check to the movement. The arytenoids are supported and held in position at their articulation with the cricoid by the crico-arytenoid capsule, and these are supported posteriorly by the posterior crico-arytenoid ligament.

The sinuses of Morgagni are two triangular pouches with their apices pointing upward and lie on either side of the larynx, between the true and false cords, constituting the first of the series of the resonators of the voice.

As the larynx acts in a double capacity, that of phonation and respiration, we necessarily must have two antagonizing or opposing sets of muscles: those which, when stimulated by the nerve impulse, contract and by their contracting open the glottis to its full extent; and those whose action, when stimulated, will bring the vocal cords and the vocal membrane as well to the median line, thus closing the glottis and approximating the cords; these are termed the constrictors of the glottis or phonatory muscles.

The muscles under the phonatory group are the two crico-arytenoideus lateralis, the two thyroid arytenoideus and the arytenoideus posticus or transversalis. The respiratory group consists of the two crico-arytenoideus posticus muscles. (See section on Consideration of the Physiology of the Laryngeal Musculature.)

Muscles acting as supporting elements by attachment to neighboring structures, functioning during phonation and respiration as well as during the act of swallowing, are the sternohyoid, sternothyroid, thyrohyoid, stylopharyngeus, and the inferior constrictors of the pharynx.

**Nerves of the Larynx.**—The cricothyroid is supplied by the superior laryngeal nerve. All the remaining muscles are innervated by the inferior laryngeal nerve.

ROBERT F. RIDPATH.

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## MIRROR LARYNGOSCOPY

Mirror laryngoscopy makes use of the first and simplest principle of optics, viz., that in the reflection of a ray of light, the angle of incidence is equal to the angle of reflection.

Before we take up the means by which the mirror is used, let us consider a few details which may be essential to the elicitation of information which we hope to obtain.

Having the patient sitting in an upright position, a suitable light on the patient's right side, the head-mirror is so adjusted that the reflected light is directed in the mouth and the examiner's eye looking through the central aperture (Fig. 391).

The mouth is then carefully examined before the introduction of instruments, and the condition of the teeth, gums, cheeks, tongue, and lips noted. Ranula or any tumefactions on the floor of the mouth may be observed and may be a cause of dysphagia or inability to protrude the tongue.

The tongue should be examined and its characteristics as well as any pathological conditions considered. By a partial protrusion the taste-bulbs may be seen, and their size, position, or diseased condition considered.

The general shape of the mouth, height of arch, characteristic of dentition, fauces, tonsils or result of tonsillectomies seen. The pharyngeal wall, length and position of uvula, and the width and capacity of the epipharyngeal floor cognized.

We are now in a position both by the knowledge gained and by the confidence of the patient to look further and complete our laryngoscopic examination.

Again asking the patient to open the mouth and protrude the tongue, we, using a linen cloth or handkerchief, grasp the tip of the tongue with the thumb and middle finger of the left hand. This permits the index-finger to steady the head by making pressure on the upper teeth. Too much traction must not be made, and care taken that damage is not done to the tongue by sharp points or caries of the lower teeth. While in this position the patient is taught how to breathe, as it is impossible to make a successful examination unless the patient breathes quietly and regularly. We therefore tell him, while holding the tongue, to breathe through the mouth in a normal easy way, as to cease breathing regularly will cause gagging, retching,

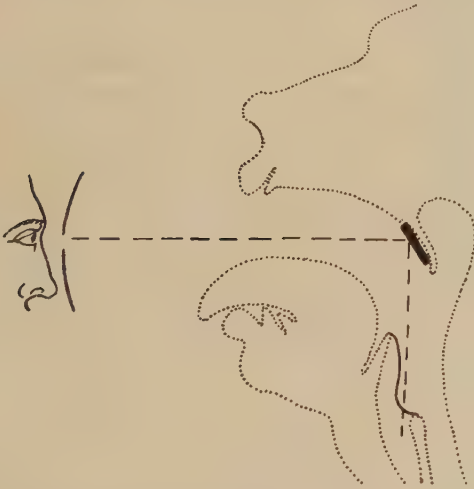


Fig. 391.—Laryngoscopy makes use of the elementary principle of optics, viz., the angle of incidence is equal to the angle of reflection. (After Laurens.)

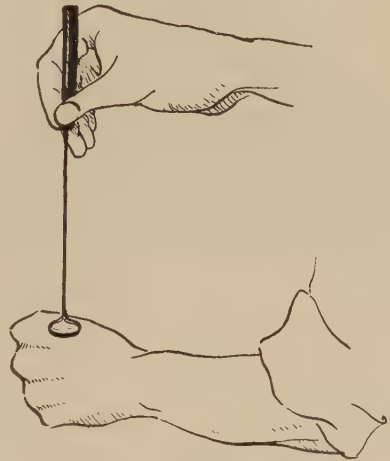


Fig. 392.—Testing the warmth of the mirror before introducing it into the mouth of the patient. (After Laurens.)

and cough. It is best to have him say "ah," "eh," or "k" at this time, to demonstrate the ease with which these sounds may be made while the tongue is held. Educating the patient to breathe regularly, instead of spasmodically, is the first advice that must be given and carried out.

As we have already obtained a view of the mesopharynx, our selection of the proper sized mirror is decided. It is desirable to use as large a mirror as the conditions of the case will permit. The larger diameter of the mirror, the greater illumination of the larynx, as well as a larger view is obtained. One having a transverse diameter of 1 inch is best, as fewer movements which may cause reflex and consequential retching will have to be made in obtaining the view. Smaller sizes may be used as the peculiarity of the cases warrant. The best view is obtained by the patient leaning slightly forward with the head thrown a little back.

The laryngeal mirror is warmed either in hot water, a spirit lamp, or by coming into contact with the buccal membrane, the glass side to the membrane, to a proper temperature to prevent moisture gathering and conse-

quent blurring of the image. If heated over the lamp or by immersion in hot water its temperature is first tested on the back of the left hand (Fig. 392).

The mirror is then introduced and is held lightly, passed well back over the dorsum of the tongue. Care must be used that the tongue is not touched, in consequence of which a spasm of coughing or gagging may result. When the uvula has been reached the mirror is then raised, pushing the uvula

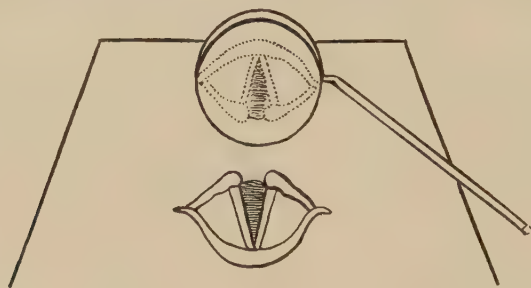


Fig. 393.—Reflection in mirror of drawn diagram of larynx. (After Laurens.)

and soft palate upward and backward, asking the patient to repeat the words "eh," "ah," or "k" when a good view of the larynx is usually obtained. The laryngoscopic image as appearing in the mirror is inverted or in reality only half inverted (Fig. 393) since, in the mirror which is held at an angle of 45 degrees to the plane of the aperture of the larynx, the anterior parts appear above and the posterior parts below. Should a figure appear on a flat surface or drawn on paper, then the image is completely inverted,

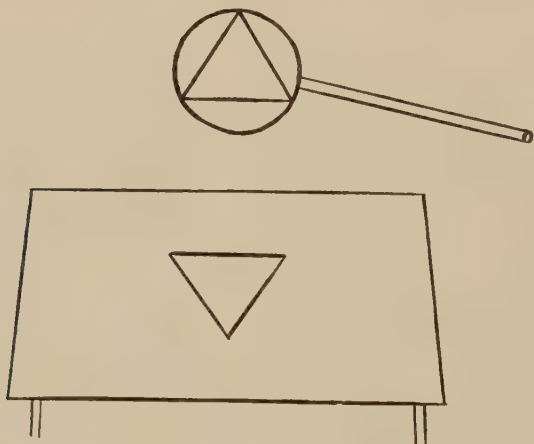


Fig. 394.—There is no reversal of the image. (After Laurens.)

the back corresponding to the front and vice versa (Fig. 394). "The right cord remains on the right side (patient's right) of the mirror. It is well to remember that the apex of the A-shaped image is anterior. The alliteration helps the memory" (Chevalier Jackson).

Should the introduction of the mirror excite or cause gagging, an application or spray of  $\frac{1}{2}$  or 1 per cent. cocaine solution to the uvula, pharyngeal wall, or base of the tongue will be advantageous to a satisfactory view.

Sipping cold water or sucking ice is also useful. Timidity in the introduction of the mirror is frequently more disastrous than firmness.

The first laryngeal object seen in the mirror is the tip of the epiglottis. We then identify the ventricular bands, the prominence of the arytenoids, and finally the vocal cords.

**Difficulties Encountered.**—In some instances the peculiar shape of the epiglottis is a source of difficulty. Occasionally it drops over the rima glottis and prevents the view of the cords. The "Omega" shaped epiglottis is also a source of difficult examination due to the lateral compression, but may be overcome in the adult by thorough cocaineizing of the crest, and the application of a curved tenaculum with gentle upward and forward traction. A long-tongued spatula with a curved end to pass over the epiglottis is occasionally needed.

In children, the left index-finger makes a very good tongue-depressor and is less terrifying than an instrument.

Fear of an examination or fancied pain or discomfort is another difficulty which must be considered. Persuasion and reassurance without impatience on your part will in the majority of cases overcome this. Many of this class of patients open the mouth insufficiently when at last we are permitted to proceed with the examination, or may protrude the lips as in the act of whistling so that a proper view is impossible. Again perseverance is the remedy, requesting the patient to protrude the tongue and close the eyes, practised with frequent satisfactory results.

The posterior part of the tongue may be sufficiently contracted or raised as to completely hide the view; should this be the case a tongue-depressor may be used advantageously, while the tongue is protruded and held forward by the patient using his left hand. In certain cases the tongue-depressor may be useful without the protrusion of the tongue being necessary. The raising of the tongue and the resultant contracted mass are usually due to the patient opening his mouth too wide.

Do not pull the tongue forward too forcibly; injuries or cutting of the frenum may result. The more the tongue is dragged down in front, the greater will be the bulging or arch behind.

The mirror must not be allowed to remain in the mouth too long, but removed, and, after a short wait, it may be introduced without discomfort.

Elongated uvula may be met with but, if reflected in the mirror, change the position by slight rotation or pressing more upward and backward.

**Demonstration of the Laryngeal Image.**—It is often desirable to demonstrate to others the mirror view of the larynx in certain patients. Looking



Fig. 395.—Method of using the laryngeal demonstrascope showing the laryngologist examining the larynx of the patient and one observer to the right of the laryngologist, the reader being the second observer on the laryngologist's left, showing what the observer sees in the mirror of the demonstrascope. A, Demonstrascope mirror. B, Pilling electric head-lamp. C, Screw for raising and lowering mirrors. E, Diaphragm of Pilling lamp. G, Head-band.

over the shoulder of the physician making the examination is not very satisfactory and is possible for only one observer at a time. The *teleloupe* of Molinie is used in France and is good. We use at the Bronchoscopic Clinic with utmost satisfaction the demonstrascope of Robert M. Lukens, of which Dr. Lukens has kindly contributed the following description:

"Clinical teaching and demonstration of disease of the larynx are greatly facilitated by the use of the laryngeal demonstrascope (Fig. 395). Two students or observers, one on each side of the laryngologist, can see the larynx at the same time. The instrument is an adaption of the old-fashioned window busybody and is used in the same way, *i. e.*, the observer looks into one of the mirrors at right angles to a line drawn between the laryngeal mirror and the demonstrascope. The lamp is operated on house current, controlled by a rheostat. The lamp is so arranged that any object illuminated by it is reflected in each mirror." (A manikin, using this demonstrascope for teaching purposes, has also been devised by Dr. Lukens. It is excellent.—EDITOR.)

ROBERT F. RIDPATH.

### DIRECT LARYNGOSCOPY

**Definition.**—Examination of the larynx through the mouth with the aid of a speculum that displaces the tissues that otherwise would obstruct a

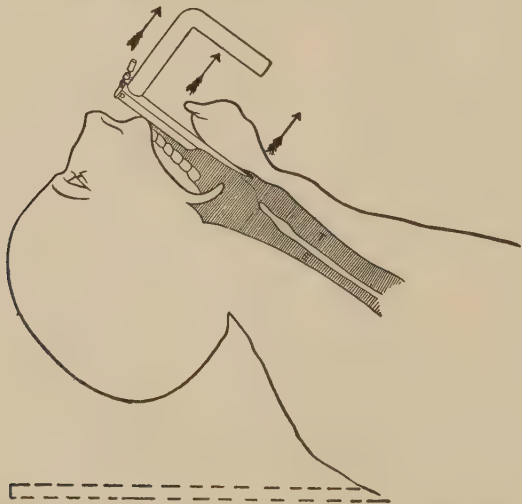


Fig. 396.—Schema illustrating the technic of direct laryngoscopy on the recumbent patient. The motion is imparted to the tip of the laryngoscope as if to lift the patient by his hyoid bone. The portion of the table indicated by the dotted line may be dropped or not, but the back of the head must never go lower than here shown, for direct laryngoscopy; and it is better to have it at least 10 cm. above the level of the table. (Note that in bronchoscopy and esophagoscopy the head section of the table *must* be dropped, so as to leave the head and neck of the patient out in the air, supported by the second assistant.)

direct view into the larynx (Fig. 396). This procedure is called "direct" in contradistinction to the indirect method by which we look not directly

at the larynx, but at a reflected image of the larynx. The appearances are quite different by the two methods (Fig. 397).

**Purposes.**—Broadly expressed, direct laryngoscopy is used for the diagnosis and treatment of laryngeal disease. It has incalculable advantages in the diagnosis and removal of foreign bodies and growths—benign and malignant; the removal of specimens of secretion and tissue for diagnosis; the introduction of intubation and anesthetizing tubes; the treatment of laryngeal stenosis; the determination of the mobility of the crico-arytenoid joints, and many other procedures. It will never supersede the mirror for diagnostic inspection of the larynx in adults and older children, but is

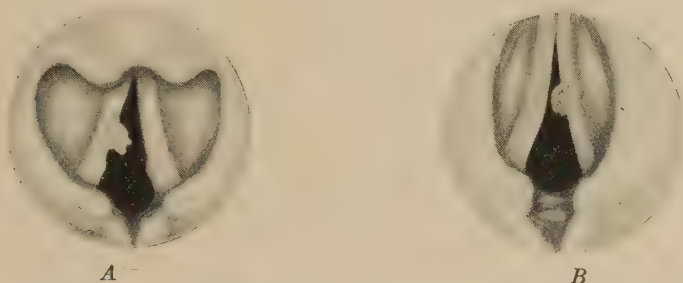


Fig. 397.—Schematic illustration contrasting the views obtained by direct and indirect laryngoscopy, respectively. At *A* we have the indirect view, in which we look not directly at the larynx, but at an image of the larynx reflected in the laryngeal mirror. The image is reversed anteroposteriorly by the mirror, but is not reversed laterally. The patient is opposite to us, however, which posture puts his right side to our left. A small growth is represented on the right cord. The normal cords look white in life, but are actually pink; they look thin, but are actually thick; the larynx looks shallow but is actually deep. At *B* is represented the direct laryngoscopic view of the same patient, in the recumbent posture. The epiglottis is lifted out of sight with the lip of the laryngoscope. The cords are not so conspicuous, and show their normal thickness, color, and depth. The patient being dorsally recumbent with his feet away from us, we have the right cord to our right. The growth on the right cord is now seen on our right. The simplification of operative or other procedure by the direct method is obvious. (Pencil sketches by Chevalier Jackson.)

often required for inspection supplementary to the mirror examination. The latter always precedes the direct examination. In the examination of the larynx of children under four or five years of age, it is the only method available, since the mirror cannot be used at this age. The larynx of any human being who can open his mouth can be exposed to direct view, provided the essentials of position and manipulation have been mastered. Exposure of the larynx to view is the first step in bronchoscopy.

The further consideration of direct laryngoscopy, its technic and general usefulness, will be found in the section on Peroral Endoscopy.

CHEVALIER JACKSON AND CHEVALIER L. JACKSON.

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## HYPOPHARYNGOSCOPY

This means the objective examination of the *hypopharynx*, which is the region of the pharynx from the arytenoid level down to the cricopharyngeal fold. This region is not visible in the laryngeal mirror; its anterior wall is the posterior wall of the larynx; it is exposed to trauma in swallowing; it is often involved in cancer, syphilis, tuberculosis, and perichondritis of the larynx—all of these facts render hypopharyngoscopy a procedure of utmost importance to the laryngologist and his patient. Hypopharyngoscopy is called for in every case of edema of the arytenoid eminences not due to obvious intralaryngeal lesions. The hypopharynx is easily exposed to view, with the laryngoscope in the hands of anyone who has acquired the knack. The position of the patient is dorsal recumbency, and the technic is the same as that of direct laryngoscopy (Fig. 396). After the larynx is exposed, the distal end of the laryngoscope is inserted posterior to the arytenoids and the cricoid cartilage. A strong lifting motion is exerted in the direction of the three darts. As in exposure of the larynx, the motion must be one of lifting, and not one of prying on the upper teeth as a fulcrum. The opening of the hypopharynx is accomplished by lifting the larynx away from the cervical spine. The procedure is not painful, but the reflexes are active in a muscular subject. A full dose of morphine hypodermically two hours beforehand, and cocaine locally just before the insertion of the laryngoscope, are advantageous. The cocaine solution should be dropped into the pharynx of the recumbent patient with an ordinary medicine-dropper. The solution should be swallowed, which involves no toxic risks in a normal adult if a total of not more than 1 grain of cocaine (gram 0.06) be used. Cocaine should not be used in children under about ten years of age; it is not only dangerous in children, but is quite unnecessary.

CHEVALIER JACKSON

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## SKETCHING THE LARYNGEAL IMAGE

Accurate records are important for the patient's best interests as well as for the accumulation of scientific data. An accurate description takes much space as well as much time in recording and also in referential use afterward. A sketch, however crude, is quickly made and later is instantly comprehended. Many laryngologists use a diagram printed or stamped on the record card. Such a diagram may serve for recording the mere location of an ulcer or growth; but is useless for recording the appearance of the larynx. A sketch is quickly made, and is a graphic record. It is idle to say one cannot draw; anyone can learn to draw if he has the incentive. Everyone has learned to draw the entire Roman alphabet in his childhood. It is easier to learn to draw the outline of the glottis than it was for each of us, in childhood, to learn to draw the letter A which the outline of the normal glottis so much resembles. Every post-graduate student should devote a few minutes daily to sketching the larynx.

**Materials.**—A good soft lead pencil, such as Dixon's Eldorado No. 4B, is best. A pink, a red, a yellow, and a blue pencil are useful for tinting lesions and contrasting normal mucosa.

**Sketching the Laryngeal Image.**—No attempt should be made to take in the whole larynx at once. Four separate steps are required. The first thing to record is the shape of the epiglottis. This varies widely in health and still more widely in disease. A few frequently encountered types are shown elsewhere herein. No attempt should be made to draw any other part until the epiglottis is drawn. Then the shape of the glottis is noted and put down in black. When the black silhouette of the glottis is placed it is easy to place the glottic borders whatever they may be—cords, fungations, tumors, or lesions as the case may be. The final step is the insertion of the aryepiglottic folds. If each step is completed by itself all is quite simple. To attempt to see and draw the whole larynx at once is bewildering. I have never yet had an assistant or a student who could not learn to draw the larynx by this simple method if he was willing to make the effort.

After the outlines are placed, a little shading may be added where the shadows fall; and lesions may be added with colored pencils if desired.

**Sketching the Bronchoscopic Image.**—What has been said above about sketching the larynx applies in a general way to sketching the bronchoscopic image. The habit of fixing in the memory a series of the panoramic endoscopic views is quickly developed if concentrated upon to the exclusion of other matters for a time. The best way to acquire this habit is to practise catching first the shape of the lumen, which is the main "dark" of the image. This is done by creating a scabbard-shaped lumen on the rubber-tube manikin or any bit of rubber tubing, by external finger-and-thumb pinching just ahead of the bronchoscope. This lumen is drawn inside a scribed circle, noting the long axis of the scabbard and changing the image by the external pinching in other axes successively. Having acquired the knack of getting the lumen image in mind and on paper, all else will be easy. Two branching lumina slightly separated will form the carina or other spur between. The important thing is to put in the darks; the lights will take care of themselves. These two values are enough to show the essentials of any image; but after the knack of doing this has been mastered it is easy to add the half-tones where they come between the high lights and the darks.

**Sketching the Esophageal Image.**—This is much the same in principle. The drawing of folds is simplified by putting down only the darks, that is, the creases between the folds. Copying the normal esophageal image as shown in the first illustration (1) in Plate XXIV, will show the way this is done. In clinical work, the habit of quickly fixing in mind the lumen and the form and direction of the dark streaks between folds is invaluable in accuracy of observation and of recording.

Further suggestions and information on this subject will be found elsewhere.<sup>1, 2</sup>

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## PHOTOGRAPHY OF THE LARYNX

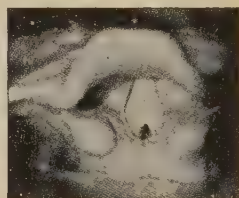
**Introductory.**—Photography in medicine is gradually increasing its sphere of usefulness as an adjunct in teaching as well as in providing accurate records. Many improvements have been made in apparatus and in furnishing adequate sources of light, so that now one may study from photographic images various organs and structures which are so situated in the body that direct or indirect inspection is difficult.

It is now possible to photograph the larynx with comparative ease and without assistance. After a proper technic is acquired, excellent photographs can be made of the living larynx in quite a large percentage of cases. Such a procedure is of great technical usefulness. It gives a more nearly correct and lasting impression of the appearances of the larynx than is possible by drawings or word pictures.

**Apparatus.**—French (1884) originated a small single lens camera with throat mirror attached. Sunlight was utilized as the source of illumination. Later he devised a method to use the arc light instead of sunlight. He secured results which were simply marvelous when one considers the



A



B

Fig. 398.—A, Monoscopic image of normal larynx in respiration. B, Monoscopic image of paralysis of the arytenoid muscle. (French.)

state of photography at that time. The monoscopic images obtained have never been improved upon (Fig. 398).

The *teleloupe* of Molinie has been used, and the results are excellent.

Garel (1899–1919) perfected a stereoscopic camera which has given brilliant results. It is of the utmost usefulness in general clinical work.

**Uses and Results.**—Photography furnishes a record of a permanent character. Unlike drawings made of the larynx, these images are exact and can be used in comparative studies. With photographs these studies can be carried on at one's leisure. Accurate observations can be made of the position and appearance of the vocal cords during respiratory and phonatory movements. The location, extent, and appearance of certain pathological conditions can be definitely recorded.

Some of the results obtained by the Garel camera are shown in Figs. 399 to 401. When viewing these images stereoscopically, a remarkable perspective is obtained which far excels that obtained by indirect laryngoscopy with the ordinary laryngeal mirror.

**Technic.**—To obtain good results one should possess at least a rudimentary knowledge of photography as well as a thorough working knowledge of the camera used. Briefly stated, the Garel camera (Fig. 402) consists of a small rectangular stereoscopic chamber (A) fitted above with a curtain shutter having a lock and release attachment (B), and so constructed at the

bottom (*C*) as to receive either the ground-glass or plate-holders (*D*). Surmounting this is a small cubicle (*E*) which holds the two objectives and a prism which reflects the rays of light at a right angle. A prism meniscus has been added to the prism at a point midway between the two objectives. This neutralizes the prism and serves as a view finder (*H*). On the right is a hollow tube (*I*) which receives the shank of the laryngeal mirror. A small tungsten lamp inclosed in a metal telescopic cylinder (*K*) and fitted with a strong condensing lens is fastened to the right side with a ball-and-socket joint. The ordinary illumination of the tungsten lamp is insufficient

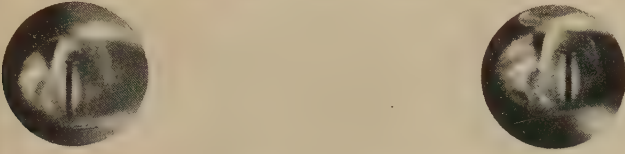


Fig. 399.—Stereoscopic image showing feeble laryngeal tensors.

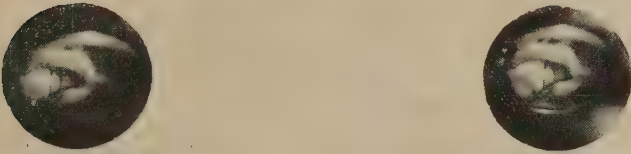


Fig. 400.—Bilateral vocal nodules. Larynx in phonatory position.

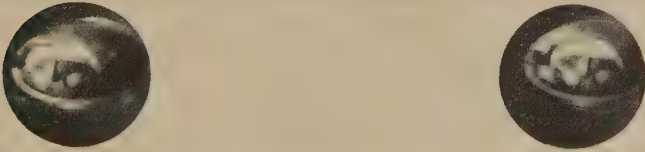


Fig. 401.—Stereoscopic image showing the larynx in a case of bilateral recurrent paralysis photographed during inspiration. (Figures 398 to 401 are reproductions of photographs of larynx.)

for photographing the larynx. To obtain sufficient light the lamp must be over-illuminated during the moment of exposure. To permit this a resistance coil (*L*) is used which is controlled by a foot-pedal (*M*). The speed of the shutter varies from 1/15 to about 1/60 of a second. It may be released by the left hand of the operator or by a special shutter release (*O*) which may be fastened to the foot-pedal and operated with the toe.

The greatest difficulties lie in focusing the laryngeal image on the ground-glass and in the illumination of the larynx.

**Focusing.**—The camera has a fixed focus which is approximately 23 cm. The depth of the average larynx is about 3 cm. It is, therefore, necessary to secure the handle of the laryngeal mirror in the tube so that the center

of the mirror is about 20 cm. distant from the prism. This distance must necessarily vary since the depth of different larynges varies. Garel advises marking the mirror handle at a point corresponding to the position for the average case and then making additional markings 1 mm. distant on each side of this. Exposures should then be made using the mirror in these positions. Checking the developed plates with the various positions will give the correct distance between mirror and camera. After considerable practice this distance can be estimated with fair accuracy. It is advisable to take at least four exposures of every larynx, varying the position of the mirror with each. To assist in focusing, a mirror can be obliquely suspended

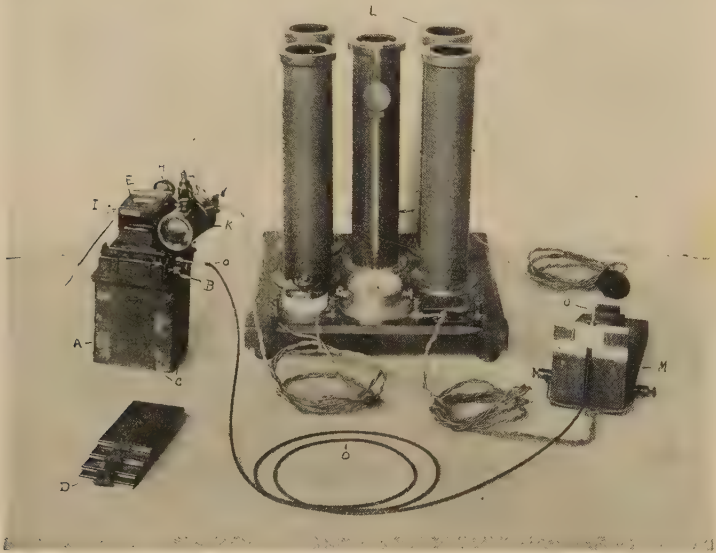


Fig. 402.—The perfected stereoscopic apparatus of Garel: *A*, The rectangular stereoscopic chamber; *B*, lock and release attachment which control the curtain shutter; *C*, slide which receives either the ground-glass or plate-holder; *D*, plate-holders numbered from one to four. These hold the plate which measures 4.5 by 6 cm. *E*, Cubicle which contains the two objectives and prisms and has extending backward a hollow cylinder (*H*) which serves as a view finder; *I*, hollow tube which supports the shank of the mirror; *K*, metal telescopic cylinder containing the lamp and condensing lens; *L*, resistance coil; *M*, foot-pedal; *O*, special shutter release which is controlled by the operator's foot. (Garel.)

beneath the ground-glass image so that it can be viewed either by the operator or by an assistant.

**Illumination of the Larynx.**—Condense the rays of light by moving the telescopic cylinder containing the condensing lens so that the circle of rays will fall slightly below and to the left of the center of the mirror. The mirror should be bent on its handle so that it will face a point midway between the lens aperture and the source of light. To secure an equal illumination of both sides of the larynx is most difficult, and requires considerable experimenting with the position of the mirror before one can ultimately attain success.

The resistance coil is intended to cut down the current sufficiently so that the lamp can be used with ordinary illumination during the preliminary focusing. At the moment of exposure the resistance is cut out

by closing the foot-switch when the lamp is momentarily over-illuminated. This period must be brief or the filament will burn out. When the resistance coil is connected with the 110 volt street-current the ammeter needle points between one and two. With the foot-switch closed and the regulator moved from above downward, the ammeter needle will slowly increase the readings on the dial. These should not be carried beyond 3 amperes. The coil is now ready for use.

**Adjustment of Shutter.**—The timing of the shutter is accomplished by a small set screw. The speed varies from 1/15 to about 1/60 of a second. It can be released either by the hand or by the foot of the operator at the same moment that the resistance coil is cut out with the foot-switch.

**Preparation of Negatives.**—The photographic plates used measure 4.5 by 6 cm. and should be of the greatest speed. Exposures of 1/20 to 1/40 of a second give good results. Developing and fixing can be done with the ordinary domestic developing and fixing solutions. Garel recommends a special formula which gives excellent results.

**Preparation of Prints or Positives.**—In order to view the positives with a stereoscope the images appearing on the negative must be transposed. They must also be separated from 62 to 66 mm. to correspond to the interpupillary distance. Glossy paper should be used for printing. The paper

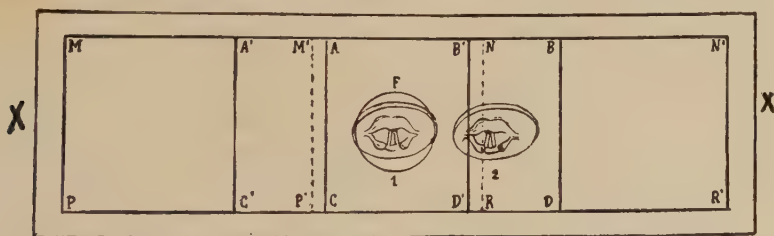


Fig. 403.—Schema illustrating the method of printing the two laryngeal images so that they will be transposed and also separated to correspond to the interpupillary distance.

should be cut in strips measuring 4.5 by 10 cm. which will fit the special printing frame. As shown in Fig. 403, the negative is placed, emulsion side up, in the printing frame (XX) with image No. 1 of A B C D in front of the opening F. Over this is placed a black paper printing mask which will block out everything but the desired laryngeal image. The printing paper is then placed in positions M N P R and the opening F exposed to the light for the time required for printing. The paper and mask then being raised, the negative is shifted to position A' B' C' D' so that image No. 2 appears before the opening, and the mask and paper are moved to position M' N' P' R'. The second image is then printed. Developing and fixing of the paper are best carried out according to the directions furnished by the manufacturers.

The view obtained with the stereoscope is not that which is seen by indirect examination; instead, it is the view obtained by direct laryngoscopy, the patient being dorsally recumbent.

To the beginner, photography of the larynx is not easy. To attain proficiency requires a considerable expenditure of time and energy. When once understood it is most fascinating. This, with the personal satisfaction of obtaining accurate images and the possibilities of adding to our knowl-

edge of the larynx, should be ample compensation for the time spent in developing the necessary technic.

LOUIS H. CLERF.

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#### HOARSENESS

**Definition and Synonyms.**—"Hoarseness" may be defined as a husky, rough, rasping, or croaking character in the sound of the voice. The voice lacks clearness and smoothness. The term is synonymous with "huskiness." "Croupiness" is sometimes used synonymously; but medical practitioners usually limit the use of the word "croupiness" to the peculiar hoarseness of cough (rather than of the speaking voice) such as is noted in subglottic laryngitis associated with laryngeal diphtheria, influenzal laryngitis, etc. Hoarseness is a symptom, not a disease; but it is a symptom that always calls for prompt investigation by every diagnostic means we possess. We should never be content with an inferential diagnosis. The diagnosis of acute or chronic laryngitis should never be made until every other possible cause of hoarseness has been excluded. Hoarseness may be classified as acute or chronic, but this distinction is uncertain and it is unwise to make it because of the intermittent character of many cases of hoarseness. It is very common for a patient to state he has been hoarse only a few days, yet close questioning will reveal the fact that an intermittent hoarseness has been mistakenly regarded as separate attacks of acute laryngitis.

Acute hoarseness should not be considered as necessarily an acute laryngitis. When a patient has become suddenly hoarse inspection of the larynx should be done at once—with the mirror in adults and older children, by the direct method in young children and infants. Only two of the many dangerous conditions whose possible presence in acute hoarseness calls urgently for prompt diagnosis need be mentioned; namely, laryngeal diphtheria in children and edema of the larynx in either adults or children.

Chronic hoarseness is so often mistakenly attributed to chronic laryngitis that cancer of the larynx has in many cases become hopelessly inoperable before it is discovered; tuberculosis or syphilis has often destroyed the cartilages and worked havoc with the other laryngeal structures in default of local examination and aggressive diagnostic methods.

**Etiology and Pathology.**—To understand the pathological mechanism of hoarseness we must remember that for clear phonation the vocal cords

must (1) approximate, (2) draw tense, and (3) vibrate. Anything that interferes with any or all of these components may cause the symptom, hoarseness. Approximation may be interfered with by (a) a tumor between the cords; (b) feebleness of muscular action; (c) paralysis; (d) fixation. Tension may be interfered with by (a) paralysis; (b) feebleness; (c) fixation. Vibration may be interfered with by (a) thickening of the cords—inflammatory, infiltrative, or neoplastic; (b) a damper-like action of a growth unassociated with thickening of the cord.

A categoric list, nearly complete, of the conditions that may be concerned in hoarseness is given below. In order to render the list of utmost usefulness no attempt is made to avoid overlapping.

- |   |   |   |
|---|---|---|
| 1. Malignant neoplasm.  | } | The lesion in these conditions may be local in the larynx, in the mediastinum, in the neck, or in the central nervous system. |
| 2. Benign neoplasm.   |   |   |
| 3. Tuberculosis.  |   |   |
| 4. Lupus.   |   |   |
| 5. Syphilis.  |   |   |
| 6. Hemorrhage.  |   |   |
| 7. Recurrent paralysis.   |   |   |
| 8. Angioneurotic edema.   |   |   |
| 9. Leprosy.   |   |   |
| 10. Scleroma.   |   |   |
| 11. Goiter—mediastinal, cervical, or endotracheal.                      |   |   |
| 12. Aneurysm.   |   |   |
| 13. Thickened pleura.   |   |   |
| 14. Tabes.  |   |   |
| 15. Disseminated sclerosis.   |   |   |
| 16. Glossolabiopharyngeal paralysis.                                    |   |   |
| 17. Syringomyelia.  |   |   |
| 18. Syndrome of Avellis.  |   |   |
| 19. Nephritis.  |   |   |
| 20. Prolapse of the ventricle.  |   |   |
| 21. Pachydermia.  |   |   |
| 22. Dislocation: (a) Thyrohyoid; (b) crico-arytenoid; (c) cricothyroid. |   |   |
| 23. Foreign body.   |   |   |
| 24. Laryngospasm, laryngomyopathy, feeble tensors, etc.                 |   |   |
| 25. Vocal nodules.  |   |   |
| 26. Perichondritis.   |   |   |
| 27. Crico-arytenoid arthritis.  |   |   |
| 28. Crico-arytenoid ankylosis.  |   |   |
| 29. Cicatrices.   |   |   |
| 30. Stenosis: (a) congenital; (b) acquired.                             |   |   |
| 31. Pericardial effusion.   |   |   |
| 32. Dilatation of left auricle.   |   |   |
| 33. Trauma.   |   |   |
| 34. Phonation with ventricular bands or other folds.                    |   |   |
| 35. Papilloma.  |   |   |
| 36. Foreign body.   |   |   |
| 37. Diphtheria—local, toxic, glandular pressure.                        |   |   |
| 38. Puberty.  |   |   |
| 39. Laryngismus stridulus.  |   |   |
| 40. Screamer's nodes.   |   |   |

41. Vocal abuse.
42. Feebleness of laryngeal musculature.
43. Influenza.
44. Acute laryngitis.
45. Chronic laryngitis—infiltrative, atrophic, subglottic, etc.
46. Glanders.
47. Actinomycosis.
48. Anthrax.
49. Acromegaly.
50. Hysteria.

Space does not permit a presentation here of the pathology of all these states. The laryngeal phases are covered in other sections of this book; the general phases are to be found in the medical literature.

**Diagnosis** as to the mere presence of hoarseness is made from the sound of the voice or from the statement of the patient or relatives as to the occurrence of a huskiness at other times. The diagnosis as to the lesion producing the hoarseness is of the utmost importance. It is well to take the list of diagnostic possibilities given in the foregoing paragraph and reach a diagnosis by exclusion. Many of the conditions can be promptly eliminated, and there is much overlapping. Therefore, the list can be quickly narrowed down to relatively few diagnostic possibilities. The most frequent diagnostic problem in adults with a laryngeal lesion is as to the presence of new growth (benign or malignant), syphilis, or tuberculosis. In dealing with this problem it is well to remember the following points:

- (a) A tuberculous patient may have syphilis.
- (b) A patient with malignant disease of the larynx may have pulmonary tuberculosis or a syphilitic lesion anywhere.
- (c) Any two or all three conditions may coexist in a mixed laryngeal lesion.
- (d) In considering duration, remember that a syphilitic lesion may be held stationary without being cured, and that a precancerous lesion may extend the duration of hoarseness for many years—as many as thirty years in some instances. When a patient with hoarseness is sent to the Bronchoscopic Clinic for diagnosis he is put through as many of the following diagnostic steps as may be necessary to establish a positive diagnosis:

1. Anamnesis.
2. Laryngoscopy, indirect.
3. Laryngoscopy, direct.
4. Complete, thorough, general physical examination
5. Roentgen-ray examination of chest and neck.
6. Blood tests, Bordet-Wassermann, etc.
7. Systemic tests: (a) Tuberculin; (b) therapeutic (with mercury, *not potassium iodide*).
8. Biopsy.
9. Bacteriology.
10. Bronchoscopy.
11. Esophagoscopy.

CHEVALIER JACKSON AND CHEVALIER L. JACKSON.

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## ANOMALIES OF THE LARYNX

Anomalies of the larynx are very uncommon.

Certain of the congenital malformations, such as congenital absence of the larynx and trachea, as noted in acephalus, are of anatomical interest only, as they are incompatible with life. Congenital stenosis or atresia of the larynx is very rare, and as a rule the child does not survive. Only those anatomical variations that have a clinical interest will be mentioned here.

In exceptional cases the larynx may be very small; associated with this in most instances is a high-pitched voice.

The epiglottis occasionally presents notches which penetrate so deeply as to appear to be a duplication of the epiglottis.

The ventricles of the larynx may be so abnormally enlarged as to form air-sacs.

Congenital curvature of the cartilages and deviations of the epiglottis may produce an actual congenital stenosis.

## DIAPHRAGM LARYNGIS

**Synonyms and Definition.**—Webs of the larynx; congenital laryngeal stenosis.

Diaphragm laryngis is the term given to the presence of a membranous formation within the larynx. This membrane seldom occurs on the posterior wall. The veil usually stretches between the true vocal cords or is immediately caudal, and is ventral in position. The diaphragm is pearly white in appearance and extends dorsally from the anterior commissure along the true vocal cords, uniting the margins by a membrane which as a rule allows a considerable range of motion. The web is concave or semi-lunar in shape and the posterior margin is free. The diaphragm varies in size and may occupy as much as two-thirds of the rima glottidis, which is normally the narrowest part of the laryngeal cavity.

On section the diaphragm is wedge-shaped; in appearance pearly white.

**Symptoms** are hoarseness, aphonia, dyspnea, and asphyxia. They may, however, be completely absent. The voice has a peculiar hoarse breathy quality which is especially prominent in crying children. Speech defects develop later.

**Diagnosis.**—The diagnosis is easy to establish by use of the laryngoscope, when a complete picture of the cavity of the larynx can be obtained, the membrane or veil being readily visible.

**Treatment.**—1. Dilation and catheterization. 2. Endolaryngeal division of the membrane. 3. Tracheotomy if indicated by marked dyspnea and respiratory embarrassment. 4. Laryngotomy with excision. The slighter form needs no treatment.

## LARYNGOCELE

**Definition.**—Laryngocele is the occurrence of an unusual widening of the laryngeal sinus (ventriculus laryngis—Morgagni), either unilateral or bilateral. The hernial protrusion of the mucous membrane may lie (*a*) within the larynx or may be turned on itself, or (*b*) may project as a hernia through the hyothyroid membrane and appear externally as a subcutaneous larynx, analogous to the air-sac which is present in apes.

**Symptoms** are absent if the mucosal hernia is small. Increase in the extent of the herniation induces interference with respiration.

**Diagnosis** is established by the presence of swelling within the larynx, which disappears on pressure and which is particularly palpable on coughing. If the sac is punctured with a needle there is an escape of air.

**Treatment** consists in extirpation through an external approach.

#### CONGENITAL THYROID AND CONGENITAL HYGROMA COLLI CYSTICA

**Synonym.**—Lymphangiomata.

These conditions are of interest in connection with the larynx only to the extent that they may produce pressure symptoms.

#### CERVICAL HEMATOCELE

**Synonyms and Definition.**—Blood cyst of the neck.

This is a very rare condition. It is caused by a cavernous enlargement of the veins and appears on the neck as a cystic tumor which makes pressure on the larynx and trachea.

**Treatment.**—Radical extirpation of a congenital cyst of the neck is usually impossible unless the tumor is quite small. Frequently the treatment must be confined to multiple punctures and injections of iodine. Complete surgical removal is the treatment of choice if it seems to be feasible.

#### CONGENITAL CYSTS AND FISTULAS OF THE NECK

**Definition.**—Congenital cervical fistulas and cysts of the neck occur as the result of a persistence of parts of the branchial system of cavities or of the cervical sinus, or of a tract or of cells that were dislocated in the descent of certain viscera, *e. g.*, thyroid and thymus.

From a clinical viewpoint they are divided into median and lateral, according to location.

No attempt is made here to include the numerous classifications of these congenital anomalies, because of the questionable utility of such a grouping.

#### LATERAL CERVICAL FISTULAS

**Definition.**—As the name indicates, lateral or branchial fistulas arise from one of the visceral arches. A large group takes origin from the second visceral arch. The inner orifice may be in the pharynx, palatopharyngeal arch, tonsillar fossa, or may open into the larynx or trachea. The last named condition, however, is exceedingly rare. More commonly the fissure in its cephalic extension becomes greatly attenuated and at times appears to end blindly. Tracheal or laryngeal fistulas are generally observed in woman only. The fistula usually takes an oblique course, and in the cases reported it seems possible that the trauma produced by probing was the cause of the tracheal fistula.

The inner opening may become dilated into a diverticulum, which appears in the esophagus or in the pharynx and produces marked difficulty in swallowing. If the diverticulum is situated in the trachea, a large sac filled with air may form in the anterior part of the neck.

The outer or external opening is usually small and is situated most commonly at the side of the neck in the neighborhood of the sternocleidomastoid muscle, midway between the hyoid bone and the sternoclavicular joint; or more medially in the neighborhood of the larynx. As many as three openings on one side of the neck have been observed.

The fistulous tract is covered by the skin, platysma, and the superficial layer of the deep fascia. It runs parallel to the medial edge of the sternocleidomastoid muscle, dips under the digastric muscle, and frequently ends in the pharynx or in one of the sites noted above. The tract is superficial to the common carotid artery. It passes between the internal and external carotid vessels and continues dorsal to the digastric muscle. Several fistulas may be present; a single tract, however, is the rule.

The development of median congenital cysts and fistulas of the neck is explained by the persistence of the whole or a part of the thyroglossal tract which runs from the foramen cecum of the tongue to the hyoid bone. The fistula is a primary incomplete cutaneous perforation which occurs later in life. The tract is in the median line and is readily palpable as a hard cord which extends to the hyoid bone or as low as the isthmus of the thyroid.

Median cysts may form anywhere in the median line from the foramen cecum to the jugular notches. They are most commonly located above or below the hyoid bone. They are usually about 5 cm. in diameter. As a rule the cysts do not appear until puberty and do not cause symptoms unless infection supervenes.

The fistulas may appear during intra-uterine life or after birth. It is not uncommon for the cutaneous opening to first appear after an attack of one of the acute infectious diseases of childhood.

**Symptoms and Diagnosis.**—Symptoms are often absent.

The diagnosis is easy to establish. The patient presents a small external opening which is discharging a serous or seropurulent exudate. The amount of discharge varies from 1 drop to 1 c.c. daily. Frequently there is a co-existent eczema of the surrounding skin. At times the fistula can be palpated as a distinct cord in the neck. If the outer opening is elevated on swallowing, a complete fistula is indicated. The injection of one of the various coloring fluids, such as methylene blue, is helpful in diagnosis.

The presence of epithelial cells in the secretion is in favor of fistula, and is evidence against a broken-down tuberculous lymph-node as the cause of the cutaneous opening. Its congenital origin and the hereditary tendencies of the patient are additional confirmatory data.

**Treatment** is often unnecessary as the symptoms are usually of no importance. Their chief danger consists in the possible development of malignancy from the embryonal cell-rests; definite observation on the relation of branchial remnants to obscure carcinoma of the neck is lacking at the present time. The utilization of methods of treatment other than surgery results in recurrence.

The injection of bismuth for Roentgen ray and the introduction of methylene blue before operation are helpful in outlining the course of the fistulous tract.

As a rule median cysts are easily excised up to the level of the hyoid bone; at this point the tract becomes friable and is easily torn. The tract, as a rule, passes in front of the hyoid bone.

Better results are obtained if no attempt is made to isolate the tract above the hyoid bone. The tract is here cored out, removing with the duct the tissues surrounding it for  $\frac{1}{8}$  inch on all sides and including  $\frac{1}{4}$  inch of the hyoid bone. Sistrunk advises the removal of the foramen with the tract. The opening in the mouth is closed and the structures of the neck are drawn

together by chromic catgut sutures. A small rubber-drain is introduced and the skin closed about it.

Inversion of the proximal parts of the tract through an oral incision may be indicated in difficult cases.

Lateral fistulas or cysts can be successfully removed only by radical excision of the entire tract. No other treatment accomplishes the purpose.

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### LARYNGOPTOSIS

Ptosis of the larynx may be said to exist when the larynx is so low that there is not at least one tracheal ring above the manubrium. It is probably in most cases congenital, though the shortening of the spine in vertebral disease may cause it; possibly also cicatricial contractions. There are usually no symptoms unless the larynx is very low; if the cricoid is entirely below the upper edge of the manubrium the voice is usually deep in pitch and dull and monotonous in tone. It is usually not noticed unless tracheotomy is being done. When I first gave the condition its name<sup>1, 2</sup> it seemed to my colleagues more rare than it actually is. My habit of always carefully palpating every cartilage and every ring, in every patient examined for any condition, has revealed 67 cases of laryngoptosis. Of course the ptosis itself did not call for bronchoscopy, therefore endoscopic examination was limited to the 14 patients in whom bronchoscopy was indicated for other reasons. Of these, 10 showed deviation of the trachea—in 2 the trachea seemed to have been made redundant in length by the ptosis, in 4 the deviation was caused by the pushing of a goitrous or other substernal mass, in the other 4 the trachea seemed normal. Whether the lateral pushing dragged the larynx downward or not it is impossible to say, but that was the impression on endoscopy. The chief clinical importance of the condition arises when a patient with a laryngoptosis needs a tracheotomy for conditions other than the ptosis; a low tracheotomy cannot be done without opening the anterior mediastinum, risking mediastinal emphysema and pulmonary atelectasis; because the tracheal rings are behind the sternum. A high tracheotomy, especially if the cricoid is cut and the cannula worn for a long period, is almost certain to cause laryngeal stenosis. We avoided stenosis by not cutting the cricoid, using a small cannula through an opening carefully made through the first and second rings while the trachea was drawn strongly up. Mediastinal emphysema was prevented by packing the whole wound open. The cannula should be got rid of as soon as possible except in the case of hopelessly inoperable cancer. In two cases a special tracheal intubation tube was used.

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## THE MUSCULATURE AND INNERVATION OF THE LARYNX

**Introduction.**—The larynx is an organ which subserves two widely different but related functions, viz., a respiratory and a phonatory function. These two characteristic functions are in a sense antagonistic, one to the other, and are accomplished by the co-operative antagonism of two different groups of muscles—a respiratory and a phonatory.

For respiratory purposes it is essential that the vocal bands or vocal folds be widely separated and the area of the glottis sufficiently large to admit the passage of the air into and out of the lungs without noticeable resistance. The investigations of Semon established the fact that the glottic area is approximately 160 sq. mm. during both inspiration and expiration under physiological conditions. This area is somewhat less than the area of either the supra- or the infra-glottic region, each of which is about 200 sq. mm., and maintained and conditioned by the position of the vocal bands.

For phonatory purposes, or when the emission of a laryngeal sound of varying pitch is desired, it is essential that the inspiratory phase of respiration be temporarily suspended and the vocal bands closely approximated and tensed until the area of the glottis is reduced to a very narrow slit, when it is known as the rima vocalis. At the same time the rima respiratoria must be obliterated, which is accomplished by the approximation of the arytenoid cartilages.

**The Laryngeal Musculature.**—The innervation of the larynx or the nerve mechanism by which the muscles of the larynx are excited to, and co-ordinated in, action for the performance of these two functions will be more readily understood if the actions of the muscles and the mechanisms they control be first briefly considered.

The muscles by which these two functions are made possible may be divided into two groups, viz., a respiratory and a phonatory. The former consists of the (1) two posterior crico-arytenoid muscles, the latter consists of (2) the two lateral crico-arytenoid muscles, (3) the single transverse arytenoid muscle, (4) the two thyro-arytenoid muscles, and (5) the two thyrocricoid muscles.

1. The *posterior crico-arytenoid muscles* lie on the posterior surface of the quadrate plate of the cricoid cartilage, one on each side of the median line from which they arise. From this origin the fibers of each muscle pass upward and outward, and in their course converge to be inserted into the dorsal aspect of the muscular process of the arytenoid cartilage. The superior and horizontally directed fibers when in action rotate the arytenoid cartilages around a vertical axis; the inferior and obliquely directed fibers draw the base of the arytenoid cartilage downward and inward. As a result of their combined action the vocal processes, are directed upward and outward and in their movement each describes an arc of a circle and, since the vocal bands are attached to the vocal processes, they too are carried upward and outward and the glottis thereby widened—the condition necessary for respiratory purposes. As the contraction of the posterior crico-arytenoid muscles has this result, they are generally designated the *abductors* of the vocal bands or the *dilatators* of the glottis or the *respiratory* muscles.

2. The *lateral crico-arytenoid muscles* lie in the lateral wall of the larynx under cover of the plate of the thyroid cartilage. They arise one each from

the ring portion of the cricoid cartilage as far back as the facet which supports the arytenoid cartilage. From this origin the fibers pass upward and backward to be inserted into the ventral aspect of the muscular process of the corresponding arytenoid cartilage. These muscles, when in action, rotate each arytenoid cartilage around a more or less vertical axis and at the same time direct the vocal processes inward and downward. As a result of this movement the vocal bands are directed to the median line and closely approximated. That this may be accomplished it is essential that the vocal processes be released from the control of the posterior crico-arytenoid muscles, an event made possible by an inhibition of their tonic contraction, coincident with an increase in the tonus of the lateral crico-arytenoid muscle until the tonic contraction is established.

3. The *transverse arytenoid muscle* lies on the dorsal aspect of the larynx, just above the upper level of the cricoid cartilage, and closes in the space between the arytenoid cartilages. This muscle consists of (1) transversely arranged fibers which arise from and are inserted into the outer surface of the opposite arytenoid cartilages, and of (2) obliquely directed fibers which arise from the outer angle of one arytenoid to be inserted into the apex of the other. In their course the obliquely directed fibers decussate in the median line. The action of this muscle is to draw the arytenoid cartilages up on the top of the cricoid cartilage and close enough to obliterate the triangular area of the glottis—the *rima respiratoria*—which remains after the advance of the vocal bands to the median line by the action of the lateral crico-arytenoid muscles. By the co-operative action of these two muscles the *rima vocalis* is established.

4. The *thyro-arytenoid muscles* are thin and quadrangular in shape, and lie between the plate of the thyroid cartilage and the wall of the ventricle. These muscles arise one on each side from the lower half of the inner surface of the thyroid just external to the median line. From this origin the fibers pass upward and backward to be inserted into the lateral border and muscular process of the arytenoid cartilages. When in action, these muscles rotate the arytenoid cartilages inward, draw the vocal processes downward and inward, and thus slightly relax the vocal folds or the vocal bands.

The upper or superior border of each muscle is somewhat enlarged, and doubtless serves as a constrictor of the supraglottic region. The inferior border is likewise enlarged and prismatic in shape and occupies the space between the two layers of the vocal folds. The close anatomical relationship between these fibers and the thyro-arytenoid proper makes it difficult to determine whether or not these fibers constitute a separate muscle to which the term "the vocal muscle" should be given. From its origin and insertion it is probable that it co-operates with its associated fibers in relaxing the vocal folds or bands.

Of the foregoing muscles the lateral crico-arytenoid and the thyro-arytenoid muscles are more especially termed by reason of the effect of their action, the *adductors* of the vocal bands. As the transverse arytenoid muscle approximates the arytenoid cartilages these muscles are collectively termed the *constrictors* of the glottis or the *phonatory* muscles. As a result of their combined action the vocal bands and the arytenoid cartilages are placed in an anatomical position which makes possible the emission of a laryngeal sound.

In the production of a sound the air that has been stored or collected

in the lungs by an inspiratory effort is forced upward through the narrow space between the vocal bands by the contraction of the expiratory muscles. In consequence of the resistance offered by this narrow outlet, the air within the lungs is subjected to pressure and, as soon as the pressure attains a certain value, the vocal bands are thrown into vibration. The vibrations in and of themselves would give rise, if not complicated by additional vibrations in the upper air-passages, to what would be termed the laryngeal sound. This sound becomes modified in various ways by the resonance of the air in the pharyngeal and oral cavities, thus giving rise to the familiar vocal sounds. So soon as the phonatory effort ceases, the phonatory muscles relax, whereupon the respiratory muscles resume their former degree of activity and return the vocal bands to the respiratory position.

All vocal sounds are characterized by three qualities: pitch, intensity, and quality or timbre. The pitch of the voice depends on the number of vibrations of the vocal bands in a unit of time—a second. The rate of vibration will in turn be conditioned by the length of the bands in vibration or the length and width of the aperture through which the air passes and the degree of tension to which the bands are subjected. These conditions are made possible by the action of the following muscles.

4. The *thyrocricoid muscles* consist of two well-developed muscle bundles which from their direction are termed vertical and oblique. The vertical bundle arises from the side and inferior border of the thyroid plate; the oblique bundle arises from the inferior cornu. From this origin the fibers composing these bundles pass downward and forward over the thyrocricoid interval, to be inserted into the lateral surface and lower border of the arch of the cricoid cartilage.

These muscles, when in action, lengthen and hence tense the vocal bands, the degree of which depends on the pitch of the note to be emitted. The manner in which this is done, however, has been a subject of much discussion. The customary statement is that the muscles when excited to activity exert traction from the cricoid arch and draw the thyroid cartilage downward and thus, by increasing the distance between the origin and insertion of the vocal bands, lengthen them. This view, however, is not supported by experimental investigations.

The result of all experimentation on the action of these muscles makes it apparent that when stimulated they pull from the thyroid cartilage (acting as the fixed point) and draw the arch of the cricoid up on to the thyroid. For this reason it is more appropriately termed the "thyrocricoid muscle." The result of this movement is the rotation of the quadrate plate of the cricoid and the arytenoid cartilages as well, backward and downward, the movement taking place around a horizontal axis passing through the thyrocricoid articulation. The result of this movement would necessarily lengthen and tense the vocal bands\* (Hooper).

**The Protective Mechanism of the Larynx.**—The air-passages are protected from the entrance of small particles of food during deglutition by several mechanisms. Just preceding and during the act of deglutition there is a complete suspension of the act of inspiration by which particles of food might otherwise be drawn into the larynx; at the same moment

\* Experimental researches on the tension of the vocal bands, paper read at the Fifth Annual Congress of the American Laryngological Association, May 23, 1883. Issued as a supplement to the Archives of Laryngology, July, 1883.

the larynx is drawn well up under the base of the tongue and its entrance closed by the downward and backward movement of the epiglottis, the result of the contraction of the aryepiglottic and thyro-epiglottic muscles.

In addition to the foregoing mechanism it is also probable from the observations of Meltzer that the air-passages are protected in the rabbit at least by the closure of the glottis itself. This experimenter noticed, while observing the interior of the larynx, both from above, through an opening in the hyothyroid membrane, and from below, through an opening in the trachea, that when an act of deglutition was excited by touching the soft palate with a sound there was, simultaneously with the contraction of the mylohyoid muscles, a firm constriction of the glottis. This was accomplished by an approximation of the true vocal bands, a close approximation and a downward and forward movement of the arytenoid cartilages, until they almost touched the anterior wall of the thyroid cartilage. This movement preceded the ascent of the larynx. When the larynx was separated from all surrounding structures with the exception of the laryngeal nerves, a touch of the palate excited the same phenomenon. Under such circumstances the constriction of the glottis must have been due to the contraction of the sphincter muscles and in consequence of a reflex action through the inferior laryngeal nerves.

**The Reciprocal Action of the Abductor and Adductor Muscles.**—In harmony with what is observed elsewhere in the body the laryngeal muscles are in the physiological condition in a state of tonus, a state characterized by a slight degree of contraction which enables them to act quickly and energetically on the arrival of the physiological stimulus—the nerve impulse. In the absence of phonatory efforts the vocal bands are widely separated and remain so sleeping or waking by reason of the tonic contraction of the posterior crico-arytenoid muscles and may so remain indefinitely. This continuous activity of the posterior crico-arytenoid muscles is well exhibited in the Trappist Monks, who have taken the vow of perpetual silence when among themselves. With the onset of phonation the vocal bands are instantly brought to the median line and the glottis almost obliterated by the sudden increase of the tonic contraction of the lateral crico-arytenoid and associated adductor muscles. With the cessation of the phonatory effort the adductor muscles undergo a diminution in their tonicity, whereupon the abductor muscles at once restore the vocal bands to the respiratory condition.

**The Sphincter Muscle of the Larynx.**—It has just been stated that the lateral crico-arytenoid, the transverse arytenoid, and the thyro-arytenoid muscles together constitute the sphincter of the glottis. The development of these muscles would indicate that they arise from a common sphincter muscle which appears in the early stages of embryonic life. At this period a muscle band completely surrounds the cavity of the larynx. With the development and differentiation of the laryngeal cavity and more especially of the arytenoid cartilages, this muscle separates into three portions. These portions then seek the points of attachment which characterize them in adult life and from which they derive their names.

These muscles are very frequently supported in their action by a superficial ring of muscle fibers, which has been described as a secondary formation but independent anatomically of them. Arising from the inner surface of the angle of the thyroid cartilage the fibers pass backward and in their

course sweep around the arytenoid cartilage of the same side to be attached to the arytenoid cartilage of the opposite side.

### THE INNERVATION OF THE LARYNX

The innervation of the larynx or the nerve mechanism by which the laryngeal muscles are excited to and co-ordinated in action is quite complex and involves the co-operation of efferent nerves, a central mechanism, and afferent nerves. A consideration of this mechanism may be advantageously approached from the following points of view: (1) The laryngeal muscles and their action; (2) the efferent or motor nerves, their separate and combined action; (3) the central mechanism; (4) the afferent nerves by which the central mechanism may be increased or decreased in activity.

The laryngeal muscles by the action of which the two characteristic functions of the larynx are made possible have been considered in foregoing pages.

1. *The Efferent or Motor Nerves.*—The two nerves which are distributed to the muscles of the larynx and excite them to action are the superior and inferior laryngeal branches of the vagus nerve.

The superior laryngeal arises from the *ganglion nodosum* and after a short course divides into two branches, a small external and a large internal. The *external* branch of the superior laryngeal passes downward and is finally distributed mainly to the thyrocricoid muscle. It has been stated by Exner and others that some of its branches are distributed as well to the lateral crico-arytenoid muscle, the transverse arytenoid muscle, and in some instances to the thyro-arytenoid muscle. The physiological character of these nerve-fibers, *i. e.*, as to whether they are motor, sensory, or vasomotor, has not been at all clearly defined. The *internal* branch passes medially and enters the larynx through the thyrohyoid membrane. It is distributed to the mucous membrane of the larynx and of the epiglottis. A small branch of this nerve passes downward beneath the plate of the thyroid cartilage to anastomose with branches of the inferior laryngeal nerve.

The inferior or recurrent laryngeal nerve arises from the vagus at different levels on the two sides. On the right side the nerve arises from the vagus as it crosses the first part of the subclavian artery, after which it passes around this artery and ascends in a groove between the esophagus and the larynx. At the level of the thyrocricoid articulation the nerve divides into two branches, an anterior and a posterior: the former is distributed to the lateral crico-arytenoid, the thyro-arytenoid, and the transverse arytenoid muscles; while the latter is distributed to the posterior crico-arytenoid and apparently to the transverse arytenoid muscles, on both sides of the median line.

On the left side the inferior laryngeal nerve arises from the vagus as it crosses the arch of the aorta. After winding behind the aorta it turns upward as far as the neck after which its course, relations, and ultimate distribution are the same as that of the right nerve.

In the study of the functions of these two nerves—more particularly of the inferior laryngeal nerve—as well as in the interpretation of clinical conditions, much difficulty has been experienced by reason of the extensive distribution of their terminal branches as disclosed by anatomical investigations. Recalling the fact that these nerves contain—in addition to their

motor fibers—sensory fibers, and perhaps vasomotor fibers, it is difficult to determine the distribution of the motor fibers alone to which clinical interest is for the most part directed. The effects, however, which follow division and stimulation of these nerves by the employment of physiological methods are more likely to disclose their distribution and function than the methods of dissection.

*The Superior Laryngeal Nerve.*—A transverse section of these nerves is at once followed by a loss of sensibility in the laryngeal mucous membrane, a paralysis or relaxation of the thyrocricoid muscles with a consequent lowering of the pitch and a diminution in the clearness of the voice. As a result of the loss of sensibility there is an inability to perceive the entrance of foreign bodies into the larynx. *Stimulation* of the peripheral portion of the divided nerve with rapidly repeated induced electric currents causes contraction of the thyrocricoid muscles with a lengthening and tensing of the vocal bands, the result of the approximation of the arch of the cricoid cartilage to the thyroid cartilage. There is no experimental evidence that any other muscle is excited to action by this procedure. Peripheral stimulation of the superior laryngeal nerve as by the contact of foreign particles develops nerve impulses which, reflected centrally, cause: (a) Sensations of pain, and (b) a vigorous contraction of the adductor muscles and a firm closure of the glottis. This latter effect, it is generally admitted, is the result of a central stimulation of the cells from which the motor fibers of the inferior laryngeal nerve take their origin, and which are distributed to the adductor muscles.

*The Inferior Laryngeal Nerve.*—A transverse section of the inferior laryngeal nerve or its stimulation by electric or mechanic methods is followed by phenomena difficult of interpretation, mainly by reason of the fact that the nerve-fibers composing it have antagonistic functions and hence respond differently to different strengths of the stimuli as well as to other conditions to which reference will be made in subsequent paragraphs.

A knowledge of the origin, course, and separation of the nerve-fibers contained in the trunk of this nerve is necessary to a correct interpretation of the results which follow its stimulation or injury from abnormal conditions.

As a result of many anatomical investigations it is now well known that the efferent or motor fibers distributed to the respiratory and phonatory muscles take their origin from two groups of nerve-cells located in the gray matter beneath the floor of the fourth ventricle and which have been termed the "laryngeal respiratory" and the "phonatory centers" respectively. From these centers the nerve-fibers pass outward in the lower roots of the vagus nerve, and after entering its sheath descend as far as the levels stated in a foregoing paragraph. The two nerve-bundles from the point at which they leave the vagus are more especially known as the inferior laryngeal nerves. At the level of the thyrocricoid articulation the fibers coming from the laryngeal respiratory center enter the posterior crico-arytenoid muscle and become histologically related to its individual muscle-fibers. The fibers coming from the phonatory center ascend as far as the crico-arytenoid articulation where some turn forward to be distributed to the lateral crico-arytenoid and to the thyro-arytenoid muscles; others turn backward and enter the transverse arytenoid muscle. The foregoing interesting anatomical facts were fully established by the investi-

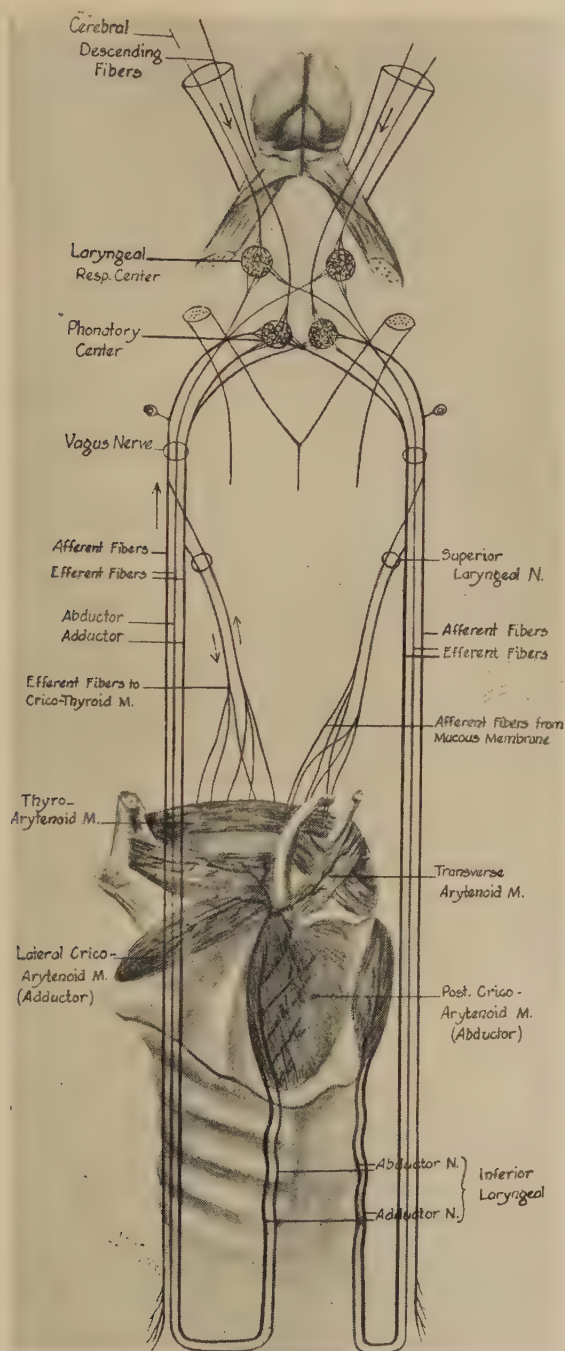


Fig. 404.

gations of Risien Russell,\* who succeeded in separating and isolating the two bundles of fibers, from their origin from the vagus to their termination.

\* Proceedings of the Royal Society, March 31, 1892.

So accurately was this done that it became possible to excite at will one or the other bundle and evoke a contraction of either the abductor muscle or the adductor muscles without evoking a contraction of the muscle of opposite function. A transverse section of one or the other bundle was followed by paralysis and degeneration of its related muscle without causing any impairment in the irritability or contractility of the muscle of opposite function. These experiments, performed on dogs, were most successfully carried out and have been very generally accepted. The results which followed stimulation or a transverse section of these two separate nerve-bundles indicate very clearly their final distribution.

Notwithstanding the apparent similarity of nerves and muscles in general, there is, nevertheless, between nerves and muscles of opposite function an apparent histological dissimilarity either in the nerve-endings or in the muscle-fibers, which causes them to respond somewhat differently to the action of various external agents—*e. g.*, the introduction of lead into the body primarily impairs the terminals of the nerves distributed to the extensor muscles of the forearms rather than to the flexors, as indicated by the falling of the hand from the wrist; ether, when applied to the leg muscles of the frog, diminishes the irritability of the nerves distributed to the extensor muscles, as shown by the fact that electric stimulation of the sciatic plexus, which under normal conditions causes extension of the leg and abduction of the toes, now causes flexion of the leg and adduction of the toes.

Different experimental procedures indicate that there is also a histological difference between the abductor and adductor nerves and muscles of the larynx. Thus, when the inferior laryngeal nerve is subjected to the action of cold, dry air or mechanic pressure, the abductor fibers lose their irritability and conductivity more rapidly than the adductors, as shown by the effects of electric stimulation; moreover, after death the abductor muscles lose their irritability and contractility sooner than the adductors. Under thorough etherization of an animal, *e. g.*, the dog, the adductor nerves lose their irritability rather than the abductors as shown by the fact that stimulation of the inferior or recurrent laryngeal nerve now causes abduction of the vocal bands, rather than adduction which is the usual result under slight anesthesia or no anesthesia at all.

Many experiments have been made on the inferior laryngeal nerve by different investigators, employing different animals, varying strengths of stimuli, under varying degrees of anesthesia, and hence various and often conflicting results have been obtained. As an outcome of all these experiments the consensus of opinion appears to be that in the adult higher mammal, *e. g.*, the dog and monkey, stimulation of this nerve causes contraction of the adductor muscle and the advance of the vocal band to the median line rather than contraction of the abductor muscle and an outward movement of the vocal band. In other words, the effect of the simultaneous stimulation of the two bundles of nerve-fibers is more manifest in the adductor group of muscles than in the abductor and this notwithstanding the greater size of the abductor muscle. Transverse section of the inferior laryngeal is followed by paralysis of both groups of muscles, with the exception of the cricothyroid which receives its innervation from a motor branch in the trunk of the superior laryngeal. The vocal bands at once assume a position similar to that observed in the cadaver.

The **central mechanism**, by which the laryngeal muscles are excited to and co-ordinated in action, consists, as previously stated, of two groups of nerve-cells, viz., a laryngeal respiratory and a laryngeal phonatory, both of which are located in the medulla oblongata.

The respiratory group or center is located in the upper part of the floor of the fourth ventricle. From the cells of this center nerve impulses are discharged and then transmitted by their fibers to the posterior crico-arytenoid muscles. From the more or less continuous activity of the abductor muscles as shown by the almost stationary position of the vocal bands during quiet respiration, it is a justifiable assumption that the laryngeal center is likewise in a state of more or less continuous activity or tonus, a condition for which several factors might be presented. Thus, it might be assumed that, as the outcome of chemical changes between the gaseous constituents of the cells and the gaseous constituents of the blood by which they are surrounded, the cells discharge nerve impulses in a steady and continuous manner; or it might be assumed that the activity of the cells is maintained by the stimulating activity of nerve impulses reflected from the terminals of some sensory nerve, presumably the vagus. Experimental proof of either assumption is largely wanting.

The location and activity of this center can be demonstrated by subjecting it to experimental procedures. Thus, if this center or area be stimulated with induced electric currents on one side only, not only will there be increased contraction of the abductor muscles and outward displacement of the vocal band on the same side, but on the opposite side as well; in other words the representation of the laryngeal respiratory muscle-movements in this center is bilateral rather than unilateral.

The phonatory group or center is located in the floor of the fourth ventricle near the ala cinerea and the upper border of the calamus scriptorius. From the cells of this center nerve impulses are discharged and transmitted by their fibers to the various phonatory muscles. Since there are good reasons for believing that these muscles are in a state of tonus, though probably not to the same degree as the laryngeal center, the probabilities are that this center likewise is in a state of tonus or continuous activity.

Stimulation of this area with induced electric currents on one side only gives rise, not only to prompt and vigorous contraction of the phonatory muscles and adduction of the vocal band on the same side, but on the opposite side as well; in other words, the representation of the movements of the phonatory muscles in the medulla is, therefore, bilateral rather than unilateral. An explanation for the bilateral action of these centers is extremely difficult to furnish. The only plausible assumption is that some of the fibers emanating from the cells of one side cross the median plane of the medulla and enter the trunk of the vagus nerve of the opposite side. Such decussation of fibers has not as yet been positively demonstrated.

**The Afferent Nerves.**—The central mechanism may be acted on by nerve impulses transmitted to it by afferent nerves coming, first, from the cerebrum and, secondly, from mucous and cutaneous surfaces. In the first instance the resulting movement would be termed direct, cerebral, or volitional, and in the second instance reflex, peripheral, or non-volitional. That the activities of the cerebrum associated with psychic states of a volitional and emotional character can and do influence the activities of

the laryngeal respiratory and phonatory centers, is a matter of observation and personal experience. If the phonatory muscles of the larynx can be made to contract and approximate the vocal bands by a volitional effort then there must be in the cortex an area of motor cells in which the movements of the phonatory muscles have their representation; hence stimulation of this area leads to a discharge of nerve energy by way of the pyramidal tract to the phonatory center in the medulla oblongata. This area of cortical cells has been experimentally located in the dog and monkey in a region corresponding in man to the foot of the precentral convolution on both sides of the cerebrum. Stimulation of this area on one side causes a bilateral adduction of the vocal bands in a manner similar in all respects to the bilateral adduction that follows unilateral stimulation of the phonatory center in the medulla. As this effect follows stimulation of either side it is apparent that a destruction of this area on one side only will not be followed by a paralysis of the phonatory muscles on either side.

The existence of a laryngeal respiratory center or a cortical center in which the movements of the abductor muscles are represented has been much discussed by reason of the very contradictory results of experimental investigations. The investigations of Risien Russell have finally demonstrated the existence of such a center which is found in close anatomical relation to the phonatory center. Hence the effect of the stimulation of this region is manifested in both, but as the phonatory center is either more responsive or has a greater influence on the phonatory center in the medulla than the cortical respiratory center has on the laryngeal respiratory center, adduction of the vocal bands is generally the effect observed.

Taking advantage of the fact previously discovered by Russell that the adductor and abductor fibers in the inferior laryngeal nerve could be isolated and separately stimulated and divided, he exposed and prepared this nerve and then divided transversely the adductor fibers and thus eliminated the effect of stimulation of the cortical phonatory center. After this procedure stimulation of an area lying anterior to and below the location of the phonatory center was at once followed by a bilateral abduction of the vocal bands.

The mucous surfaces of the body, and especially the mucous surface of the larynx, trachea, stomach, and intestines, are associated with the phonatory center in the medulla more especially through the intermediation of the afferent fibers of the vagus nerve. The wide anatomical distribution of the afferent fibers of this nerve readily explains how stimulation of this or that mucous surface may be followed by adduction of the vocal bands and the arytenoid cartilages as well.

It is only necessary to recall the spasmodic contraction of all the phonatory muscles that follows the entrance of a foreign body, small or large, into the larynx thus stimulating the terminals of the superior laryngeal branch; or to recall the spasmodic condition known as laryngismus stridulus developed by stimulation of the vagus terminals by gastric and intestinal disorders, especially in children. Other instances of peripheral stimulation will be readily recalled by the clinician.

ALBERT P. BRUBAKER.

## MALINGERING AND HYSTERIA

The laryngologist may feel at times that he should not be beset with hysteric, neurotic, and highly imaginative individuals who take from him time that should be devoted to the treatment of patients with organic lesions. The most careful and thorough examination is necessary, however, before reaching the conclusion that a patient has no organic lesion. Hundreds of patients with cancer of the esophagus coming to the Bronchoscopic Clinic have given a history showing clearly that they had been considered hysteric or neurotic because of the vagueness of the early symptoms so difficult for them to describe. Not until after objective examination including endoscopy, besides the Roentgen-ray examination, the Bordet-Wassermann, and the complete general physical examination, should it be decided that no organic disease is present.

Even in the absence of an organic lesion the patient may be suffering from a no less real functional disorder. All patients in whom no lesion to account for their symptoms is found should be referred to the neurologist. No matter whether the condition results from a desire for sympathy or a desire to escape work or duty, or from some other cause, the neurologist is best fitted to treat it. In a few patients incipient mental disorder will be discovered. No attempt should be made by the laryngologist to cure hysterical conditions by suggestion or minor operation. Such "cures" as may be obtained will be ephemeral. If any benefit is to be obtained by suggestion, which is doubtful, it should be carried out by the neurologist. In a few instances the later developments may reveal a lesion not discoverable at the first examination. Various forms of hysteria and malingering and their differential diagnosis will be found under the diseases simulated.

CHEVALIER JACKSON.

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 THE NEUROSES AND NEURONOSSES OF THE LARYNX

**Classification.\***
**A. Sensory Neuroses.**

- (a) Anesthesia.
- (b) Hyperesthesia.
- (c) Paresthesia.

**B. Motor Neuroses.**

*Laryngeal Spasm.*—Reflex and central spasm of all or some of the laryngeal muscles. Two forms:

- (a) Glottic spasm in adults.

Synonyms: Spasm of the tensors of the vocal cords; phonatory spasm.

- (b) Adductor spasm (clonic spasm of the adductors).

\* *Laryngismus stridulus* (Synonyms: Spasm of the glottis in children; spasmodic croup; false croup; adductor spasm; thymic asthma) and congenital laryngeal stridor (Synonym: Infantile respiratory spasm) are not now regarded as neuroses, and are therefore not included in this classification.

*Laryngeal Paralysis.*

I. Paralysis of the intrinsic muscles supplied by the superior laryngeal nerve.

Paralysis of the cricothyroid muscles. Synonym: Paralysis of the external tensors.

II. Paralysis of the intrinsic muscles supplied by the recurrent laryngeal nerve.

Paralysis of the adductors of the vocal cords.

(a) Unilateral adductor paralysis.

(b) Bilateral adductor paralysis.

(c) Paralysis of the arytenoideus—the transverse or central adductor.

Paralysis of the thyro-arytenoideus interna and externa.

Synonym: Paralysis of the internal tensors of the vocal cords.

Paralysis of the abductors of the vocal cords.

(a) Unilateral abductor paralysis.

(b) Bilateral abductor paralysis.

C. *Incoördination Neuroses.* (Incoördination in action of the muscles.)

(a) Spasmodic laryngeal cough. Synonym: Laryngeal chorea.

(b) Laryngeal vertigo. Synonym: Laryngeal syncope.

## A. SENSORY NEUROSES

(a) **Anesthesia of the Larynx.**—*Etiology.*—The causes may be peripheral. It is met with in anemia and old age, and in some general affections, *e. g.*, influenza, pneumonia, typhus, etc., or may be due to nerve affections, *e. g.*, the peripheral neuritis of diphtheria, or paralysis of the superior laryngeal nerve. It may also be caused by affections of the central nervous system, *e. g.*, bulbar lesions, hysteria, epilepsy.

*Symptom.*—Dysphagia.

*Diagnosis.*—Local anesthesia must be differentiated from that resulting from central nerve disease.

*Prognosis.*—Danger of food entering the larynx and causing pneumonia. Peripheral neuritis of diphtheria is usually recovered from in a few weeks.

*Treatment.*—Tonics, faradic current. In severe cases of dysphagia patients should be fed through a nasal feeding tube.

(b, c) **Hyperesthesia and Paresthesia.**—Excessive or altered sensation. The two conditions may be considered together, since they are generally associated.

*Etiology.*—Abnormal conditions of the general nervous system, *e. g.*, anemia, hysteria, fatigue, indigestion, influenza, alcoholism, gout. May be met with in laryngitis, hypochondriasis. Reflex origin, *e. g.*, teeth, tonsils, thyroid gland; irritation of, or pressure on, the superior laryngeal nerve.

*Symptoms.*—Continuous or paroxysmal sensation of a foreign body in the larynx, burning, raw, pricking, or choking feeling, which gives rise to spasmodic cough and hawking.

*Laryngoscopic Examination.*—Shows nothing abnormal.

*Diagnosis.*—Organic disease, *e. g.*, early locomotor ataxia, gout, etc., must be excluded. Sensations may be referred to the larynx from adjoining parts.

*Prognosis.*—In some cases may be difficult to cure.

*Treatment.*—The cause must be ascertained and treated by suggestion, electricity, attention to diet and hygiene, tonics, sedatives, anodyne sprays.

## B. MOTOR NEUROSES

## LARYNGEAL SPASM

(a) **Glottic Spasm in Adults.**—*Synonym.*—Phonatory spasm.

*Etiology.*—Hysterical spasm is a common form of functional inspiratory spasm in adults, occurring in epilepsy; reflex irritation of larynx, pharynx, or nares, *e. g.*, catarrh, ulcerations, growths; irritation of recurrent laryngeal nerve, *e. g.*, by aneurysm, tumors, glands; central nerve lesions, *e. g.*, locomotor ataxia, tetanus. Cases may occur in elderly people who are alcoholic, rheumatic, or gouty, or suffering from emphysema and diseases of the heart or aorta.

*Symptoms.*—Choking feeling and cough, globus hystericus, rapid noisy inspiratory stridor of a crowing character followed by asphyxia. The accompaniment of a "brassy cough" suggests aneurysm of the aorta or mediastinal growth.

*Laryngoscopic Examination.*—The vocal cords during inspiration are closely approximated, simulating double abductor paralysis.

*Prognosis.*—The condition is rarely fatal, except when some cardiac lesion or arteriosclerosis exists. Tracheotomy is rarely necessary.

*Treatment.*—The general principles for the treatment of the various acute or chronic conditions which give rise to the spasm should be carried out. Cocaine spray during attack, inhalation of chloroform, or amyl nitrite. Encourage patient to take quiet, rapid, shallow respirations which relax the spasm. In functional cases—suggestion, avoidance of any source of reflex irritation, attention to general condition, diet, hygiene. In tabetic crises—bromides and other sedatives.

(b) **Adductor Spasm.**—*Synonym.*—Clonic spasm of the adductors. Rhythmical contractions of the vocal cords, usually associated with twitchings of the soft palate and postpharyngeal wall. Occurs after meningitis, in syphilis, tumors of the brain, paralysis agitans, etc.

## LARYNGEAL PARALYSES

1. **Paralysis of Muscles Supplied by the Superior Laryngeal Nerve.** **Paralysis of the Cricothyroid Muscles.**—*Synonym.*—Paralysis of the external tensors.

Is rare, especially if occurring alone.

*Etiology.*—Injury from operation, enlarged glands, malignant growths, diphtheria.

*Symptoms.*—Weak and rough voice, easily fatigued.

*Laryngoscopic Examination.*—The paralysed cords have a wavy outline, and flap owing to loss of tension. If unilateral the paralysed cord lies at a lower level than the normal one.

*Treatment.*—Faradic current, strychnine, blisters.

2. **Paralysis of the Intrinsic Muscles Supplied by the Recurrent Laryngeal Nerve.** **Paralysis of the Adductors of the Vocal Cord.**—(a) **Unilateral Adductor Paralysis.**—*Synonym.*—Unilateral recurrent paralysis. Is a rare affection, one crico-arytenoideus lateralis muscle being involved.

*Etiology.*—Is usually functional (hysteria), but has been observed in cases of syphilis, lead poisoning, smallpox, enteric and typhus fever, etc.

*Symptom.*—Hoarseness of voice.

*Laryngoscopic Examination.*—One vocal cord lies fixed in a position midway between that of quiet respiration and phonation. The active

cord crosses the middle line in its attempt to meet the paralysed cord, and the arytenoid passes in front of that on the paralysed side.

*Diagnosis.*—It is liable to be mistaken for complete paralysis of one cord.

*Treatment* is the same as that for bilateral adductor paralysis.

(b) **Bilateral Adductor Paralysis.**—*Synonyms.*—Functional aphonia, hysterical aphonia, nervous aphonia.

*Etiology.*—Occurs most frequently in young females, and results from anemia, neurasthenia, shock, fright, uterine conditions, catarrh of the larynx, over-use of the voice, and toxic conditions, etc.

*Symptoms.*—Sudden in onset, whispering voice, with history of previous attack followed by sudden recovery, catarrh of the larynx.

*Laryngoscopic Examination.*—During respiration the cords abduct normally, but on phonation they remain immobile, and do not meet in the middle line.

*Diagnosis.*—By the presence of complete adduction of the cords during coughing or attempts at phonation.

*Prognosis.*—May disappear suddenly, or recovery may be slow.

*Treatment.*—Suggestion and tact, encouragement of talking and singing, faradic current. Attention to general health, tonics, *e. g.*, strychnine.

(c) **Paralysis of the Arytenoideus** (the transverse or central adductor) may occur alone, but is usually found in association with paralysis of other muscles.

*Etiology.*—These cases usually occur in connection with hysteria, but may result from acute or chronic catarrh of the larynx, voice strain, etc.

*Symptoms.*—More or less complete aphonia is present.

*Laryngoscopic Examination.*—Shows on phonation the vocal cords in contact in their anterior three-fourths, with a triangular space posteriorly. The front portions of the cords are brought together by the crico-arytenoidei externi and thyro-arytenoidei muscles.

*Treatment.*—Treat the accompanying laryngitis by suggestion, faradic current, tonics.

**Paralysis of the Thyro-arytenoideus Interna and Externa.**—*Synonym.*—Paralysis of the internal tensors and adductors of the vocal cords.

The paralysis of these muscles generally coexists with abductor paralysis. It is very rare for it to occur alone.

*Etiology.*—Hysteria, diphtheria, neurasthenia, laryngitis, voice strain, general debility.

*Symptoms.*—Weak voice and easily produced fatigue of voice on talking.

*Laryngoscopic Examination.*—The arytenoids are approximated by the arytenoideus muscle, and the cords adducted by the crico-arytenoidei laterales, but owing to paralysis of the internal tensors the vocal cords are concave with an elliptical space between.

**Paralysis of the Abductors of the Vocal Cords.**—(a) **Unilateral Abductor Paralysis.**—One posterior crico-arytenoid posticus muscle only is involved. The left vocal cord is more frequently paralysed than the right.

*Etiology.*—In the majority of cases due to pressure on, or injury to, one recurrent laryngeal nerve, or the trunk of the vagus, *e. g.*, by goiter, enlarged cervical (peritracheal) glands (Fig. 405), tumors in neck, malignant disease of esophagus, pleural adhesions, mediastinal tumors and glands, aneurysm of aorta (most frequent cause); not infrequently due to bulbar

lesions; occasionally to peripheral neuritis of toxic origin, also to infiltration of the abductor muscles by tuberculosis, syphilis, malignant growths.

*Symptoms.*—The voice is not affected in the early stage of unilateral abductor paralysis, but in the later stage when the cord is in the “cadaveric position” the tone and pitch are altered, and there is a tendency for the voice to crack. In many cases the voice improves owing to increased compensatory action of the normal cord, which extends across the middle line so as to meet the paralysed cord.

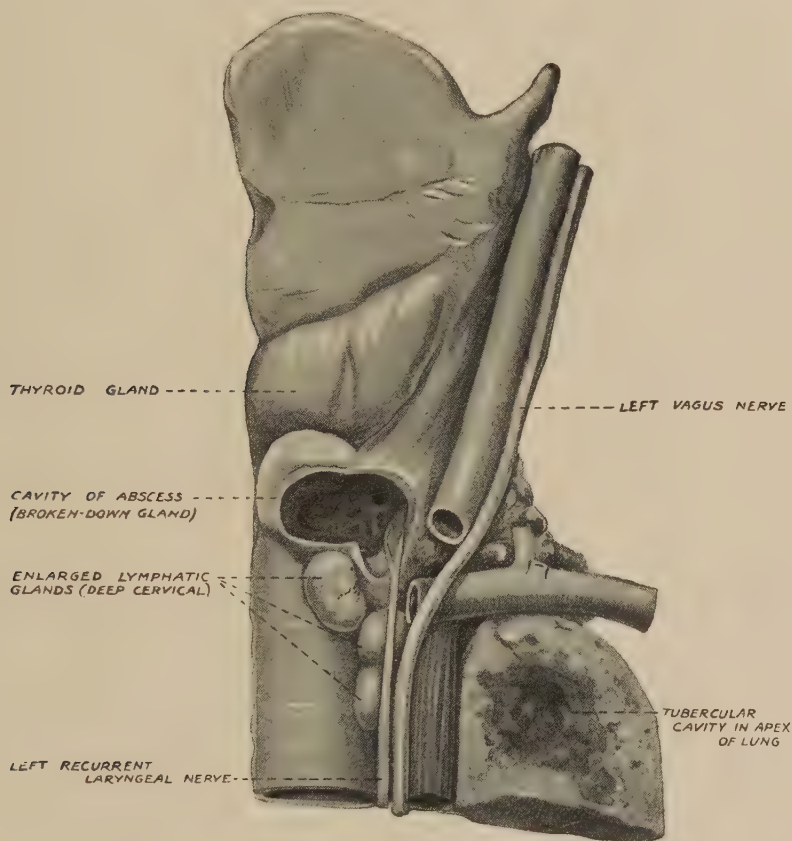


Fig. 405.—Left recurrent laryngeal paralysis due to involvement of the nerve in the abscess cavity of a broken-down peritracheal gland. (Irwin Moore's case.) The larynx in this specimen was split open from the front in the middle line to the thyroid alæ stretched apart. It shows very clearly the atrophy of the left vocal cord. Dissected by Professor S. G. Shattock, F. R. S., and now in the Museum of the Royal College of Surgeons, London.

*Laryngoscopic Examination.*—Shows, in the first stage—in which the abductor fibers fail first—the vocal cord immobile during ordinary respiration, and lying close to the middle line of the larynx, while on deep inspiration the paralysed cord fails to abduct beyond the “cadaveric position.” On phonation the paralysed cord approximates with the normal cord, hence the voice is not affected. This paralysis is the most common form met with, and is generally accidentally observed during routine laryngoscopic examination. When discovered it at once suggests either a thoracic aneurysm,

or some other pressure on, or damage to, the nerve trunk. The left cord is most frequently involved. In a later stage the paralysed cord is fixed in the "cadaveric position."

*Diagnosis.*—Bulbar and cervical lesions must be eliminated. Roentgen ray of chest and Wassermann reaction assist diagnosis. Must be differentiated from a cord impeded in its movements by infiltration, or interfered with by a growth.

*Treatment.*—Electricity. Removal of cause, *e. g.*, pressure of enlarged thyroid lobe. Re-education of the voice.

(b) **Bilateral Abductor Paralysis.**—A rare condition in which both the crico-arytenoidei postici muscles are completely paralysed.

*Etiology.*—Generally due to bulbar lesions (in 90 per cent. of cases locomotor ataxia), but may be caused by bilateral pressure on the recurrent nerve by metastases from mediastinal glands, etc.

*Symptoms.*—Expiration is normal, but marked dyspnea is present on inspiration, especially on exertion.

*Laryngoscopic Examination.*—The vocal cords lie parallel and immobile in the middle line of the larynx, in almost close contact, showing only a slight chink during respiration. On inspiration there is no separation of the cords, only a slight flapping taking place, with sucking inwards and downwards of the vocal bands and cords. Phonation is not interfered with because the vocal cords are in the phonatory position. If the paralysis is complete, which is extremely rare (Semon), and both cords become fixed in the "cadaveric position," the voice is reduced to a whisper.

*Diagnosis.*—The possibility of peripheral neuritis must be eliminated, also loss of movement caused by interarytenoid growths. The question of ankylosis of the crico-arytenoid articulation must also be considered and differentiated from a paresis.

*Prognosis.*—Unfavorable. The paralysis comes on slowly, and the danger is sudden death from asphyxia before tracheotomy can be performed. It has been suggested that many sudden deaths from cerebral apoplexy may be explained in this way, the stertorous breathing in apoplectic coma lending probability to the suggestion. Partial paralysis due to syphilis may disappear under treatment, or injury to the nerves during removal of goitre; so also some cases of toxic origin may recover. If paralysis is complete there is very little hope of improvement.

*Treatment.*—An early tracheotomy is indicated to avoid impending suffocation. If there is a history of syphilis, antisymphilitic treatment should be carried out. Removal of the thyroid isthmus—if caused by pressure of a goiter—may result in cure.

### C. INCOÖRDINATION NEUROSES

(a) **Spasmodic Laryngeal Cough.**—*Synonym.*—Laryngeal chorea.

*Etiology.*—Some local source of irritation, *e. g.*, adenoids and enlarged tonsils, nasal trouble.

*Symptoms.*—Short barking explosive cough, or laryngeal cry, continuing at regular intervals during day and ceasing at night. May continue for months or years.

*Diagnosis.*—Sudden onset, explosive character, no loss of voice, no expectoration, no shortness of breath. Direct inspection or mirror examination is necessary for diagnosis; inference may be misleading.

*Treatment.*—Attention to general health, hygiene, electrical treatment, tonics, *e. g.*, iron, arsenic, strychnine, cod-liver oil.

(b) **Laryngeal Vertigo.**—*Synonym.*—Laryngeal syncope.

A rare form of spasm of the larynx, transitional between a sensory and motor neurosis.

*Etiology.*—Unknown. Occurs in adults between thirty and seventy years of age. The use of alcohol and tobacco are considered to be predisposing causes. Has been found in those suffering from arthritis and arteriosclerosis.

*Symptoms.*—Irritation of the larynx with severe coughing attack and spasm of the glottis, simulating whooping-cough. Congestion of head and asphyxia, occasionally convulsion of face and extremities. Patient becomes unconscious or semiconscious and falls to the ground; followed by rapid recovery.

*Pathology.*—Uncertain. Regarded by some as allied to petit mal; by others as due to spasm of the glottis, or as analogous to the vertigo of Ménière's disease (Charcot). McBride attributes it to a closed glottis and forced expiration, followed by engorgement of the heart and large blood-vessels, and syncope.

*Diagnosis.*—Must be differentiated from the marked laryngeal spasm of whooping-cough.

*Prognosis.*—Favorable.

*Treatment.*—Attention to any reflex source of irritation. Bromides, chloral, trinitrine, etc. During attacks the inhalation of chloroform. (Allaying fears of the patient is helpful. The greater the effort the less air he inspires. Holding the breath will often cause the spasm to relax.—EDITOR.)

#### OPERATIVE METHODS FOR THE RELIEF OF STENOSIS OF THE LARYNX CAUSED BY BILATERAL PARALYSIS OF THE ABDUCTOR MUSCLES

Professor Hobday, in 1921, suggested that it might be possible to permanently reopen the air-way in cases of double abductor paralysis, by the operation of ventriculectomy, as carried out in the horse for "roaring" (unilateral recurrent paralysis). It has made one consider whether something more than tracheotomy cannot be done for those unfortunate people who suffer from this distressing condition, as that procedure is a life-saving device only and leaves the patient dependent for life on a cannula. Can the air-way in these cases be reopened and respiration through the natural passages be restored?

The adductor and abductor muscles of the vocal cords are supplied by the inferior laryngeal nerves, and a destructive lesion in the nerve, or in its centre in the medulla, paralyses first the abductor muscles and, later, the adductors. In double abductor paralysis it is the unopposed action of the crico-arytenoideus lateralis with paralysis of the crico-arytenoideus posticus and the thyro-arytenoideus which draws the cords into the laryngeal air-way and produces the stenosis; the cords become flaccid, will not separate, and their edges flap up and down. Complete paralysis of the recurrent nerves in which the cords lie in the cadaveric position, usually obviates urgent dyspnea and the necessity of tracheotomy, as there is enough air-way.

As previously mentioned bulbar lesions are the most common causes of this condition—99 per cent. of the cases are tabetic in origin. Of 53 cases of

abductor paralysis recorded between 1892 and 1898, only 6 developed complete recurrent paralysis. Of the remainder 22 had unilateral and 25 bilateral abductor paralysis, showing how small a chance there is that the cords will reach the complete paralysed stage (cadaveric position).

#### OPERATIVE TREATMENT \*

It is said that opening the air-way by operation should not be attempted in the first six months, as recovery may occur spontaneously. Chevalier Jackson advocates waiting a year; beyond that time recovery is no longer within the sphere of probability. But a disability attaches to certain operative procedures after a year's delay, because of the contraction and atrophy of the thyro-arytenoideus muscle, or fixation of the crico-arytenoid joint.

The following operations have been attempted in the past for the relief of this condition:

1. **Simple Division of the Recurrent Nerves, with the Object of Placing the Cords in the Cadaveric Position to Relieve the Stenosis.**—The results of this procedure have been very disappointing, and Chevalier Jackson regarded the operation as a failure in the one case on which he tried it.

2. **Re-establishment of Nerve Continuity by Resection and Anastomosis)** (a) *Without Transplantation.*—Suture of the recurrent laryngeal nerve has been carried out (as quoted by Chevalier Jackson), with an excellent result, in a case of unilateral paralysis recorded by J. Shelton Horsley and Clifton M. Miller. (b) *With Transplantation of the Recurrent Laryngeal Nerve with the Pneumogastric Nerve.*—Experiments on monkeys and goats by Ballance and Colledge, during 1924–28, in end-to-side anastomosis of the recurrent laryngeal nerve with the vagus, showed that tension returned to the paralysed cord, but no spontaneous movement. (c) *The Recurrent Laryngeal Nerve with the Descendens Noni.*—End-to-end anastomosis of the above nerves in luminals, by the same observers, showed that no movements occurred in the paralysed cord. Frazier (Philadelphia), at the suggestion of Chevalier Jackson, previously carried out a similar anastomosis in 2 patients with vocal cord paralysis. The results showed, as in the above experiments, that descendens noni recurrent laryngeal anastomosis is useless for the relief of paralysis of the vocal cord. (d) *The Recurrent Laryngeal Nerve with the Phrenic.*—End-to-side anastomosis of the above nerves in a monkey showed that it was possible to reproduce the normal movements of the vocal cord in tranquil respiration (Ballance and Colledge). Further, on the monkey being killed three years after operation, microscopic examination of the larynx showed complete recovery of the muscles and nerves concerned.

In June and July, 1926 E. Broughton Barnes and Sir Charles Ballance carried out an anastomosis of the recurrent laryngeal nerves to the phrenic, in a patient aged fifty-two years, suffering from double abductor paralysis, following removal of an adenoma of the thyroid gland. The patient was relieved of attacks of dyspnea from which she suffered, and one year after operation definite improvement occurred in the tone of the right vocal cord, with definite movement of the cord, which showed strong adduction, and abduction to one-third of the normal. The left cord remained almost

\* This paragraph, including some of the illustrations, was formerly published, in 1923, in the Proceedings of the Royal Society of Medicine, and Journal of Laryngology and Otology. It has been revised, added to, and brought up to date. (By kind permission of the Editors.)

motionless in the midposition with no abduction; due it was believed to tension at the site of anastomosis.

3. **Corpectomy (Excision of Vocal Cords Only).**—This has been carried out by (a) the indirect method with the laryngoscopic mirror, or directly with the aid of the autoscope of Kirstein; (b) by thyro-fissure. It has failed because of the excessive granulations which follow the operation, necessitating the reinsertion of the tracheotomy tube.

4. **Arytenoidectomy.**—This operation was employed by veterinary surgeons on horses (from 1834 to 1905) for the relief of "roaring" caused by paralysis of the left vocal cord. It was first performed on man by Ivanoff in 1911, the patient being a male with syphilis who had been tracheotomized and whose cords were abducted. Unilateral arytenoidectomy was performed, but later the cord became loosened, the obstruction recurred, and the cord had to be partially removed. In a few other recorded cases the results were also unsatisfactory.

5. **Ventriculectomy, or "Stripping" of the Lining Membrane of the Ventricle.**—In 1906 Williams (Cornell University, U. S. A.), finding that removal of the cords with their muscles in the horse predisposed to recurrence of stenosis, originated *per se* excision of the lining membrane of the ventricle. He excised the everted ventricular mucosa, putting out of action the adductor power of the vocal cords, which became attached, by cicatricial contraction, to the lateral wall of the larynx, in a position of forced abduction. Since that date veterinary surgeons have preferred ventriculectomy to arytenoidectomy in cases of recurrent nerve paralysis. In 1910 Professor Hobday considerably improved the operation by entering the larynx through the cricothyroid membrane only, and stripping both ventricles at the same time. Sargnon and Toubert suggested, in 1914, that ventriculectomy should yield equally favorable results in the human subject, and they concluded that while it was easy to perform in the horse because the operator could introduce the end of his index finger into the ventricle, it was very difficult to perform in man, even on the cadaver, and that in the human subject the only method applicable was by scraping and curettage of the membrane.

Vlasto, in 1921, contrasted the anatomy of the parts in man and in the horse, and showed that in the horse the ventricle and sacculus form one large cavity which extends downward below the vocal cord, and lines its outer surface, and that its attachments are very loose, so that it could easily be stripped off in one piece, leaving opposed raw surfaces, which, adhering, cause a lateral retraction of the vocal cord. In man the ventricle is very difficult to evert in the same manner. Hence he does not regard the operation as practicable or possible for the human subject.

Monselles (Florence), in 1900, attempted to reproduce eversion of the ventricle artificially in 33 human subjects after death, but without success, for in each case the superficial layers of the mucosa, instead of the whole thickness of mucous membrane, was torn in the attempt to draw out the lining membrane of the ventricle.

Piersol pointed out that in man the upper surface of the true cord slants only slightly downward and outward. It is, therefore, doubtful whether the superior and outer surface of the cord, if stripped of its membrane, is of sufficient extent to adhere to the raw surface of the lateral wall of the ventricle and so cause lateral retraction.

I have found, in operating on the cadaver, that the ventricle and sacculus cannot be everted and removed by the same method as in the horse; in every case the forceps tore through the mucosa. But by dissection it is

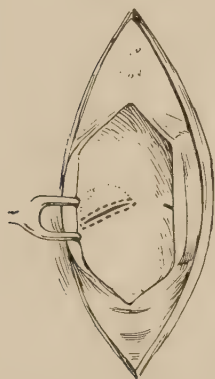


Fig. 406.—Ventriculectomy in man. A thyro-fissure has been performed. The broken lines show the incision through the mucosa surrounding the orifice of the ventricle. The dotted lines represent the outline of the ventricle together with the opening, anteriorly, of the sacculus.

possible to detach and remove the ventricular lining in one piece if, after complete dissection (Fig. 406), it is excised at its anterior attachment to the mouth of the sacculus, the sacculus being left *in situ*. It is very difficult to detach the sacculus, either with the ventricle or even after removal of the ventricle, because of the firm attachment of the suspensory ligament of Hilton, which slings up and supports the sacculus. And the mucous membrane over the inner surface of the sacculus is very thin, and so firmly adherent to the saccular wall that attempts at separation readily tear it. In only one case in 10 attempts was I able to remove the sacculus along with the ventricle.

If removal of the ventricular wall is ever attempted for treating abductor paralysis in man, it will probably be found advisable to remove only the ventricular lining, leaving the sacculus undisturbed so that it can continue its mucus-secreting function. With regard to adhesion of the rawed surface of the vocal cord to that of the lateral wall of the ventricle, it is suggested that temporary fixation of the vocal cord to the lateral wall by a suture through the vocal cord and thyroid ala and tied externally might encourage permanent adhesion and retraction.

#### 6. Evisceration or Ablation of the Vocal Cord and the Soft Parts Lining the Larynx.—

This can be carried out endoscopically or by thyro-fissure. Jackson has had one successful endoscopic case, and states that he finds "evisceration" by thyro-fissure ideal where no other lesion is present beyond the actual bilateral paralysis, though he considers that endoscopic evisceration is preferable (Fig. 407). Two of the patients he dealt with by the latter method had fairly loud voices and were permanently decannulated. In both of them not only the cord but also all its soft tissue was removed by dissection on both sides, leaving the perichondrium intact.

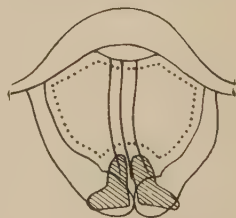


Fig. 407.—Evisceration of the vocal cords and soft parts lining the larynx. The dotted lines show the parts removed. (After Chevalier Jackson.)

7. **Ventriculocordectomy.**—This method, recently introduced by Chevalier Jackson for the cure of that form of stenosis of the larynx associated with bilateral recurrent paralysis, when the stenosis is due solely to the paralysis, can be carried out either endoscopically or by thyro-fissure. Excision is made (by punch forceps) of the vocal cord along with its supporting tissue forming the floor of the ventricle of Morgagni. He has carried it out endoscopically in 18 cases, under cocaine anesthesia, without operative mortality. In 11 cases the operation did not open the air-way because there was cicatricial stenosis, and other means had to be employed; in the other 7 there was a relief of the dyspnea.

Chevalier Jackson describes the technic of the operation as follows: "The larynx is exposed with the direct laryngoscope, and through it the punch forceps are inserted. The ventricular band is elevated, and the forceps applied as in Fig. 407a. Thus the floor of the ventricle and part of the mucosa of its outer wall are removed at one clip. A clean cut is necessary. The tissues must not be hacked. In some of the cases operated upon the ventricular bands were in tight apposition so that the forceps had to be insinuated between them before expanding the jaws. Great care is necessary to avoid getting too far outward between the thyroid and cricoid cartilages lest the crico-arytenoideus lateralis be injured. With the special forceps recommended this accident is easily avoided. Great care should also be taken not to excise any part of the arytenoid cartilage." Chevalier Jackson found that clipping off the extreme tip of the vocal process of the arytenoid was necessary, in some of his cases, be-

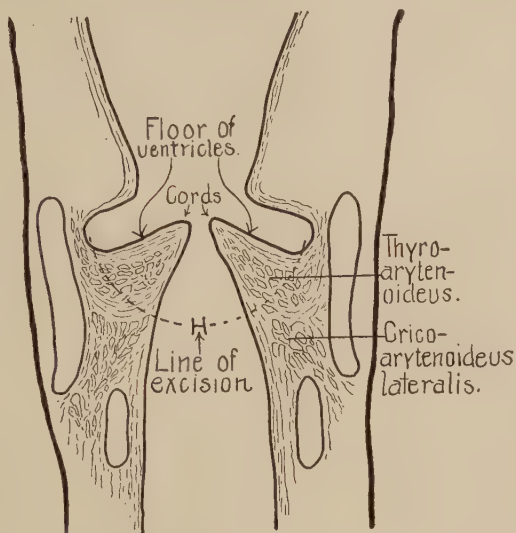


Fig. 407a.—Schematic drawing of coronal section of the larynx showing the tissues removed in ventriculocordectomy. (Chevalier Jackson.)

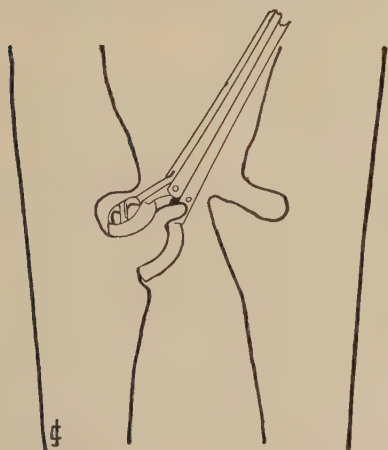


Fig. 407b.—Technic of ventriculocordectomy: The ventricular band is elevated and the forceps are applied beneath it to the cord and the entire ventricular floor anterior to the vocal process and antero-external surface of the arytenoid; the tissues are excised at one clip. (Chevalier Jackson.)

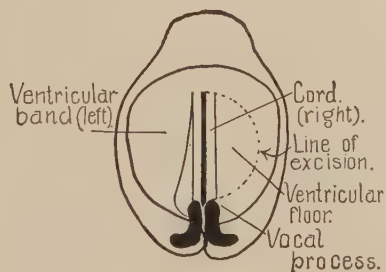


Fig. 407c.—Schematic drawing of tissue removed in ventriculocordectomy, as could be seen through the direct laryngoscope (recumbent position) if the ventricular band did not obscure the view of the ventricular floor. The removal is in rounded form and anterior to the vocal process. After healing of one side, the other side is similarly operated on. (Chevalier Jackson.)

cause of the shortness of the cords, but the excision of more than this is unnecessary, and should be avoided. The technic can be well understood from the illustrations (Figs. 407a-407d).

No after-treatment is necessary. The surface of the wound heals by granulation in from ten days to three weeks, after which the second vocal cord is operated upon.

Chevalier Jackson is of the opinion that, "considered in the light of the degree of preservation of the chief functions of the larynx, *i. e.*, phonetic, protective, and expectorative, ventriculocordectomy not only surpasses any previously devised operation, but is simply ideal for those cases in which neural and muscular atrophy has rendered resumption of normal cordal motility hopeless, by either spontaneous recovery or neuroplastic surgery." He however emphatically states that "if success is expected from ventriculocordectomy alone, its performance *must be limited* to cases totally free from cicatricial stenosis." Again he points out that to perform this operation education of the eye and fingers in endoscopic technic is required.

Howarth, in 1925, recorded 2 cases in which he had performed ventriculocordectomy, by suspension laryngoscopy, for double abductor paralysis

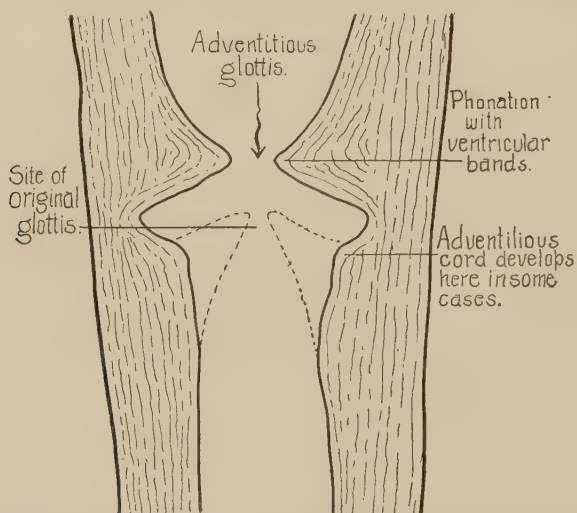


Fig. 407d.—Schema showing probable coronal cross-section of larynx after ventriculocordectomy, judging by appearances on mirror examination in cases in which there was no formation of an adventitious cord. (Chevalier Jackson.)

following operations on the thyroid gland. In each case he states that the immediate result was excellent, but the later results were unsatisfactory owing to fibrous tissue cicatrization and contracture.

**8. Anteroposterior Displacement of the Vocal Cords.**—Molinié (Marseilles), in 1913, suggested the possibility of deviating the paralysed vocal cords laterally from the mid-line by displacing the anterior attachments inward (anteroposteriorly). On the cadaver he made an incision through the mid-line of the thyroid cartilage without opening the laryngeal cavity, *i. e.*, he did not incise the internal perichondrium. Then two similar lateral incisions were made on each side 5 mm. from the mid-line incision, leaving two loose lateral pieces of cartilage attached to perichondrium, which could be pressed inwards towards the center of the larynx and kept in position by a steel band, so reducing the anteroposterior diameter of the laryngeal box and causing deviation of the cords towards the lateral wall of the larynx (Fig. 408). Thus in the cadaver he obtained an ovoid glottis. Thinking he

could get the same result in a living subject, Molinié carried out the operation in a male patient aged twenty-five who had bilateral abductor paralysis. It failed, and a tracheotomy tube had to be reinserted.

9. **Cordopexy or Anterolateral Transplantation of the Vocal Cord.**—In 1922 Wilfred Trotter suggested to me that it might be possible to reopen the obstructed air-way by transferring one, or both, of the paralysed cords from the middle line to the lateral wall of the larynx. He proposed that an incision should be made transversely across the middle line of the thyroid cartilage, a retractor inserted, and the larynx opened, so as to obtain a good view of the anterior insertion of the vocal cords. Having located these, the portion of cartilage to which the cords are attached might be separated from the thyroid ala by a circular incision, drawn forward and carried laterally along the transverse incision through the thyroid ala. On removing the retractor, the separated halves of the thyroid cartilage would come together and fix the cords in their new position (Fig. 409). Following out these suggestions on the cadaver, I found that the approach to the larynx by a transverse incision was not satisfactory, as one cannot accurately locate the anterior insertions of the vocal cords, which are separated from those of the false cords by only 2.5 mm. Later, the possibility presented itself of locating the origin of the vocal cords from the outside and reaching them through a small incision in the middle line of the thyroid cartilage. Piersol pointed out that the true vocal cords arise a little above the middle of a line extending from the bottom of the thyroid notch to the lower border of the thyroid cartilage. Taguchi showed that in men the average distance from the notch to the vocal cord is 8.5 mm., and from the lower border of the thyroid cartilage 10.5 mm. (in women the distances are 6.5 and 8 mm.). In both sexes the distance between true and false cords is 1.5 mm.

I thought that it might be possible to ascertain the anterior insertion of the cords from the outside of the larynx. After careful measurement, an incision  $\frac{1}{4}$  inch in length was made through the center of the middle line of the thyroid cartilage, and a bent probe inserted to locate the point of attachment of the cords (Fig. 410). Then a piece of cartilage (forming a triangle with the previous incision) was excised, to which, I concluded, the anterior extremity of the cord was attached. From the apex of the triangle another incision was carried laterally through the thyroid ala for  $\frac{1}{4}$  inch. The separated triangular piece of cartilage was then drawn forwards and carried laterally between the cut halves of the thyroid ala. When the larynx was opened in the middle line, it was found that it was the ventricular band

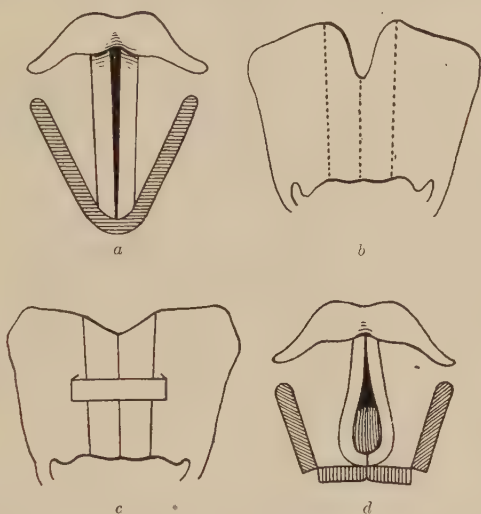


Fig. 408.—Molinié's operation of anteroposterior displacement of the vocal cords.

which had been transferred, not the true cord, so that one found that accuracy in defining the true cord could not be assured in this manner; and that this could only be obtained by first performing thyro-fissure.

In my next attempt, thyro-fissure was first performed, a triangular piece of cartilage being excised (along with the attached cord). I found that the anterior end of the true cord was not as free as in the other attempt, and that it could not be transferred laterally on account of the attachment of the perichondrium and muscular fibers of the thyro-arytenoideus. However, elevation of the perichondrium in the vicinity re-

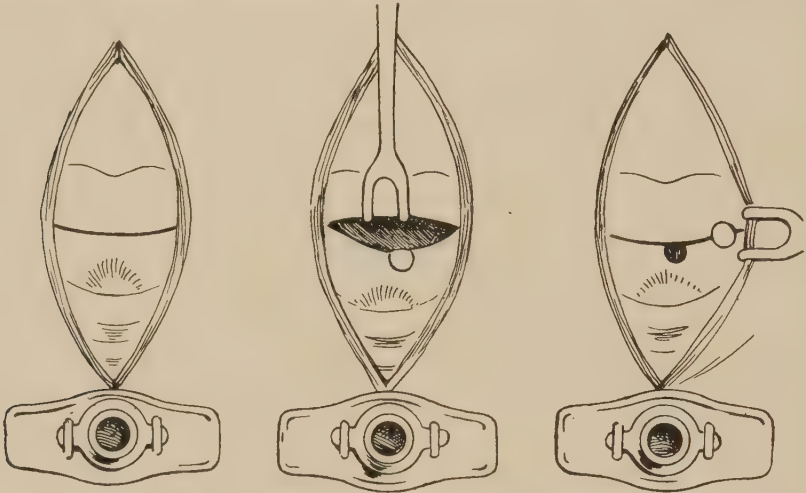


Fig. 409.—Operation as suggested by Wilfred Trotter.

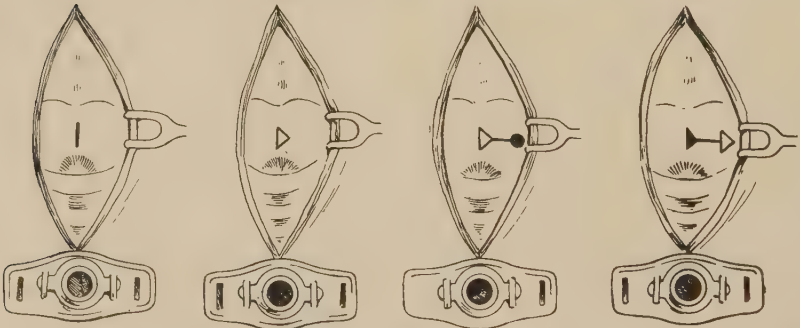


Fig. 410.—Cordopexy or anterolateral transplantation of the vocal cords. Shows the attempts by the author, which failed, to localize the anterior insertion of the cords without a thyro-fissure.

leased the cord and permitted the piece of cartilage, with the attached cord, to be easily drawn along the horizontal incision and anchored (Figs. 411, 412). Professor Shattock pointed out to me that strangulation of the tissues of the cord might ensue because of their being tightly gripped by the cut edges of the cartilage. To avoid this, a small half-circular piece of cartilage was punched out at the extremity of the horizontal incision, forming a circular opening in which the cord could lie. By this operation the vocal cord, or cords, could not only be displaced laterally, but also shortened anteroposteriorly, thus increasing the tonicity of the paralysed cord tissues. The

transference of a cord  $\frac{1}{4}$  inch from the middle line was sufficient to fix it in the position of complete abduction (Fig. 413).

The cause of failure in abductor paralysis, to reopen the air-way by placing the cord in the cadaveric position by means of simple division of the recurrent laryngeal nerve, is probably due to the shortening (contraction) and atrophy of the adductors (see Fig. 413), owing to the period during which they have remained contracted without any antagonism, in combination with ankylosis of the crico-arytenoid joint, through want of use. The paralysis comes on slowly, and takes a long time to develop, attention only being drawn to the condition when the dyspnea threatens.

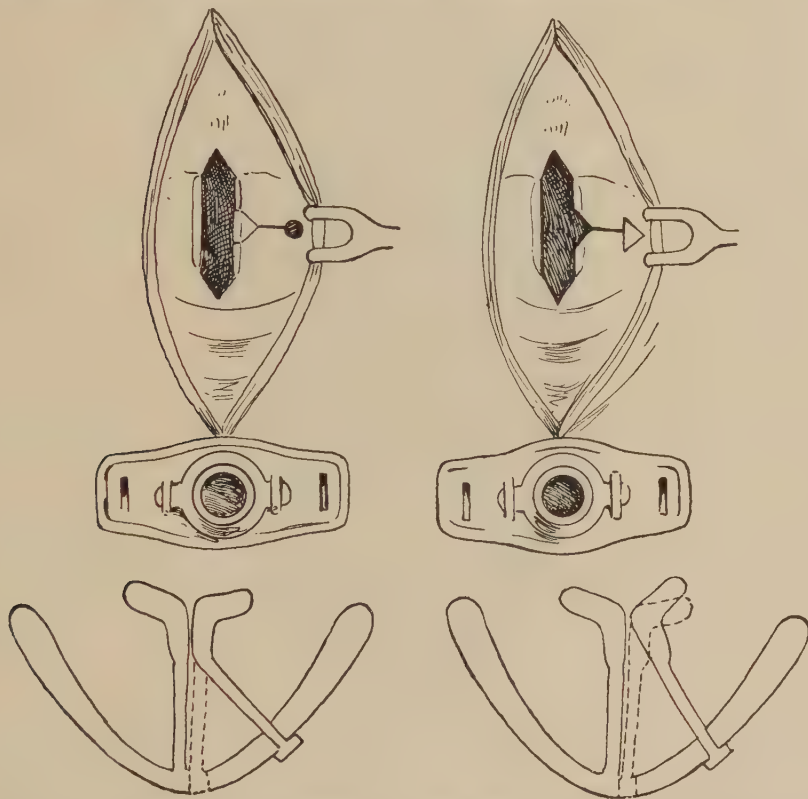


Fig. 411.—Cordopexy. As devised and suggested by the author (Irwin Moore). Shows in the upper figures a thyro-fissure followed by anterolateral displacement of the vocal cord, and in the lower figures the glottic space obtained if the crico-arytenoid joint is mobile, or if the joint is fixed. It is obvious that when both vocal cords are transposed laterally the glottic space secured is double that shown in the lower figures.

These pathological factors must be taken into account in connection with attempts at re-establishment of nerve continuity either by direct or transplantation anastomosis. The recent work and observations of Balance and Colledge would therefore appear to be only applicable in *recent* peripheral cervical lesions, *e. g.*, cases in which the recurrent nerve has been accidentally severed during thyroidectomy operations.

In unilateral abductor paralysis the question of anastomosis does not arise, since sufficient air-way is present without causing dyspnea. We know that bulbar lesions, as previously stated, are the most common causes of

double abductor paralysis, hence the cases must be very limited in number in which nerve anastomosis is applicable.

In the majority of cases the problem would appear to be purely a mechanical one, and it is suggested that it can be best dealt with by such an operation as cordopexy.

A few days after presentation of these investigations and experiments to the Royal Society of Medicine, in February, 1923, Musgrave Woodman (Birmingham) carried out this operation of cordopexy, according to my directions, on a girl, aged fifteen, who was suffering from double abductor paralysis, following division of the recurrent nerves during removal of both

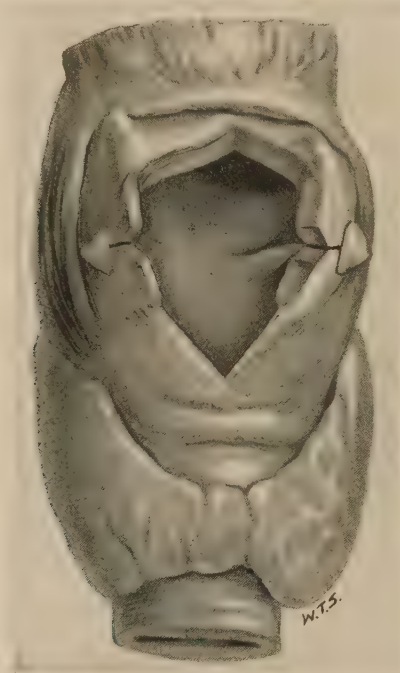


Fig. 412.—Cordopexy. Larynx removed and dissected after the operation of cordopexy has been carried out on the cadaver. Shows, after a thyrofi ssure, the anterior extremities of both vocal cords transferred laterally from the midline of the larynx.

lobes of a very large goiter. Though a considerable amount of dyspnea was present, and a good deal of coughing followed the operation for two or three days, the patient made a good recovery, and the result was reported to be satisfactory, the vocal cords remaining in their newly placed position.

**The Voice After Operative Procedures.**—In cases of double abductor paralysis we have to deal with a very small glottic chink, which is closed more completely on inspiration; there is strong inspiratory dyspnea. The voice is normal or nearly so, but there is an accompanying stridulous noise. It is only in the later stage, when the abductor muscles are affected, that the voice is much altered.

Chevalier Jackson says it was believed that permanent loss of voice followed excision of the cord, but 2 patients on whom he operated had a fairly loud voice, though a rough one, and speech was mainly in a mono-

tone. Jackson also says that in all the 7 successful cases of ventriculo-corpectomy the voice was "louder than an ordinary whisper, and loud enough to carry on an ordinary conversation in a reasonably quiet room."

**Conclusions.**—If we have an operative procedure by means of which a permanent cure of stenosis in cases of double abductor paralysis can be guaranteed, the natural air-way reopened, and the patient decannulated, ought we not to carry this out in the early stage of the paralysis, even at the expense of some voice impairment, rather than wait the uncertain occurrence of the complete stage of paresis, when the cords might possibly attain the

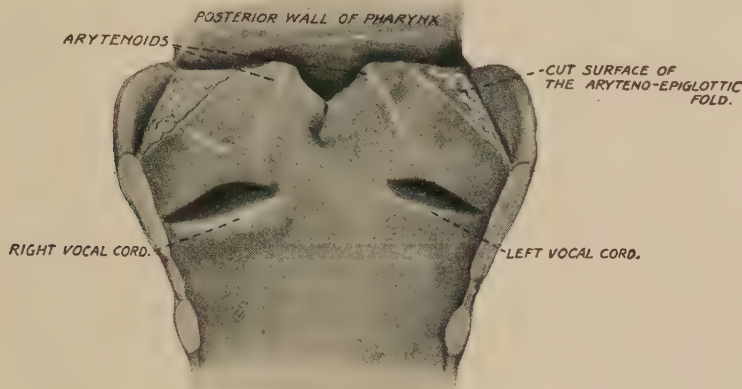


Fig. 413.—Left recurrent laryngeal paralysis. The larynx in the specimen (Fig. 405) has been split open from the front in the middle line and the thyroid alæ stretched apart; shows very clearly the atrophy of the left vocal cord.

cadaveric position? Disappearance of the paralysis in bilateral abductor paralysis is very rare, though it has been reported in a case due to syphilis, and in a few cases to which Jackson refers. Gleitsman (New York) states that there is only one known case of recovery from bilateral recurrent paralysis.

IRWIN MOORE.

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## INJURIES OF THE LARYNX

Injuries of the larynx are unusual on account of the intrinsic elasticity and mobility of the larynx, and the protection which is afforded by the mandible.

**Dislocation of the Larynx.**—Dislocations of the cricothyroid and cricoarytenoid articulations have been described but are exceedingly rare.

**Concussion of the Larynx.**—Severe dyspnea, syncope, and death have resulted from the application of trauma to the larynx without the presence of a demonstrable solution of continuity.

**Treatment.**—Theoretically, artificial respiration, and possibly tracheotomy are indicated.

**Wounds of the Larynx.**—Incised or stab wounds of the larynx are usually self-inflicted or are the result of attempted murder. In recent years these wounds have been incurred by broken glass in automobile accidents. Self-inflicted incised wounds are usually transverse and may involve the hyothyroid membrane, the thyroid cartilage, or the cricothyroid space.

**Symptoms** consist of bloody frothy expectoration and frequently the presence of emphysema. Dyspnea may be caused by the flow of blood into the trachea, by edema, by submucous hematoma, and by abscess. Wounds of this type gap widely, and the lower flap may be drawn into the larynx during inspiration and create respiratory obstruction.

*Diagnosis.*—A diagnosis is made by the presence of the wound and the symptoms mentioned.

*Treatment.*—The chief necessity in the treatment of wounds of the larynx is the maintenance of a free passage of air to the lungs and the arrest of bleeding. Tracheotomy should always be performed if there is the slightest embarrassment of respiration.

If the operation is in the hands of a skilful surgeon who is careful to completely obliterate all dead spaces primary suturing of incised wounds of the neck with associated injury to the larynx or trachea will often succeed. On the other hand it will probably be better in average hands if only the deep structures are sutured, leaving the muscles and superficial layers to be closed at a later date. The space should be packed with gauze and after the deep tissues have united secondary closure should be done. In this way extensive subcutaneous emphysema can be prevented and certainly secondary wound infection will be minimized. In an experience which began in 1892, with a patient who developed enormous areas of emphysema following railroad injuries to the chest, up to the present time I have never seen a supuration of an emphysema although I know that cases have been reported. The use of suction for the removal of blood from the trachea is of great value.

**War Wounds.**—Treatment of war wounds of the larynx in no way differs from treatment of wounds received in civil life except that primary closure of a projectile wound of the soft parts of the neck, trachea or larynx, is seldom possible. Furthermore, owing to the irritability of the air-passages, chemical sterilization of the wound is impracticable. The wounds heal slowly, even after repair by suture, as infection usually supervenes.

**Fractures of the larynx** are more common in adult life and are seen usually in men. Either direct or indirect force may be the cause of these injuries. Slight fractures, such as fissures or simple fractures, may escape attention and be treated as simple contusions of the neck. Gunshot wounds of the larynx are considered compound fractures; usually the larynx is splintered and the injury is associated with varying degrees of trauma to the vocal cords. On account of its greater prominence and its projecting angle, the thyroid cartilage is the one usually injured. Any or all of the cartilages, however, may be involved. Lateral compression or antero-posterior pressure against the vertebral column, with flattening of the sagittal diameter, is the contributory cause. The fracture may be in any direction or may affect the cornua only. Many of these fractures are transverse; frequently they are comminuted and even compound. Fracture of the cricoid cartilage is unusual. A single fracture is generally dorsal; multiple fractures are scattered. The arytenoids may be loosened from their attachment to the cricoid, which produces a relaxation of the vocal cords. Combined fractures of the thyroid and cricoid cartilages are rare. The associated injury of the soft parts is liable to be extensive. The external perichondrium may rupture and effusion of blood occurs within the larynx and infiltrates the surrounding tissues. Accompanying injuries may occur to the great vessels of the neck, the vagus nerve, hyoid bone, mandible, and other structures in the anatomical neighborhood of the injury. The mucosa may be torn, which allows the passage of air into the cellular tissues of the neck and results in great swelling and emphysema. Portions of cartilage may be exfoliated. Danger of infection along the respiratory tract and mediastinum is always present.

*Symptoms.*—Any or all of the symptoms mentioned below may be present. There may be deformity, abnormal mobility, cartilage crepitus, and dysphagia. In almost all instances marked shock occurs, and consciousness is temporarily lost. Pain is severe especially when coughing or swallowing or on manipulation. The voice is rough, harsh, or may be absent. Cough, usually of the convulsive type, is a frequent occurrence. Hemoptysis is an unfavorable symptom. Dyspnea may occur and be most pronounced. Asphyxia may appear early and lessen; later become still more marked and stridor develop. The patient is apprehensive of choking to death and becomes cyanotic. The pulse is small and the skin pale and moist. Extension of the edema and ecchymosis of the neck, which are caused by infiltrating blood, will increase the respiratory embarrassment. Emphysema is a most serious symptom and the tissues of the entire body may be invaded; this symptom sometimes appears with fulminating rapidity, but may be delayed in cases where the injury is not severe. To this clinical picture must be added the classical symptoms of fracture.

*Diagnosis.*—Laryngoscopic examination may show swelling, congestion, hemorrhage, or the extent of the interference with respiration. A skiagram should be taken and will generally reveal the fracture. When the diagnosis is doubtful the injury should be treated as a fracture.

*Treatment* consists in maintaining a free passage of air to the lungs. To this end a tracheotomy should be performed in all cases where a diagnosis of fracture is made. When the patient is seen soon after the accident, in shock and often unconscious, with face and neck swollen and purple, tracheotomy should be performed at once. The obliteration of anatomical landmarks by the effusion of blood and air makes the procedure more difficult.

Dr. Chevalier Jackson recommends a low tracheotomy, as this is much less apt to be followed by stenosis. If the trachea must be opened high, a more deliberate lower opening should be made for the insertion of the tracheotomy tube. Dr. Jackson advises splitting open the entire front of the neck exactly in the midline so as to obtain a large wound in which to feel for the trachea. Aspiration of the tracheobronchial tube in order to draw out the blood and mucus should be instituted if respiratory movements fail to follow the tracheotomy. This is accomplished by introducing through the cannula a catheter to which an aspirator is afterward connected.

Expectant treatment is indicated in the absence of deformity, depression of the fragments, hemorrhage, emphysema, or where laryngoscopy reveals no encroachment on the lumen of the larynx. The patient should be placed in a quiet room and the atmosphere kept moist and warm. All effort to use the voice must be prohibited. Opiates are usually indicated. Nourishment and fluids must be supplied by rectum. Constant surveillance should be maintained. Sudden severe dyspnea and suffocation may develop and patients should never be placed beyond the reach of prompt assistance.

*Prognosis.*—The prognosis is generally grave. A fatal outcome is attributable to suffocation; this may be caused by edema, an accumulation of blood in the lungs, mediastinal emphysema, or aspiration pneumonia. As a rule death is due to interference with respiration and may occur immediately or may be delayed for sometime. If delayed it is caused by infection or pneumonia. Very late disturbances are ascribed to a reparative narrowing of the lumen of the larynx.

Bronchoscopy may be indicated for the removal of firm clots. Bronchoscopic insufflation of oxygen is invaluable after clearing the air-passages. Similarly, artificial respiration may be resorted to after the air-passages are clear.

In the event of recovery, hoarseness may persist for a long time and the voice may be permanently altered. The tracheotomy tube may be necessary for an indefinite period on account of stenosis. Practically all cases of laryngeal stenosis can be cured, however, by a laryngostomy, which can be successfully closed later by a plastic operation.

*Anesthesia.*—As a rule general anesthesia is contraindicated, any form of local anesthesia being preferred.

CHARLES F. NASSAU.

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## ACUTE LARYNGITIS

**Definition.**—Acute laryngitis may be defined as an acute inflammatory involvement of the mucosa of the larynx and vocal cords—characterized at onset by deep-seated burning or tickling irritation in the larynx, sense of heat or warmth in the hypopharynx, distressing feeling of fulness in breathing and later indicated by hoarseness, dry cough, difficult or painful phonation or complete aphonia. It is usually an accompaniment of acute infectious epiglottitis and in turn is often associated with an acute infective type of tracheitis. Its definition must necessarily be a comprehensive one, as it is usually secondary to other foci of infection, varies greatly in severity of involvement, and is frequently associated with other types of disease which are given priority in diagnosis.

Its synonyms under common usage include simple acute laryngitis, acute catarrhal laryngitis, and laryngorrhea.

**Pathology.**—Acute laryngitis represents an acute congestive reaction, customarily a localized peripheral disturbance as distinguished from one of neural or central origin. It is an acute inflammatory reaction to an irritant applied locally, one conveyed in lymphatic or blood-stream, or may be a combination of these.

Pathologically the congestion is accompanied by leukocytosis and round-celled infiltration in the mucosa and underlying tissue. The exudate is at first scanty and watery. The whole lumen of the larynx appears somewhat obstructed and movements of muscles and cords a little sluggish. There is often some interarytenoid fulness without ulceration and examination itself is irritating.

If the acute course be brief, the inflammation subsides in thirty-six or more hours, though hoarseness, discomfort in phonation, and deglutition continue somewhat longer, and the swelling in the looser area of ventricles and between arytenoids is more gradual in resolution. In the early stage the mucopurulent secretion does not become so evident or profuse, but if phlegmonous stage follows, the whole surface may be bathed in secretion, more or less opaque from the number of leukocytes thrown out. This secretion tends to accumulate in the intercommissural areas, as well as above the free borders of the cords. With this progressive edema of deeper

tissues there is a marked degeneration of the mucous glands and increased exfoliation of surface epithelium. Secretions become thicker and often streaked with blood from superficial ulceration. This becomes a careful point in differential diagnosis as it may be mistaken for deep-seated tuberculous or malignant lesion.

**Etiology.**—Acute laryngitis is almost invariably secondary to invasion from infective foci in the nose and throat above. The etiological factors are of particular importance because of the so frequently correlated involvement of the entire rhinopharyngeal tract, sometimes including the nasal accessory sinuses, and because acute laryngitis so frequently tends to repetition. This may be irregular or seasonal with regular recurrence in spring and autumn and consequent tendency to chronicity. Its prognosis, therefore, becomes dependent both upon the elimination of antecedent factors and successful combating of the insidious reinvasion. Such regulation of causal factors becomes of vital importance in the public speaking and singing professions. Auto-intoxication, intestinal toxemia, vicious metabolism, and defective elimination are causes.

An intelligent analysis of hygienic, systemic, focal, and coexistent disease factors is the only effective means of directing treatment.

**Hygienic.**—Under hygienic factors should be included irregular habits of bathing, eating, and sleeping, inattention to action of bowels and kidneys, bodily exposure particularly of extremities, abuse of alcohol or tobacco, inhalation of noxious vapors, occupational contact with finely dusted particles, irregular wearing or changes in clothing, climatic exposure, and overwork. All these predispose to the disturbed vasomotor balance, inviting laryngeal attack.

**Systemic** conditions which lower general vitality are near the hygienic borderline and include intestinal toxemia, poor vasomotor balance, metabolism disturbance, cardiac and renal insufficiency, hepatic torpor, and such organized factors as syphilis, tuberculosis, and the exanthematous diseases.

**Focal.**—The focal sequence from higher respiratory areas is a logical one; diseased faucial tonsils or any part of the Waldeyer ring, crowned and defective dentition, sinus engorgement, chronic discharging ears, general infective adenitis. It might be noted, however, that some authorities believe that the squamous band in the mesopharynx aids in the barrier to contiguity of tissue infection from the higher nasopharyngeal tracts. Distal foci, such as the gall-bladder, appendix, and prostate, may also follow the circulatory stream path.

An acute catarrhal invasion, always apt to follow strain, exposure, prolonged fatigue, overheating, and more especially the sudden chill, would likely attack here, in an individual with susceptible larynx.

**Coexistent Pathology.**—Coexistent disease with access from blood-stream or lymphatic transportation must confuse the etiological picture. Extension through tissue continuity or contiguity, irritation from toxins elsewhere elaborated or simultaneous exposure to bacterial shower, are important to consider. A retropharyngeal abscess in the hypopharynx, acute inflammation in thyroid or adjacent deep collar of cervical lymphatics may both have etiological significance and obscure the diagnosis. Perhaps should be included in this list, involvement of the thymus or other structures in superior mediastinum.

The bacterial flora of the rhinopharyngeal tract—*Micrococcus catar-*

PLATE VII



1. Chronic laryngitis with tuberculous involvement of left arytenoid and inter-ventricular space.



2. Acute laryngitis with swelling of both arytenoids.



3. Marked edema especially encroaching on anterior commissure.



4. Atrophic chronic laryngitis with hemorrhagic ulcerations on the cords and adjacent laryngeal areas.



5. Hypertrophic laryngitis with vocal cords almost obscured.



6. Acute laryngitis with hemorrhages involving cords and interarytenoid spaces.



rhalis, streptococci, and staphylococci of varying strain, bacillus of influenza, pneumococci, and incidental presence of varied other types, aid the toxic weakened background. The actual inciting cause will sometimes be clear but this would not be effective in producing the disturbed metabolism balance of the so-called "cold," without the basis of weakened resistance. The spasmodic type in children, while often sequent to systemic toxemia or adjacent focal infection perhaps more often follows an immediate exposure from sitting or playing on damp, cold surfaces, or meeting streptococcic laden strata of air. Predisposition and heredity play their part and these attacks are frequently recurrent.

**Clinical Picture and Symptomatology.**—The clinical picture of acute laryngitis is the same as acute invasion of mucous surface elsewhere. At onset the mucous surface is congested from dilatation and engorgement of the capillaries, surface is red, dry, and glazed with more scanty secretion than in the similar invasion of the nasopharyngeal tract. The larynx appears congested, cords are of a pinkish hue, and there is a slight fulness in the ventricular areas and often punctate hemorrhages appear on the surface.

On account of the difficulty in early irritative stage to obtain adequate mirror examination, subjective symptoms of cough, disturbed phonation, embarrassed respiration, local pain, and fulness may have to be relied upon for diagnosis. An application of weak solution of cocaine and effort to obtain short tones in the upper register will "train" the larynx to some success in examination. In addition to the rose tint and evident thickening of the cords, a somewhat tardy or sluggish movement of the cords will be noted, due to mechanical infiltration impeding muscular movement, or degenerative changes in the nerve-endings; and there may be a temporary paresis of the crico-arytenoid muscles, both groups, enough to account for the aphonia. Frequently small surface ulcerations yield crusts and coagula of blood may come away. In the acute dry type, laryngitis sicca, these brownish-gray crusts may be present in considerable quantity, almost simulating the deposit of membrane. In the rheumatic type of acute laryngitis, pain is increased, there is tenderness to external pressure, general depression and weariness are more pronounced.

In the supraglottic invasion a ring of congestion often appears on the epiglottis, the relaxed interarytenoid and ventricular areas become swollen and tinged, the early proliferative changes become exudative as the disease progresses and the exudate itself lighter in color but more dense. In the subglottic involvement, stenosis becomes marked, stridor is evident in both inspiration and expiration, and bilateral reddened sausage-like masses often appear below the cords.

**The Picture and Symptomatology of Acute Laryngitis in Children.**—The pseudomembranous croup is the more formidable type. The child, usually of the strumous, even the status lymphaticus type, with exposure during the day, or particularly the evening before, awakens in the night with hoarse, stridulant breathing, embarrassed in both inspiration and expiration. There is muscular retraction in varying degree in the supra-sternal notch, epigastrium, and lateral borders of the neck. At least a suggestion of cyanosis is present, and this is often marked. The diagnosis is clear: Metallic cough, stridor, voice if present, muffled and deep toned, cyanosis, and rapid pulse. Temperature is often 104° to 105° F. If sub-

glottic, as in the severer types, tracheotomy may become necessary, but the attack will probably permit sufficient time for tranquil type of procedure.

**Diagnosis.**—Diphtheria will be recognized by the type of membrane and bacterial culture, though doubtful cases may be returned negative for the first one or two cultures.

Foreign bodies should give history of sudden suffocative attack and onset without other antecedent etiology.

Perichondritis of cricoid area may confuse the borderline between laryngitis and tubercular involvement, but elimination of the latter will be aided by chest examination, microscopic study of sputum, and study of the neighboring glands.

Swelling below the cords in "false croup" cases in children adds to the seriousness of obstruction. The secretion coughed up may be streaked with extravasated blood in the more congested types, and areas of ecchymosis may be seen. Paroxysms of coughing by increasing the tension on the mucous surface produce these slight hemorrhages and ulceration may follow.

**Treatment.**—The treatment for acute simple laryngitis should include first such general measures as keeping the patient absolutely in bed, imposing complete rest of the voice and maintaining an even room temperature of about 70° F. Hot mustard foot-bath and hot lemonade, moderate catharsis by administration of calomel, 1 grain in divided doses, followed by saline and enema if necessary. Increased elimination by bowels, skin, and kidneys are fundamentals in the treatment. For this reason opiates are contraindicated (Jackson).

It will be an advantage to have continuous steam in the room, to which may be added either compound tincture of benzoin, spirits of camphor, menthol, turpentine, or creosote. Ordinary pan or croup-kettle may be employed if not too irritating to the patient, or the croup-tent. In the initial stage cold water compresses for brief periods, or the ice-collar may be used to advantage, say for two or three hours at a time. The use of ephedrine or menthol and camphor sprays, by relief of the congestion in the upper passages, may negatively ease the situation. Gargles do not reach the larynx, chlorine vapors have been tried, with still uncertain success. The topical application of even low percentage silver salts probably irritates more than aids.

If in spite of the above measures edema of the larynx or adjacent structures supervenes, these areas could be scarified with guarded laryngeal scarifier, to produce free bleeding and relieve obstruction.

In threatening edematous, phlegmonous, or membranous type, stenosis and dyspnea render the situation suddenly serious and one should be prepared to relieve by intubation or, preferably, tracheotomy if emergency demands. Tracheotomy should always be low regardless of the thyroid isthmus. High tracheotomy is the commonest cause of chronic laryngeal stenosis and difficult decannulation. The cardinal signs of obstructive laryngeal dyspnea are given elsewhere in this book.

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## ACUTE INFECTIVE LARYNGOTRACHEITIS

**Definition.**—The association of terms in this title is appropriate because the course of invasion of acute infection of the trachea is normally from the upper respiratory foci downward, and must also involve the larynx and immediate subglottic area.

This may be (a) in the rapid progression of a typical influenza with onset of coryza, diffuse hyperemia, and acute rhinopharyngitis; (b) the streptococcic complications so frequently secondary to measles and scarlet fever, or a mixed infection which is first pronounced in its subglottic laryngeal and tracheal symptomatology. With invasion of the latter group, unexplainable cough of the so-called "nervous type" so often assigned to vague pharyngeal or laryngeal influence, presupposes the discovery of at least some subacute tracheal pathology. Epidemic, highly infective types are usually termed *influenza*; they confer immunity lasting a few months or a year (Chevalier Jackson).

**Etiology.**—Occurrence in acute epidemic periods with other systemic symptoms makes the etiology self evident and its danger dependent upon the relative immunity of the individual and upon the virulence of the infecting strain.

The upper nasopharynx and higher postnasal spaces harbor septic foci and will yield active culture of varied bacterial flora—hemolytic and non-hemolytic streptococci, pneumococci of varying type, diphtheroid and influenzal bacilli, or more mixed types, as the case may be. The upper respiratory passages with their accessory sinus chambers may frequently serve as focal carriers during an apparently quiescent state.

Any acute congestive obstruction to rhinopharyngeal drainage and intranasal ventilation, including block to normal sinus outlet is an important contributing factor. Lowered resistance from faulty metabolism, the association of other systemic disease, coexistence of malignant growths, syphilis, or tuberculosis are significant alike in etiological predisposition and diagnostic difficulty.

**Pathology.**—In acute laryngotracheitis of infective origin there is a universal hyperemia and injection involving both mucous and submucous areas. The surface secretion will probably be scanty at first, the glandular structure is hyperactive as secretion increases and this becomes thicker from increased mucin, desquamated epithelium, lymphatic cells, and pus. There is a marked increase in polymorphonuclear count without much fibrin element. Here and there surface erosion takes place, discharge is streaked with blood or even frank hemorrhage may occur. The hyperplasia may become so marked, especially in acute mixed type of infection, as to seriously encroach upon the lumen of the subglottic larynx and upper trachea and necessitate surgical relief for stenosis. The turgescence, erectile-like tissue in this subglottic area predisposes to such result.

**Symptomatology.**—Its symptomatology, somewhat dependent upon causative factors outlined, is subjectively a feeling of contraction, burning or fulness behind and just above the sternum, often tender to pressure in the upper tracheal and laryngeal areas. Cough is a very early symptom, dry, irritating, and often spasmodic enough to suggest pertussis. The strident inspiration, however, may occur as often during as at the end of the spasm of cough and emesis be absent. There is a feeling of soreness along

the trachea and both breathing and deglutition may be painful. The cough perhaps fails to relieve the mucus in early stages and sonorous râles suggest obstructed lumen through walls thickened by tenacious mucus. Later expectoration though not excessive is more free and there is considerable amount of mucopurulent secretion. Definite pain may be experienced just below the clavicle and auscultation show the raucous breathing increased in approaching the substernal area. In subacute or more chronic states when patient remains up, cough increases after rest in warm bed at night until mucus is relieved and considerable accumulated mass is cleared away in the early morning. Singers find early day singing difficult and voice timbre is changed.

Objective symptoms seen by mirror examination in a favorable throat or by bronchoscopic study show the general hyperemia and injection, red granular surface, though in later stages mucous area may become paler and covered by a layer of mucopurulent secretion. The active area seems in posterior membranous surface, though mucus may be seen hanging from anterior and lateral walls. In consequence of this posterior loose-fold arrangement, and stream of secretion coughed out, the posterior laryngeal commissure may retain and show an excess collection.

**Treatment.**—The most important treatment in acute laryngotracheitis is absolute rest in bed, with free elimination by bowels and kidneys. Room should be of even temperature, use of voice forbidden, diet semiliquid and nutritious. Supportive and diaphoretic remedies are indicated. Beyond inhalations (steam or benzoin) the free use of paraffine oil and intermittent cold compresses in acute stage, topical local treatment is irritative and of no positive value. Applications of local astringents should be avoided. Authorities agree on the significance of the inspiratory indrawing of the suprasternal notch, epigastrium, and closing of rib interspaces as a diagnostic indication, regardless of cyanosis, for prompt tracheotomy interference. (The agonizing strangling cough is promptly arrested by alkalinizing the tracheobronchial secretions by the internal administration of sodium bicarbonate (3 grams) every three hours. Opiates will not give the same relief and are injurious.—EDITOR.)

From its vulnerable position and frequent bombardment, the trachea would seem on the whole to carry a special resistance and immunity, and if given a chance will aid in spontaneous reconstruction.

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### CHRONIC LARYNGOTRACHEITIS

Influenza, whether due to Pfeiffer's tiny Gram-negative organism, aided by some non-filtrable virus, or stimulated by other symbiotic relation, is essentially an upper respiratory tract invasion.

The high fever, body pain, and toxemia with coryza and rhinopharyngitis, may in twenty-four hours proceed downward into the larynx and trachea, with customary tracheal fulness, painful deglutition, irritating cough, and embarrassed breathing. After acute symptoms have run their

course a chronic type may persist for one or two, or many weeks, with very characteristic secretion. Chevalier Jackson has admirably described this type of tracheal sputum, as a gray, opalescent, tenacious, more or less globular mass, with darkened, mottled color like moss-agate, often projected like a "ball of putty," and more scanty than in most bronchial or pulmonary diseases. He has aptly termed this "*moss-agate tracheal sputum*" (Figs. 414, 415) characteristic of *chronic tracheitis* of any origin. The soot-like discoloration is not from soot or black dust in the inspired air, but is from the blood-pigment (Chevalier Jackson).

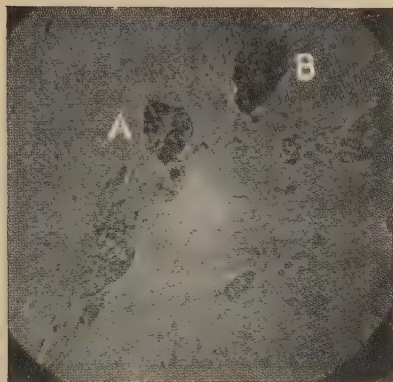


Fig. 414.—Moss-agate sputum from a case of subacute tracheitis in a man aged fifty years. Endothelial cells showing granules of hemosiderin. The cells at *A* and *B* are very heavily loaded with the granules. (Chevalier Jackson, Moss-agate Tracheal Sputum, Penna. Med. Jour., Proceedings, Meeting of the Penna. State Med. Soc., October, 1921.)

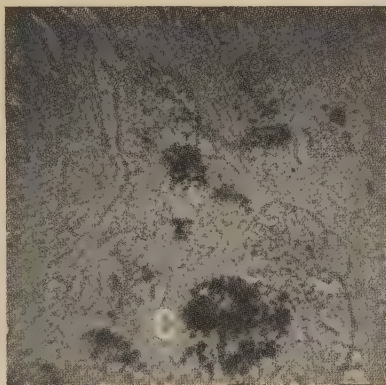


Fig. 415.—In the lower part of the illustration (*C*) is seen a group of endothelial cells, of distinct outline, containing large and small granules of hemosiderin. (Chevalier Jackson, Moss-agate Tracheal Sputum, Penna. Med. Jour., Proceedings, Meeting of the Penna. State Med. Soc., October, 1921.)

The **treatment** here should particularly emphasize absolute rest in bed and avoidance of all irritating or astringent applications. Silver nitrate is particularly contraindicated.

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## LARYNGOTRACHEITIS DUE TO WAR GASES

War gases, which were first used by the Germans in 1915 as one of the most dreadful inventions of the 1914-18 conflict, are not all to the same extent of interest to the laryngologist. His attention, however, must be attracted by two of these gases, namely, "arsine," a diphenylarsine chloride, and especially "yperite" or "mustard gas," a dichlorethyl sulphide. Arsenine and yperite derive their laryngological interest from the importance of the lesions which they cause at the level of the upper respiratory tract, especially of the larynx and trachea, and from the sequelæ which they produce. These two gases cause troubles and lesions quite

similar, if not identical; but, bearing in mind that yperite was more frequently used than arsine, the laryngotracheitis of yperite victims will be taken as a type for our clinical study.

**Clinical Study.**—Yperite intoxication is characterized by three main groups of symptoms:

1. *Ocular troubles* (keratoconjunctivitis) appear first, as early as six to twelve hours after exposure to the gas.

2. *Upper respiratory symptoms* follow on the third, fourth, or fifth day, with bronchopulmonary complications in severe forms.

3. *Skin manifestations*, namely, vesications, conspicuous especially in covered regions such as the inner aspect of the thigh and the genital organs. These symptoms are usually early, sometimes even immediate. In some cases, however, they appear only from the fifth to the tenth day.

**Functional Signs.**—Dysphonia marks the onset of laryngotracheitis, and reaches its maximum in a few hours; it is an intense hoarseness hardly modified by productive cough and, in almost one third of the cases, it leads to complete aphonia, which subsides after a few days, giving place to a very persistent raucity of the voice. Cough is almost constantly observed. It is paroxysmal, sometimes whooping, and occurs chiefly at night. Hoarse and tracheal in tone, after a lapse of two or three days, it becomes productive, and the copious expectoration is mucoid or mucopurulent, aerated, acid, and occasionally blood-streaked. Yperite victims complain of an uncomfortable *sensation of irritation*, a tickling or burning which is often unbearable, definitely localized to the larynx and beneath the sternum. This sensation is generally of short duration, but may last several weeks.

Not infrequently these laryngotracheal signs are associated with nasal and pharyngeal symptoms: (a) A mild form of coryza of short duration (about ten days) without sinus complications; (b) slight but recurring epistaxis; (c) early and persistent anosmia and agnosia; (d) moderate and rapidly subsiding dysphagia.

An important bronchopulmonary symptom of the intoxication is a pulmonary type of continuous dyspnea without any laryngeal difficulty in breathing whatever.

**Objective Signs.**—1. *Mild Form.*—This variety is characterized by an extensive congestion of the whole mucosa of the upper respiratory passages, which recalls the aspect encountered in grippal laryngotracheitis. The soft palate, the pillars, and the posterior wall of the pharynx are red. Indirect laryngoscopy, which is rendered difficult by violent pharyngeal reflexes, also reveals a diffuse redness of the laryngeal mucosa, and streaks of mucopus. The epiglottis, aryepiglottic folds and arytenoids are slightly edematous. In the interarytenoid space the mucosa is folded and velvety, thus preventing the perfect coaptation of the cords in their posterior segment. Likewise, the ventricular bands are slightly tumefied. Large, intensely red, or simply greatly vascularized, the cords on contraction appear to be crescentic (paresis of the internal thyro-arytenoid muscle). Mirror examination may also detect hyperemia of the trachea.

2. *Severe Form.*—This variety is characterized by ulcerations of peculiar aspect, site and evolution. The oropharynx and soft palate are intensely congested and present ecchymotic patches with small, sloughy areas, either isolated or multiple, somewhat similar in aspect to mucous patches. A favorite site of these sloughy ulcerations is the base of the uvula, where

they appear to be oval in shape, of about the size of a pea, and sometimes double and symmetrical.

Mirror examination of the larynx is exceedingly difficult and shows a dark red mucosa. Ecchymotic streaks and areas of ulceration are also noticed on the epiglottis, aryepiglottic folds, ventricular bands and cords. These ulcerations appear on the third or fourth day, vary in shape and extent according to their site, and contrast markedly, by their ivory-white leukoplakia-like aspect, with the intense redness of the neighboring mucosa.

In very severe cases the ulcerations are covered by a diphtheroid membrane, in a few hours by a false membrane such as is observed in burns due to caustic. These may be encountered in any part of the larynx, but their most common site is the vocal cords, where they have peculiar characteristics. Very commonly, oval-shaped ulcerations are met on the free margin of the anterior two-thirds of both cords. Generally they last up to about the twentieth day, but the voice remains altered for a long time, during which the cords present a more or less marked redness and a slightly thickened free margin.

These laryngeal lesions are accompanied by paretic phenomena due usually to involvement of the thyro-arytenoid muscles, together with, not infrequently, the interarytenoid muscle.

Exceptionally, the tracheal mucosa may be inspected in these cases. It is found to be the site of lesions similar to, although not quite so conspicuous as, those encountered in the larynx.

**General Symptoms.**—Elevated temperature, rising to 39° and 40° C., and a more or less involved general condition, are met in the severe form.\*

**Course.**—The healing of lesions requires from fifteen days to a month; congestion gradually disappears from the larynx and pharynx as the laryngo-tracheal secretions dry up. In severe cases slow resorption of exudate sometimes leads to ulceration of the mucosa.

**Prognosis—Sequelæ.**—Aside from often fatal pulmonary complications, the prognosis of laryngotracheitis in victims of war gases is good, "quo ad vitam," but grave so far as sequelæ are concerned.

1. *Hoarseness and Aphonia.*—Victims of yperite and arsine are usually left with an extremely delicate upper respiratory tract, hoarseness recurring, with accompanying objective signs of catarrhal laryngitis. Others are permanently hoarse and subject to persistent cough; in these cases, mirror examination of the larynx reveals red and tumefied ventricular bands masking more or less pinkish and swollen vocal cords.

Complete aphonia is far from being an infrequent sequela of war-gas laryngitis; its chief causes are the heretofore described lesions of the ventricular bands and vocal cords, and myositis of the crico-arytenoid and cricothyroid muscles, as seen especially in cases of arsine intoxication. Furthermore, so-called "*sine materia*" aphonia has been observed. Its prognosis is much more favorable. Of nervous origin, this condition appears at the very onset of the intoxication, notwithstanding the almost complete integrity of the larynx. It is relatively more often encountered in "arsine" cases, and has almost always responded to adequate treatment, namely: gradual re-education and strychnine. Nervous aphonia due to

\* Histobacteriological examinations of specimens taken from patients whose death had been caused by yperite intoxication, have shown ulceration, false membrane, and a great number of microbes, with streptococci predominating.

laryngeal paralysis is entirely different; it is undoubtedly more important and generally resists all treatments.

2. *Cicatricial Lesions and Laryngeal Stenosis*.—Seldom observed, such sequelæ result especially from intoxication by yperite, and in cases where, in the acute stage, the mucosa was the site of deep and extensive sloughy ulcerations. Stenosis, a rather late sequela, manifests itself by suffocating attacks and finally by a laryngeal type of continuous dyspnea, which, if threatening, may necessitate surgical treatment.

3. *Chronic Tracheitis*.—This common inflammation is a frequent, but hardly troublesome, sequela of war-gas intoxication. It fortunately responds to thermal therapeutics, as do the usually co-existing bronchopulmonary sequelæ.

4. *Tuberculosis*.—Primary tuberculosis has several times been detected in the larynx of gas victims, although it has not been possible, so far, to say to what extent the action of gas was a predisposing factor to the development of the Koch's bacillus infection.

**Treatment.**—1. *Laryngotracheitis*.—The treatment has two chief indications: relief of the painful irritation and disinfection of the upper respiratory tract. The nasal and pharyngeal cavities are disinfected with non-irritating antiseptic oils, salves, gargles, and collutories to which the addition of a little cocaine appears to be of advantage. Soothing inhalations, very slightly antiseptic (eucalyptol, gomenol), or alkaline sprays, are indicated for the pharynx.

Irritation of the larynx and painful cough are relieved by the insufflation of analgesic powders. Lubet Barbon recommends the following formula:

Morphine chlorhydrate	} Equal parts
Boracic acid	
Gum arabic	
Lactose	

Intratracheal injections of weak solutions of gomenolated oil, under the control of indirect laryngoscopy, are used with gratifying results for the disinfection of the trachea and bronchi.

2. *Sequelæ*.—A sulphurous thermotherapy is sometimes remarkably effective. Exercises in phonetic re-education and general stimulation by strychnine are to be considered.

FERNAND LEMAITRE,

ROBERT MADURO.

(Translated by Edmond Aucoin.)

## LARYNGEAL COMPLICATIONS IN GENERAL DISEASES

Laryngeal complications occurring in the course of certain general diseases merit careful consideration, since their recognition at an early stage may not only afford relief to an already sick patient but at times may prevent the development of the most serious of all sequelæ, suffocation. Possibility of laryngeal involvement in the following diseases should ever be kept in mind and no time should be lost in applying remedies for the alleviation of symptoms before the need for tracheotomy arises.

**Exanthemata.**—The group of infections comprising the acute exanthemata (measles, scarlet fever, smallpox, and chickenpox) must be carefully watched for laryngeal symptoms. With measles the condition usually takes the form of an acute laryngitis evidenced by huskiness of the voice or aphonia, and rarely leading to edema, ulceration, or abscess. In scarlet fever the laryngeal manifestations are usually inflammatory extensions from the pharynx, occasionally resulting in edema of the glottis in cases complicated by nephritis. The same is true of smallpox and chickenpox. In all these the treatment should be anti-inflammatory and directed primarily to the pharynx before extension below has taken place. Gargles, sprays, inhalations, and topical applications to the pharynx are all useful, particularly direct irrigation with a solution consisting of  $\frac{1}{2}$  dram each of sodium chloride, sodium bicarbonate, and sodium borate to 4 ounces of water. The mouth should be rinsed and a gargle used after each feeding in order to prevent accumulation of, and infection from, food particles.

**Bronchial Inflammations.**—Such diseases as influenza, bronchitis, tracheitis, and pertussis often lead to a laryngeal irritation from excessive coughing and expectoration, sometimes to real laryngitis from extension upward, resulting in fibrinous inflammation, submucous infiltration, and even perichondritis, and paralysis. Treatment should be directed to the initial condition and to relief of the laryngeal irritation by use of vapor inhalations of eucalyptus, compound tincture of benzoin, or creosote. Further consideration of these conditions will be found in other parts of this book.

**Laryngeal Spasms.**—Certain diseases are at times complicated by a spasmodic contraction of the laryngeal muscles resulting in more or less complete respiratory block for a varying length of time. (See Spasm of the Larynx.)

**Laryngismus Stridulus.**—*Synonyms.*—Abductor spasm, false croup, spasmodic croup, and glottic spasm.

*Etiology.*—This condition occurs in children between the ages of three months and two years, complicating intestinal disorders, rickets, nasopharyngitis, and malnutrition. It is considered to be due either to a spasmodic affection of the nervous system or, according to Sir St. Clair Thomson,<sup>3</sup> to a collapse of unusually feeble laryngeal tissues which, on being sucked in by a strong inrush of air, occlude the glottic opening. This condition maintains until absorption of air in the lung raises the CO<sub>2</sub> level, resulting in stimulation of the respiratory center and opening of the glottis. Attacks tend to be initiated by prolonged and inadequate nasal breathing such as that occasioned by adenoid obstruction.

*Symptoms.*—The attacks come on quite suddenly, usually at night, waking the child up from a sound sleep, and producing the appearances of intense dyspnea, with violent efforts to expand the chest, clutching at the throat, and cyanosis. This crisis may last from a few seconds to minutes, when the attack usually terminates with a deep inspiratory crow and a sudden inrush of air into the lungs. Convulsions may occur, death but rarely. Following this spasm the child is quite comfortable until seized with a further attack in a short time or at a considerable interval. Such severe seizures are less common than milder ones in which the child appears to be merely holding its breath. During the intervals there may be a residual cough but laryngoscopy fails to show any characteristic laryngeal pathology.

*Diagnosis.*—A typical attack of sudden glottic spasm in an otherwise essentially well child, particularly at night, is not likely to be confused with any other laryngeal condition. Foreign body should be ruled out. Whooping-cough and acute catarrhal laryngitis are usually accompanied by cough, fever, expectoration, or loss of voice. True croup or diphtheria is of more gradual onset and presents a characteristic malaise, prostration, and hoarseness. In bilateral abductor paralysis the glottic closure is constant but incomplete, while in laryngismus stridulus it is complete but not constant. Inferential diagnosis is misleading and dangerous. Direct laryngoscopy (*q. v.*) is the only certain means of diagnosis. The larynx of any child can be examined without an anesthetic, general or local.

*Prognosis.*—In fairly healthy children the attacks cease as suddenly as they begin and tend to decrease in frequency with advancing age.

*Treatment.*—Immediate treatment of an attack should consist in sitting the child up, slapping it on the back, induction of sneezing by tickling the nose with a feather, partial immersion in a hot mustard bath, or inhalations of chloroform, ether, or ammonia. Cold water may be applied to the face, and the tongue drawn forward by introduction of the finger into the mouth. Hypodermic injection of adrenaline or rectal instillation of choral hydrate may be tried.

Between attacks a sedative like bromide will be helpful together with general treatment of the underlying cause. Tracheotomy may rarely be required to avert suffocation.

**Tabes Dorsalis; Laryngeal Complications.**—Of a similar nature are the laryngeal crises in tabes dorsalis, the result of a bilateral abductor paralysis. Here, however, simple spasm may be succeeded by a true obstruction resulting in death by suffocation unless immediate tracheotomy is performed.<sup>9</sup> An analogous condition is sometimes seen in bulbar paralysis and hysteria. (See Index for further consideration of this subject.)

**Typhoid Fever; Laryngeal Complications.**—While not common the laryngeal complications of typhoid fever require close attention, and are to be regarded as a potential source of serious and even fatal sequelæ.

*Etiology.*—The condition may be due to: (1) the typhoidal toxemia on which are superimposed the traumatic effects of phonation, coughing, and swallowing or exposure to cold or draught; (2) an arterial or venous thrombosis; or (3) infection, as evidenced by the recovery of typhoid bacilli from ulcerations caused by the blood-stream infection.<sup>8</sup> The laryngeal mucosa may also be bathed in infected secretions from the septic mouth contents and food remnants, particularly in the presence of a lessened cough reflex.<sup>9, 10</sup>

*Pathology.*—The mucous membrane of the larynx becomes dry, the voice weak, hoarse, and uncertain, presenting the picture of a severe catarrhal laryngitis and leading in some cases to ulceration and perichondritis (*q. v.*). This may be followed by extensive necrosis of the cricoid and arytenoid cartilages resulting even in complete destruction and exfoliation.<sup>9</sup> Epiglottic ulcerations are usually superficial and necrosis here as in the thyroid cartilage is rare. Such ulcerations, either superficial or deep, and occurring usually on the postero-internal wall, probably result from the disintegration of lymphoid follicles harboring typhoid bacilli, although simple dryness of the mucous membrane, especially on the posterior wall, may be the initial cause. The time of laryngeal involvement is usually at

the height of the disease and at a period when ulceration of the ileum is likely to occur.<sup>8</sup> Paralysis of the laryngeal muscles may be due to a local neuritis.<sup>9, 10</sup>

*Symptoms.*—The symptoms, the onset of which may be quite insidious,<sup>8</sup> are local pain, dysphagia, sudden aspiration of food into the respiratory tract, hoarseness, irritable cough, and aphonia. A perforation of an ulcer has been known to lead to general emphysema. Sudden supervening edema of the glottis will require tracheotomy which should never be delayed and for which emergency one should be prepared as soon as the slightest symptoms of laryngeal obstruction are noted.<sup>9</sup> Any of these should indicate a mirror examination of the larynx and a close observance of their course. Early treatment should consist in rigid oral hygiene and inhalations of benzoin vapor.<sup>8</sup>

**Erysipelas of the Larynx.**—Erysipelas of the head and neck may be accompanied by an edema of the larynx, manifested by hoarseness and dyspnea due probably to external pressure and inward extension of the inflammatory infiltration of the neck. Tracheotomy may be necessary.

**Rabies and Leprosy of the Larynx.**—Two rare conditions subject occasionally to laryngeal involvement are rabies and leprosy. In the former dysphagia and a husky voice may be followed in the excitable stage by spasm so severe as to extend to the trachea and resist even tracheotomy. In the latter the symptoms are due to involvement of the mucous membrane resulting in hoarseness or aphonia.

**Pemphigus of the Larynx.**—Laryngeal pemphigus is almost without exception an accompaniment of pemphigus of the skin, beginning with a bleb-like spot under the mucous membrane, varying in size from a flax seed to a small bean. An elevation of the epithelium follows the formation of blebs, which are marked by large yellow patches, later more or less covered with a white secretion, an appearance characteristic of pemphigus. This yellow covering is later cast off and the base of the affected area becomes red and granular, usually healing without loss of substance but sometimes leading to cicatricial formation with fusion to the soft palate. The diagnosis, prognosis, and treatment call for consultation with the dermatologist.

**Other Conditions.**—A variety of conditions in external structures affect the larynx by pressure. Such are retropharyngeal abscess, enlarged cervical glands, and hypertrophy of the thyroid.

Mention should also be made of a few miscellaneous conditions, such as edema due to over-administration of potassium iodide,<sup>9</sup> muscular weakness in myasthenia gravis, acute edema in Bright's disease, and angioneurotic edema. In all these as in the more important conditions much may be accomplished by the realization that laryngeal symptoms are danger signals which unheeded may lead to disaster but which heeded in time are usually quite amenable to local treatment.

LYMAN RICHARDS.

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## THE RELATION OF THE THYROID GLAND TO DISTURBANCES OF THE LARYNX

Disturbances within the larynx have been attributed to the hypertrophy or to some other disease of the thyroid gland. After investigation, however, it would seem that the relation of disturbances of the thyroid to the larynx



Fig. 416.—Malignant growth of the thyroid gland, involving the recurrent laryngeal nerve with its branches. A, Esophagus; B, malignant adenoma; C, recurrent laryngeal nerve.



Fig. 417.—Hypertrophy of the thyroid gland. The recurrent laryngeal nerve is not involved. Note its relation to the esophagus: A, Esophagus; B, hypertrophied lobe of thyroid; C, recurrent laryngeal nerve.

has been over-estimated. The so-called "saber trachea" may result from pressure, but even extensive enlargements of one or both lobes of the thyroid which displace the larynx and trachea apparently have no effect on the voice.

Individuals with hyperthyroidism may suffer from hoarseness or weakness of the voice, but this condition is due to the same tremor and muscular weakness as that which affects the entire body musculature, such as is seen when the fingers are extended, rather than to a disturbance of the innervation of the larynx.

If the lobes of the thyroid are enlarged they may project into the pharynx from behind or laterally and they may be large enough to obstruct the larynx. However, the recurrent laryngeal nerve is more intimately related to the esophagus than to the trachea and therefore it is not subject to firm pressure from these enlargements.

A benign enlargement of the thyroid gland does not cause laryngeal paralysis, so that when laryngeal paralysis is present in a patient with a non-malignant enlargement of the thyroid gland, the cause of the paralysis should be sought for in the mediastinum or elsewhere. On the other hand, bilateral abductor paralysis with the cords in the midline in an individual with an enlarged thyroid should always provoke the suspicion that a malignant growth of the thyroid is present, as the nerve usually becomes involved in the malignant infiltration.

In surgical operations on the thyroid gland it is very easy to draw the recurrent nerve out of its bed in making traction on the gland and to subject it to trauma in securing hemostasis. Postoperative injury may occur by infection or the nerve may become involved in scar tissue.

W. V. MULLIN.

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## CHRONIC LARYNGITIS

**Definition.**—Chronic laryngitis is a term broadly used to cover chronic non-specific inflammatory diseases of the larynx.

**Pathology.**—Chronic laryngitis includes several types of fixed tissue change—differentiated by the stage of inflammatory progress—hyperplastic, trachomatous, even perichondritic. While acute laryngitis represents the early stage of surface change, chronic laryngitis adds to this, involvement of connective tissue, glandular and vascular layers. This in its simplest form is merely a general hyperplasia of all structures, cords, ventricular area, arytenoids and interarytenoid space, with injection and loss of luster. Its story is one of pathological sequence—diffuse and continuous hyperemia (either primary or secondary to attacks of acute laryngitis); general hypertrophic change succeeded by overdevelopment of fibrous tissue; transformation of greater or smaller areas from ciliated to squamous epithelium; loss of glandular secretion; retention of inspissated masses in the larynx; gradual disappearance of blood-supply, and finally, more or less atrophic stasis, with occasionally somewhere through these stages the permanent laryngitis nodosa and perhaps the laryngitis sicca or dry stage with scanty secretion and difficult expulsion.

Areas of cartilage may become denuded and resultant ulceration occur with formation of adhesions, or ankylosis of movement—the Roentgen ray may reveal general sclerosis and premature osseous change.

**Etiology.**—The etiological factors in production of the chronic laryngeal stage are so manifold, yet so closely correlated, that it is difficult to assign distinctive priority. These may be as patent and simple as injury, or the excessive use of tobacco or alcoholic spirits, failure to check a simple, persistent evidence of cold, unfavorable climatic environment, misuse or abuse of the voice in singing, or occupational demands for continued forceful speech, perhaps even the prevalent habit of inordinate and organized athletic cheering. From whatever cause, it would seem to particularly occur in individuals whose resistance is low.

The sequence may be as obscured as in the passive hyperemia consequent upon cardiac and hepatic disorders, or hematogenous changes due to irritation at any point along the upper respiratory tract. Auto-intoxication and endocrine disturbance have also been emphasized.

Any factor producing a chronic diffuse hyperemia in the laryngeal tract may be the primary cause of chronic laryngitis, such as focal disease of the sinuses or tonsillar ring of lymphoid tissue, mouth-breathing with its resultant irritative and infected air, diseased dentition and infection of the neighboring glands, repeated low-grade attacks of tonsillitis or influenza, the constant clearing of the throat in hypopharyngeal hypertrophy, overfulness and infection in the lingual tonsil area, occupational exposure to dust and débris.

Tuberculosis, syphilis, and incipient malignant disease must be included as well as the debility and toxemias secondary to diphtheria, typhoid, and pneumonia.

**Symptomatology and Clinical Picture.**—The symptomatology is in part expressed by a review of its causative factors, and will necessarily review some of the above mentioned phases; this is particularly true where symptomatic pathology is concerned.

In the initial stage, that of diffuse hyperemia, subjectively expressed by feeling of fullness and heat in the laryngeal area, a constant desire to cough, perhaps strangling in type, speech somewhat of an effort, and voice easily fatigued, the objective picture is one of general redness of all intralaryngeal structures—arytenoids and interarytenoid spaces, true and false cords, and the ventricular pouch between. The epiglottis may share in the disturbance and become stiffer than normal, as well as thickened. The false cords above may become so full as to occlude the view of the true vocal bands. This hyperemia or redness will be rather a rose color shading to gray and may be patched or slightly serrated in appearance.

Interlaryngeal moisture may be varied from an almost exudative state to complete dryness, and the cough will somewhat follow this variation. Temperature and pulse-rate will not be particularly affected, at least not indicative; surface of mucous membrane loses its normal luster and infiltration around the intrinsic muscles may be sufficient to partially inhibit the function of the cords.

The succeeding hypertrophic change tends to manifest itself in three locations or phases: (1) That invading all the upper areas of the larynx; (2) the subglottic type of involvement; (3) small approximating nodules—the so-called “singers’ nodes.”

1. *In the first type* congestive symptoms are accentuated with pronounced tendency to aphonia, difficulty in breathing, and dysphagia.

To the patient this may give the feeling of a fixed or movable foreign

body, and the strangling type of cough will be increased in the effort of expulsion. An arched and serrated appearance of the cords may occur and simulate the picture of paralysis.

2. *In the second or subglottic variety* suffocative symptoms occur often to the borderline of intubation or tracheotomy necessity, but less threatening and spasmodic than in acute laryngitis. Secretions become troublesome, accumulating in the interarytenoid space, and are expelled with difficulty.

3. *In the third type* nodes appear at the position formed by the junction of middle and anterior thirds, sometimes in the posterior segment of the vocal cords. Besides causing hoarseness, these render voice placement difficult, shorten tones, and may even misplace register.

If this normal succession be too long continued, and not interrupted by more organized vascular or neurological events, there will follow the inevitable sequence of overfibrosis and diminution of blood-supply—an atrophic termination. The physiological picture now changes and the arytenoid areas and cords lose normal luster and clearness, become pallid with dark discolored secretions, frequently present a sticky, tenacious, and inspissated appearance. The intralaryngeal picture becomes one of dry, atonic, and sometimes wrinkled appearance, and plugs expelled with difficulty may rest in the anterior and posterior commissures.

Fortunately, for diagnosis, ulceration does not usually take place and tuberculous exclusion aids in the diagnosis. In this atrophic condition, however, with its dry and glazed appearance, crusts, and strings of mucus may be confusing.

In this stage the throat is uncomfortable with a constant sense of dryness, burning or pricking, and cough is consequently expressive—dry, hacking and irritative in character. Attacks resembling croup with strangling may occur, dependent on the blocking from dried secretion and naturally worse at night. These exacerbations will resemble the more acute type.

**Diagnosis.**—Differential diagnosis in advanced states of chronic laryngitis must eliminate syphilis, tuberculosis, and malignant growth. The Wassermann test is primarily important for diagnosis of syphilis. Tuberculosis is usually secondary to similar involvement in lungs; this, however, may have become quiescent, and painful deglutition, loss of weight, and smear examination will help to decide.

**Treatment.**—The treatment of chronic laryngitis must carry a guarded prognosis for more than partial alleviation of symptoms, and must always recognize the necessary reversing of stages productive in the chronicity. The study of steps in etiology is the more important in recognizing the hygienic, dietetic, localized or general irritant factors in the background.

The treatment for simple chronic laryngitis of the diffuse or so-called "catarrhal type" must begin with a careful eliminative study of these etiological factors. By its very nature there will be time for such study.

The Waldeyer ring should receive early attention; adenoids should be studied, by mirror or digital examination, and upper postnasal space reviewed by nasopharyngoscope. If the hypertrophy be enough to cause postnasal block or if it indicates chronic low-grade infection, the mass should be thoroughly removed.

The tonsils, in presence of chronic congestion of anterior pillars, peri-

cryptal redness, inflammation of lymphoid elements, or with evidently bacteria-laden exudate, should be eliminated, including all suggestive lymphoid remnants at the lower pole or on margins of the plica triangularis. Lingual tonsils may, too, present chronic follicular infection and indicate topical treatment or amputation. Galvanocautery or other partial methods will more likely lead to sealing of surface tissue with a continuance of deeper absorption.

Sinuses should be studied by transillumination, and better with added Roentgen-ray examination and if with both frontals, antra and even sphenoids clear, the ethmoidal area presents a hazy, clouded appearance, suggesting hyperplastic change, a careful review of intranasal pressures should be made, to determine deflection or other offending irregularities of the septum. Careful wiping of exit areas in middle meatal fossæ and subsequent observation will often reveal suspicious exudate, and the nasopharyngoscope may show an undue polypoid degeneration of the posterior ends of the middle turbinates when anterior were not suspected. Pathology should receive its appropriate drainage or more complete surgery.

The teeth, especially devitalized types, often with massive fillings or Richmond crowns, and diseased borderlines of the gums may be sufficient to give the focal discharge directly into oropharynx or by lymphatic current. Examination of the ear will often reveal an almost forgotten perforation with very slight, offensive secretion, both indicating deeper attic focus and discharging via eustachian tube or directly through lymphatic stream and glandular chain into the superficial and deep cervical nodes.

Coincident with this systemic derangement should be studied hepatic, renal and intrathoracic disorders; alcohol and tobacco history should be scrutinized and with the search for more intricate foci, such simple causes as hygienic environment, climatic susceptibility, and individual faults in clothing, bathing, etc., must not be overlooked. (Defective elimination must be combated by increasing the elimination by bowels, skin, and kidneys.—EDITOR.)

Surface topical treatment in chronic laryngitis is not particularly encouraging, and sprays and gargles give usually but temporary relief and do not reach the area. In certain types the guarded use of cocaine, adrenaline, or ephedrine is permissible. Inhalations of benzoin, iodine, menthol, etc., may be more effective, and argyrol 20 to 50 per cent. solution is often employed. Perchloride of iron in 5 per cent. strength may be occasionally tried. The essential topical effort is to produce an antiseptic, astringent, and sedative reaction without unduly sealing the surface crypts. Alterative assistance of varied types and tonic help of such combinations as syrup of the iodine of iron and cod-liver oil, hypophosphites, and other nutritional adjuvants are distinctly indicated.

In this, as other types of laryngeal involvement, it is assumed that the voice will be given all possible rest, and climatic conditions made completely favorable.

In the localized, particularly subglottic, type the problem is a more serious one and brings a stenosis factor into the field which may require bougie or other dilatation methods. Astringent applications of alum powder, 2 or 3 per cent. zinc or silver solution may help. External massage, faradism, and the high-frequency current are of very doubtful value. If there be distinct tenderness on external pressure near the

larynx or other rheumatoid suggestion, the salicylates and alkalies may well be pushed.

A marked hypoglottic hypertrophy with spasm will invite consideration of the tranquil type of tracheotomy and putting the larynx at rest for sufficient period to pursue local measures with impunity. Local tuberculous, specific, and malignant indications must be carefully differentiated in this more localized type.

If the chronic type has merged into or been succeeded by the atrophic or dry laryngeal picture, systemic medication should be pushed, topical applications must clear the dried mucoid secretions, and the administration of ammonia preparations may be used to stimulate the local expectoration.

Ulceration occurring in this type may produce spitting of blood which must be distinguished from a true hemoptysis. Granulating surfaces may be controlled by cautious use of silver, zinc chloride or trichloroacetic solutions, but the danger of irritation must be borne in mind.

Chorditis nodosa or the "singers' nodes" require, first, voice rest, re-teaching of voice placement, astringents, and occasionally, in aggravated cases, actual removal of the offending projections. (See Index for further consideration of this subject.)

In final advice, a guarded prognosis should always be given in the treatment of chronic laryngitis.

JAMES A. BABBITT.

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## CONTACT ULCER OF THE LARYNX

**Definition.**—Contact ulcer is the name I have given to superficial ulceration occurring on one or both sides of the larynx posteriorly, the ulcerated surface coming in contact, on phonation, with that of its fellow of the opposite side, the latter being ulcerated or not, according to whether the ulcer is monolateral or bilateral.

**Incidence.**—The lesion is not a common one, inasmuch as I have seen only 217 cases in forty years.

**Etiology.**—The location and the evident contactual pressure seen in the mirror during phonation seem to indicate vocal abuse as the chief etiologic factor. This is corroborated by the fact that the condition is observed in persons using the voice excessively, and it is further corroborated by the observation that cure is usually impossible without vocal rest. Most of the cases have been seen in conjunction with chronic laryngitis, which would justify the inference that chronic inflammation of the laryngeal mucosa is also a cause, or at least that the ulcer and the chronic laryngitis are dependent upon a cause common to both. Many of the patients complained of waking with a choking, strangling cough at night, relieved after secretions were coughed out of the trachea. This suggested leakage into the larynx of oral secretions that should drain away downward into the esophagus. Such secretions would carry oral infection. This symptom was not present in all cases, however.

**Pathology.**—Histological examinations by Ralph Duffy, Joseph H. Barach, Ernest Willets, Ernest Funk, W. M. L. Coplin, B. F. Crawford,

Herbert Fox, and C. J. Bucher in the various cases in which specimens have been taken to exclude malignant and tuberculous disease, have all shown the usual histological characteristics of chronic inflammation with ulceration. No specific histological characteristics have been found so far. Bacteriologically the lesion has been found to be most frequently associated with Vincent's organisms, but in many typical cases these organisms were absent. While the ulcer is not deep, the vocal process of the arytenoid cartilage, because of its superficial position, is often involved and it may become necrotic.

**Symptomatology.**—Hoarseness is present in most cases, though it may be slight. Pain or at least discomfort in the larynx is usually present, though it may be overlooked by stolid individuals. In a few cases it was severe and radiated to the ear of the ulcerated side. The symptoms are usually those of the accompanying chronic laryngitis. Choking and strangling at night from overflow of oral secretions into the larynx was present in a number of cases.

**Diagnosis.**—Exclusion of cancer and tuberculosis is called for in all cases. In many cases the extremely superficial character of the ulcer, rather an erosion or loss of epithelium than a true ulceration, taken to-

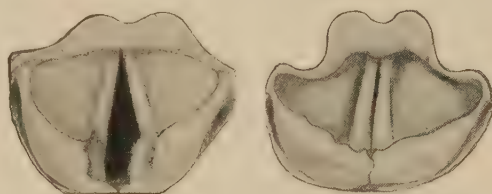


Fig. 418.—Sketch illustrating bilateral contact ulcer of the larynx in a man aged fifty-two years. At the left the ulcers are shown as exposed during inspiration; the little white points represent the exposed tips of the vocal processes of the arytenoid cartilages in the beds of the respective ulcers, on the right and left sides. In the right-hand illustration the ulcers are shown as hidden during phonation.

gether with the free mobility of the arytenoid, renders it advisable to await the result of a régime of absolute silence and omission of all irritant forms of treatment before taking a specimen. If, however, the ulcer persists after a month or two, biopsy is advisable. The utmost care and precision are required to avoid injury to the crico-arytenoid joint. When necessary for diagnosis the excision usually has the added advantage of promoting cure of the ulceration. At biopsy the necrotic vocal process of the arytenoid cartilage is often found in the bed of the ulcer. No harm is done if a very small amount of the extreme tip of the process is removed, and in fact healing may follow.

**Laryngoscopic Appearances.**—In the mirror the ulcer is seen on edge, involving the almost perpendicular internal surface of the arytenoid cartilage (Fig. 418). On inspiration the ulcer is exposed to view but on phonation its internal surface is in contact with the internal surface of the opposite arytenoid eminence, which may or may not be ulcerated. Usually the thickened border of the non-ulcerated mucosa at the upper margin of the lesion is seen, but the ulcerated surface is not. In some instances the border forms a tiny roll-rim around the ulcer, and the vocal process of the opposite arytenoid fits into the tiny bowl-like lesion. In some instances

the denuded vocal process of the ulcerated arytenoid is found in the bottom of the bowl. The usual site of the ulcer is the area in the neighborhood of the vocal process.

**Prognosis.**—Under mild, gentle, careful treatment in almost all cases ultimate recovery has followed; but the progress has been rather slow in most instances. Energetic ill-advised treatment is usually followed by edema, loss of cartilage, impairment of the arytenoid joint, and increased hoarseness. In one patient sent to us extensive cauterization had caused perichondritis with acute edematous stenosis, followed by chronic cicatricial stenosis. If the patient persists in vocal abuse prognosis as to cure is unfavorable. In two such cases cancer ultimately developed. In two other cases in patients with pulmonary tuberculosis the ulcers ultimately became tuberculous.

**Treatment.**—The fundamental requirement is absolute silence, the patient writing every word he has to say. Even whispering should be forbidden. All irritant applications such as silver nitrate, or any other medicaments on a swab are contraindicated. If two months of silence have failed to cure the ulcer it is better to excise it with great care and precision. Care should be taken to avoid damage to the crico-arytenoid joint and to avoid removal of a normal vocal process. If the vocal process is necrotic, removal of a small portion may promote healing. When there is a thickened rim of epithelialized mucosa around the ulcer careful nipping off of this rim with small cupped forceps usually promotes healing in from the edges. After operation residence at the seashore promotes healing. The ozone, chlorine, and vapor of salt water inspirated twenty-four hours out of the twenty-four are of great benefit, not only to the ulceration, but to the accompanying chronic laryngitis.

CHEVALIER JACKSON and CHEVALIER L. JACKSON.

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## THE VOCAL ORGAN IN SINGING. OVERUSE AND MISUSE OF THE VOICE

The laryngologist should unquestionably have a thorough understanding of the various vocal disorders which he is in a position to observe in singers, orators, and what one might call professional voice-users. He should also know the therapeutic methods suitable to apply to them. We are far beyond the time when hoarseness and other manifestations observed in this category of professional voice-users were attributed to "granular" pharyngitis or laryngitis.

In order thoroughly to understand the pathology of these morbid manifestations one must know not only the anatomical constitution of the vocal apparatus but also, and even more important, the manner in which it behaves in the course of its function, particularly in singing: in a word, what one may ask of it, and what one must not ask of it, except at the risk of altering and injuring it.

Like any musical instrument, the vocal organ is composed of three essential parts, which are: first, a vibrating apparatus, the vocal reed formed by the musculomembranous cords with which we are familiar; second, a bellows or pulmonary reservoir, a sort of bow destined to put in vibration that apparatus; third and finally, the resonators serving to amplify the sound and to give it quality and timbre. The resonance cavities are numerous and varied in shape. There exist, in fact, two kinds of resonators: the superior, situated above the vocal cords, and the inferior, which are those of the subglottic region.

The superior resonators comprise: the vestibule of the larynx, the ventricles of Morgagni, the hypopharynx, the soft palate, the nasopharynx, and all the nasal cavities, with the sinuses and the buccal cavity. The inferior resonators comprise the trachea, the bronchi, and all the contents of the thoracic cage; these are the ones which serve particularly to produce the so-called "chest sounds." It is possible for the singer with a good vocal training to cause the use of the inferior resonators to predominate over the superior, and inversely, or to use both at the same time. Such are the three parts of the vocal apparatus which cause it to resemble a musical instrument. Let us now discuss its function during singing.

During inspiration the vocal cords separate to allow the external air to enter, while at the moment of phonation the two arytenoids come together posteriorly, the cords coming in contact by their free borders. If at this moment the air in the lungs is expelled, the vocal cords are caused to vibrate. There has therefore been, at first, approximation of the vocal cords, due to the contraction of the constrictor muscles of the larynx. This movement accomplished, the tensor muscles (thyro-arytenoidei and cricothyroidei) come into play, tensing the cords, and consequently permitting the reed to vibrate either throughout its length or only in part. The reed can tighten itself to varying degrees, to give notes of different pitch, and the pulmonary bellows can augment or diminish the volume of the sound.

It is possible to modify at will the quantity of air utilized, a fact which enables the artist to modulate his voice. Thus, one may make use of the three different types of respiration well known to the physiologists: the superior costal type, which consists in raising the superior portion of the thorax while the inferior portion remains almost immobile; the inferior costal type; and finally, the diaphragmatic or abdominal type. Normally the female uses the superior costal type of respiration, while the male uses the abdominal or the inferior thoracic type. Certain authors assert, however, that women who have not been accustomed to wearing corsets breathe in the same manner as men.

Granted that the singer can moderate at will his respiratory power, it is easy to understand that artists requiring only a small volume, such as little coloratura sopranos (the Lakmé type), need to store only a small quantity of air, and use the superior costal type of breathing. On the contrary, artists of large volume must employ at the same time the inferior costal and the diaphragmatic, or even all three types.

I insist on this, for I have been much astonished in reading the treatises on singing written by doctors such as Bataille, Mandl, and others, to see that these practitioners, after describing the diverse respiratory types, do not think of advising adaptation of the type of breathing to the reed which the particular singer possesses and which he must put into vibration. Even

Baratoux does not make this distinction; Isnardon alone, a professor of singing at the Paris Conservatory, has declared, in his "Chant Théâtral," that one must adapt the bellows to the dimensions of the reed. This view seems so reasonable that it is strange we do not find some similar recommendation in all the treatises on singing.

An artist having a small larynx, with slender and consequently delicate vocal cords, should not be permitted to use all the respiratory power at his command, especially if he has large lungs. To do so would be like using a bass-violin bow to play a violin. When in examining a singer, man or woman, the laryngologist sees that he has a slender vocal organ and a large chest, he must immediately advise his patient of the fact, saying, "Madam (or Sir), you have very delicate vocal cords; do not subject them to a great blast of air, unless you wish to ruin them. Cut down your pulmonary capacity, therefore, by restricting the lower portion of your thorax with a rubber girdle or a corset." The corset may be a useful article, if it is intelligently used.

**Registers.**—I now arrive at the question of register. In discussing the singing voice the registers are often confused with range. Baratoux himself, in a book which he has just published, often uses these two terms interchangeably. What is *range* exactly? With Faure, we shall define it as follows: "*The series of consecutive notes produced with facility, and having good quality and timbre; it is the natural voice production. A register is, on the contrary, the series of notes which one produces with the aid of the same vocal mechanism.*"

When a singer places his vocal cords in the position for singing and expels the air from his lungs, if he produces a deep sound, that is, one in the so-called "chest register," the vocal cords, which are approximated, vibrate throughout their entire length and thickness. This fact, well known to all the physiologists, was demonstrated long ago. In order to activate the vocal cords in this position a large volume of air is needed. Accordingly, during the production of notes in this deep register, the larynx is under a great strain, because it is obliged to resist a violent blast of air. The singer, when forced to sing at the top of his voice, is obliged to bring into play all his resonators, the vocal apparatus performing a maximum of work; and if he does not modify the position of his cords as his voice mounts the diatonic scale, that is, if the cords continue to vibrate throughout their entire length, he is obliged to augment the power of his breathing, which results in increased fatigue of the larynx. It has been shown, in fact, by the experiments of Muller, that it is possible to raise pitch by augmenting the quantity of air passing through the cords, without changing their degree of tension; but, as the singer can use only that quantity of air determined by his pulmonary capacity, he cannot continue to mount the scale with his cords remaining in the same position. There arrives a time when the glottis can no longer be activated, and it becomes necessary to modify the length of its vibrating portion.

Therefore, when an artist says that he has sung a "high C" in the chest voice, he is using an inexact expression, because one does not sing "high C" in the chest voice, for the simple reason that in order to do so one would need to have steel cords (Crosti) and super-powerful lungs. The singer arrives, therefore, at a place in the scale (in the vicinity of G and A) where he must modify the position of his vocal reed, in order to pass from the so-called

“chest,” or “thick” (Garnault) register to the mixed register. The cords have now diminished in length, the arytenoids have come into closer approximation, and the glottic orifice has become more narrow, with the result that, the breath no longer having to put into vibration but a small part of the cords, the vocal effort is much less great, and therefore less fatiguing for the singer.



Fig. 419.—Vocal cords in position for phonation.

In the mixed register the vocal cords vibrate in only a part of their length, and thanks to the modifications which are produced in the reed, the voice is able, *without augmenting the quantity of breath*, to sing up the scale. The artist does not fatigue his cords because he raises the pitch with his reed and not with his breath. As he passes from *E* to *F* he will further

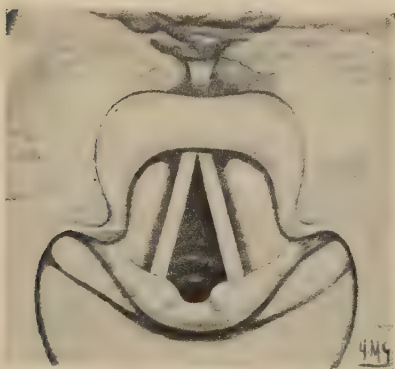


Fig. 420.—Larynx during inspiration.

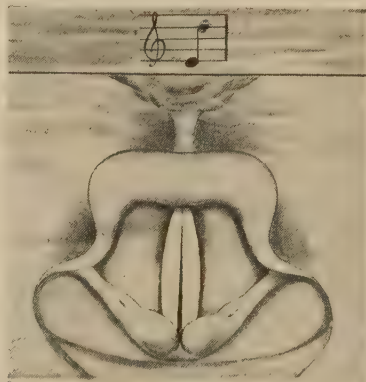


Fig. 421.—Position of the vocal cords during the production of the register spoken of as the “chest voice.”

diminish the length and thickness of his reed, the slit through which the air will pass will be further reduced, and he will be able to get up the scale without difficulty; he will thus pass into the so-called “falsetto voice” (head register) without realizing it, and *he will have obtained the maximum of voice production with a minimum of effort*.

On the other hand, once in the mixed register, the timbre of the voice will be more agreeable to the audience, because, abandoning the lower,

the singer will use chiefly his upper, resonators; he will sing "into a mask," a rather good metaphor for one who understands its scientific explanation. What I say for the singer applies equally to the orator, who, when he speaks in the low register, soon fatigues his voice, while he demands much less of it if he speaks in the middle register.



Fig. 422.—Position of the vocal cords during the production of the mixed register.

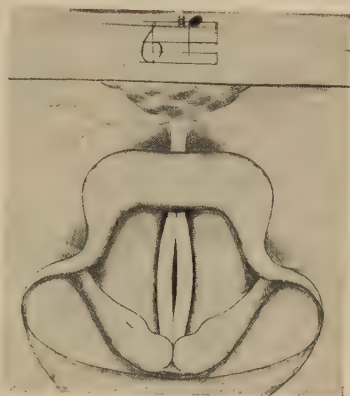


Fig. 423.—Position of the vocal cords during the production of the high register.

**Range.**—A singer's range, as I have just said, is the series of notes that he produces easily, and with good tone quality. In using the larynx for singing one must take into consideration its peculiar anatomical construction. Each one of us has a certain range, very short in some, consisting of only three or four notes; more extensive in others, attaining one, two, or



Fig. 424.—Position of the vocal cords during the production of the very high register.



Fig. 425.—Same as Fig. 424.

even three octaves of the scale. The maximum range of the human voice is from  $C^1$  to  $F^5$ ; a singer possessing this ideal voice would be able to sing without fatiguing himself in any range. Unfortunately such a voice does not exist.

Musicians, for the sake of facility in writing music, assign to each category of singers a definite vocal range, thus creating definite types, for



limits. Therefore, the intermediary types were created, such as basso-profundo, singing base, and deep baritone; then between the tenor and the ordinary baritone was placed the Martin or Verdi baritone, etc. Likewise for women, in addition to the operatic soprano, the dramatic soprano was created, such as la Dugazon; between the soprano and contralto we find the mezzo-soprano, below the contralto, the mezzo-contralto, or the lyric singer of opera or comic opera. When an artist has been fortunate enough to have an especially beautiful voice, even though it does not conform to a mold recognized by the musicians, a new type has been created and given the name of the artist in question, whence the types of voice designated by the names Falcon, Dugazon, Galli-Marié, etc.

I am opposed to the premature classification of voices. When a singer comes to a professor, the latter should say to him, *without concerning himself with classification at the outset*: "We are going to cultivate the notes which you have and perfect them; we shall see later if your voice can be classified for the stage." In doing this the professor will not only have conserved the voice which is entrusted to him, but he will also have suppld and molded it. This is unfortunately not what occurs in Conservatories of Music (notably in Paris) nor under many of the professors.



Fig. 428.—Total range of the human voice.

The voices of pupils are nearly always classified at the beginning of their studies, and if there is a mistake, woe to the beginner!

What I say is not the result of recent observation; specialists have always concerned themselves with this question, and with this kind of vocal misuse. Morrell Mackenzie, a well known author, has written on this subject: "One can no more transform a baritone into a tenor than a blackbird into a grasshopper." The metaphor, although it may seem exaggerated, is very apt. One may, by practice, improve tone quality and modify timbre, but one can neither increase the vocal organ in size nor change its proportions.

#### OVERUSE OF THE VOICE

*By overuse of the voice is meant excessive production demanded of a vocal organ poorly trained or not having the constitution necessary to bear the work to which it is subjected.*

Vocal overuse can come from different causes. First, it may result from a faulty relationship between the different component parts of the vocal apparatus, as, for example, great pulmonary power and a small larynx possessing slender and therefore unresistant vocal cords; this anatomical fault is rather frequent in artists who make the mistake of having their

voices classified according to timbre and volume. These singers interpret rôles for which they are not equipped. It is thus that a tenor or a light baritone engages himself as a heavy tenor or grand-opera baritone, a lyric soprano as operatic soprano, etc., demanding of their vocal organs an excessive voice production. In this regard, one must wonder if the operatic tenor or operatic soprano is born, in a word, whether there exists a vocal



Fig. 429.—Hemorrhage on the left vocal cord.

apparatus made to sing at sight rôles of the repertoire adapted to this kind of voice. It is not to be expected that an artist can give the maximum production from the beginning of his study. Even having the larynx of an operatic tenor or an operatic soprano, a prudent artist should at the beginning abstain from interpreting such rôles.

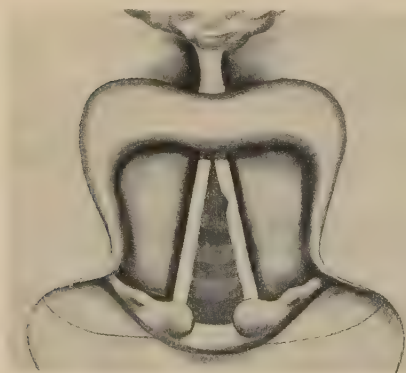


Fig. 430.—Notch in the vocal cord following a rupture of the thyro-arytenoid muscle ("coup de fouet").



Fig. 431.—Paresis of the thyro-arytenoidei. Muscular asynergia.

Faure wrote in his method: "It is the audience, fond of extraordinary effects, which causes the ruin of artists (and of tenors in particular) especially the provincial audience, which has adopted the motto, "Tell me how high you go, and I shall tell you how good you are." Duprez, on the other hand, required extreme vocal efforts of his pupils. He told them to "sing or burst!" and unfortunately they sometimes did the latter.

Although grave, the consequences of overuse are infinitely less so than those of misuse. A singer who overuses his voice experiences at first only a passing hoarseness. On awaking, his voice is not free, he is obliged to try it out, to warm it up before he is able to produce even fairly high notes. Later, the voice becomes less sonorous, it is less powerful and less well timbred, lacks charm and suppleness, and becomes tremulous. These vocal manifestations correspond to anatomical lesions. The latter are very slight at first, however, and if the practitioner's eye is not experienced he may not note them when he examines the larynx. However, if he has the patient attack different notes under observation with the mirror, he readily sees that the borders of the vocal cords vibrate badly and allow too much air to escape. There is a slight delay in the emission of the sound; the singer has no longer the power to cause a sufficiently rapid muscular contraction to tense the borders of the cords. This is a sign of fatigue to which the greatest attention must be paid. In the more serious cases one may see produced what I have called the "*coup de fouet laryngien*." This occurred on one occasion when a tenor who had cords too slender to interpret the heavy tenor roles was singing "William Tell." Having arrived at the famous "*Suivez-moi*," which is written for a high range, and which the artist must sing at the top of his voice if he wants the audience to accord him any applause, his voice suddenly failed him; he made a squawk and was obliged to stop. When I examined him the next day I found on his left cord the lesion which I have reproduced here; he had a submucous hemorrhage which explained the vocal difficulty he had had. He was obliged to cancel his engagement and await the return of his voice. It did return, but the following year while singing in another city he had the same accident, and this time I found that he had not only a hemorrhage but a muscular rupture which resulted in the loss of his singing voice.

I had occasion, subsequently, to see other examples of the same condition. One case was that of a stage director well known for his violence. Reviling an artist with whom he was angry, he suddenly lost his voice, and I found in him the same lesions that I had observed in the singer mentioned above. Another case was that of a lawyer who had the bad habit of pleading too violently and flying into a rage. He presented the same picture as the above patient, and was obliged to abandon his career.

These various troubles, slight or severe, may follow overuse of the voice in anyone who makes exaggerated vocal efforts. There are to be found in the history of singing many analogous examples. There is first that of Mlle. Falcon, whose voice broke suddenly while she was singing at the Opéra. Duprez and Nourrit saw their careers terminated in much the same manner. Overuse leads to other accidents less grave, but no less important, for example emphysema. It is thus that one often sees middle-aged artists with apparently enormous lungs and scarcely any breath; they have emphysema because they have overused their bellows.

With respect to the resonators, various slight disorders may occur; for example, hypertrophy of the turbinates, obliging the singer to have the mucosa of the nasal fossæ cauterized, or to undergo some more radical operation in order to diminish the chronic tumefaction of his inferior turbinates.

I do not mention granular laryngitis, which for a long time occupied an important place in the pathology of the vocal organ and the throat.

Granular laryngitis and pharyngitis have disappeared from the category of diseases, considered as primary and independent entities.

The treatment of vocal overuse is very simple; it consists in calling the attention of the patient to the harm he is doing and warning him of what may happen if he does not listen to advice. If the diagnosis is promptly made, the treatment indicated is vocal rest, and the patient should be instructed to sing with more moderation, not trying to augment the volume and power of his voice. One may supplement this by some local treatment, sprays, etc.

#### MISUSE OF THE VOICE

I once defined misuse, with Dr. Bouyer, as: "misdirection which exaggerates, hinders, deflects, or alters the normal physiology." Vocal misuse has been known for a very long time; it may be pathological or physiological.

*Pathological misuse* consists in singing under unfavorable conditions, as for example during the course of a cold, or following a treatment which had caused a state of congestion. It constitutes misuse of the voice to sing in the course of a mineral water treatment, in particular one of the sulphurous type, which causes a certain congestion of the pharyngo-laryngeal mucosa. It is also misuse of the voice to sing after a full meal, in the open air, or in an atmosphere of tobacco smoke. Unfortunately, directors do not always ask their artists if they are in good voice; therefore it almost inevitably happens that the singers misuse their voices. However, such temporary misuse is not serious.

This is not the case with *physiological misuse*. The most important cause of this is premature classification of the voice. I have already written above that it is impossible to classify a voice by laryngoscopic examination, for between a baritone's larynx, a Martin baritone's larynx, and a tenor's, there may be a very slight objective difference. Therefore, there can be no classification according to the ear, and no classification by the laryngoscope, but the professor of singing and the laryngologist could together make a classification, if the vocal apparatus conformed exactly to the types created by musicians. This is not, however, the case, and therefore I am entirely opposed to early classification, even by the united efforts of two experts, for an error is always dangerous. If one is mistaken, loss of voice results. The number of singers misclassified in the course of their studies is considerable, and those who have succeeded in spite of it have been sufficiently prudent to stop of their own accord and begin again in a new range. I have known some who from baritone have become tenor, and inversely; others who from *basse chantante* have become baritone, etc. Singing in unsuited ranges is a very bad thing for the voice. Very often, in opera companies, the director asks an artist to sing, at a twenty-four-hour interval, rôles of totally different range; it is thus that a baritone sings one day an opera of Verdi, and the next, a deep baritone rôle such as that of Nevers in "Les Huguenots." It will not be surprising to see him temporarily hoarse after that, and if such misuse continues, inevitably there will occur more serious disorders of his larynx.

Faure has attracted attention to this deplorable situation, as follows: "The most pernicious of pluralities is that which is required of operatic sopranos. Except in cities of the first order the *forte chanteuse* is at the same time soprano, mezzo, and contralto; she sings today "La Juive," tomorrow "Le Prophète," and the day after "La Favorita," so that she is at the same

time Falcon, Stoltz, and Viardot. She is the jack-of-all-trades of the company, and it may be said that her repertoire is the more rich as the company is the less so."

In addition to this form of misuse by change of range there is another form which consists in bad use of the bellows, that is, in adopting an identical mode of respiration for all singers, whether the larynx be large, medium, or small, and whether the vocal cords be slender or thick. When a professor of singing has decided that a singer should always use the abdominal type of breathing, regardless of the vocal equipment of particular pupils, he will force them all to use the type of respiration which stores the most air. This would be, in the case of a little soprano, just like having a violinist play his violin with a cello or bass-viol bow. It is in the course of training that the singing teacher should realize the degree of resistance of his pupil's vocal reed, and the quantity of air to employ in order to put the vocal cords into action.

One may also misuse his instrument by attacking his notes with the breath, that is by putting the bellows into action before the vocal cords are in position for phonation; thus one fatigues the lungs and does not permit the pupil to have his full reserve of air, for he has already lost some of it before commencing to produce a sound. This fault is clearly shown in the artists who do not always sing very accurately, and who reattack a note produced too low, by using their bellows. The artist who continually produces glottal stops, making exaggerated efforts, is also misusing his voice.

I arrive now at the most important part of my subject, to that form of vocal misuse which produces the most grave disorders—abuse of the chest register. If one refers to what I have said above on the position of the vocal reed during the production of this deep register, and if one recalls that the vocal cords vibrate throughout their entire length and in their full thickness, it is easily understood that if the artist goes up the scale keeping the larynx in the same position he fatigues his cords, for he requires a maximum effort of them. It is, therefore, necessary for the artist to use his chest register as little as possible. My views on the abuse of the chest register are not personal with me; these facts have been known for a long time. Tosi, in 1723, wrote:

"Because of their lack of experience, some teachers oblige the pupil to sustain whole notes on high tones, forcing the chest voice. As a result of this, from day to day, the throat becomes more inflamed, and if the unfortunate singer does not lose his voice, he at least loses his high notes."

Here is the opinion of Faure, eminent artist and conscientious observer:

"Without emphasizing the inconveniences that result from use of the chest voice, apart from its limitations with respect to verity of expression, one may easily see the disastrous effects which this régime must produce on the voice, destroying its richness and sweetness, if indeed it does not effect its total loss. One must use the chest voice only with the greatest of care. It is a two-edged sword, as dangerous as it is difficult to wield. The disturbance which the use of the upper notes of the chest voice produces in the junction of the two registers and in the homogeneity and balance of the voice, destroys little by little its charm and purity; if one does not stop in time, these disturbances must inevitably lead to impairment of the voice, and often even to its *total loss*."

Melchissedech, formerly professor at the Conservatory of Paris, who preserved his voice to a very advanced age (he died at the age of eighty years) wrote:

"There is nothing more edifying on this subject than the life of Mlle. Falcon, born in Paris in 1812, who entered the Conservatory in 1827, where she had as professors: Henri, creator of the rôle of Sulpice in "The Daughter of the Regiment," 2d bass (one realizes what a singer he must have been) as professor of vocalization; then Pellegrini, who died insane in 1832, composer and singer, and Bordogni, for singing; she was after that the pupil of Nourrit for dramatic declamation (one spoke then of "dramatic singing"). . . . She obtained the first prize in dramatic singing in 1831. She made her debut July 20, 1832, at the Opéra, in "Robert le Diable," and she created the rôle of Alice with immense success.

"At the time of her conservatory successes, a certain critic, somewhat clairvoyant, had said to Mlle. Falcon, who was tall, beautiful, and distinguished, musician, tragédienne, comédienne, thus possessing all the gifts: 'Be careful, mademoiselle, of the way you try to unite the chest voice to the head voice.'

"To make a long story short, five years later, one evening on the stage at the Opéra, Mlle. Falcon found herself without voice, quite aphonic, and for ten years she was taken about in Italy and Sicily with no relief."

Here is another anecdote reported by the same author, Melchissedeck, about La Grassini, the celebrated singer, who was then seventy-six years of age, having been born at Varese in 1772:

"In 1849 the tenor Reviel, creator of the 'Cheval de Bronze', gave a soirée on the occasion of his nomination as Professor at the Conservatory. La Grassini was present and was asked to sing. She complied, and sang a very pretty Italian air in which there were some very high notes. She was given an ovation. A certain gentleman approached, and thinking he was paying a compliment, said: 'Ah Madame, it was admirable, so much the more so because you do not use the chest voice!' La Grassini replied in the Neapolitan accent she had never gotten rid of: 'The chest voice?' What is that? The gentleman then produced a chest sound. 'Oh, don't do that, you will ruin your voice,' said La Grassini. She had never sung chest tones and yet didn't realize it."

The author continues his book in a rather unpleasant way, calling the singing teachers assassins, rascals, ignoramuses, and other similar names.

I had occasion, several years ago, to give a lecture on this subject in Madrid. At the close of the lecture I had several Spanish singing teachers come to me and say, "Here, we never use the chest register; we are totally ignorant of that register in Spain." One may, I think, say as much of the Italian singers. Fortunate countries! Is it the same in America?

**Results of Misuse.**—I once made a study, with A. Bouyer, of the disorders, functional and local, resulting from misuse of the voice. *First* there are what we called the minor indications. They consist of a fatigue manifesting itself in a persistent need to clear the throat, and a slight dysphonia. After a moment's singing the tone produced is no longer as clear as at the moment when the artist began to sing; soft notes are no longer so easy nor so pure, the attack of the notes does not have its accustomed purity, and one feels that the reed no longer vibrates so well. It is especially in women (sopranos in particular), and in the light tenors, that these signs are noted. A little later the passage from one register to another is neither easy nor pleasant to the ear, the timbre lacks purity, and if the singer is observant, he realizes that he is producing a sound with his breath, the air passing before the cords have put themselves in position for phonation.

*In the second stage* the disorders are much more marked. The artist has hawking spells, and at every moment he is obliged to clear his throat in order to rid himself of the particles of secretion which encumber his larynx. This symptom was formerly attributed, erroneously, to so-called "granular laryngitis." The singer must warm up his voice, which is embarrassed and often hoarse on awaking. After singing, he feels a burning in the throat and a certain degree of hoarseness.

If, in spite of these warnings, he continues to misuse his voice, he will

soon arrive at the *third stage*, in which he is constantly hoarse. He is no longer master of his voice, and often he makes a horrible squawk, a warning that his vocal cords are gravely involved. This is the final stage: the sound of the voice is rough and muffled, and, in spite of his efforts, one may, if standing close to him while he sings, hear a sort of whistle resulting from a faulty tension of the reed, which allows the air to pass between the poorly

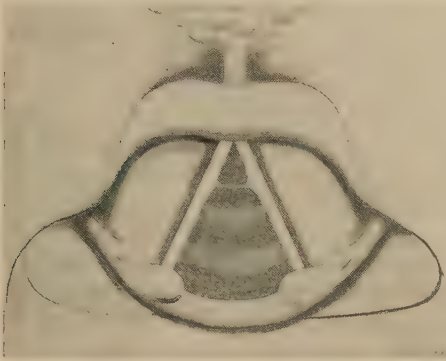


Fig. 432.—Early vocal nodules with tenacious string of secretion between the cords.



Fig. 433.—Slight nodular swelling seen during phonation.

tensed and insufficiently approximated vocal cords. At this moment the career of the artist is gravely compromised; he sings one day and not the next; then he comes to consult the specialist, when it is too late.

*To what lesions do the symptoms observed in these three stages correspond?*

1. At the beginning the lesions are of little importance; it is only with the aid of questioning the patient and with the specialist's experience that one



Fig. 434.—Vocal nodules during phonation.

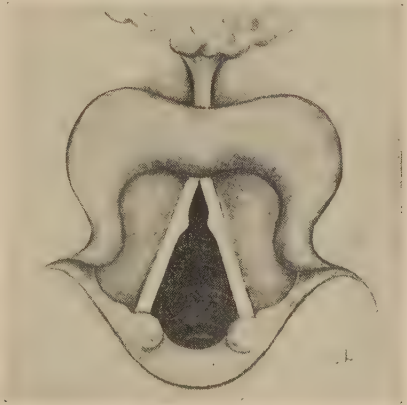


Fig. 435.—Vocal nodules.

may realize the harm done to the larynx. Sometimes, however, the vocal cords appear a little dull, and one of them is slightly more pink than its fellow. One may see, on mirror examination, that when the singer wishes to emit a sound in the head voice or in the mixed voice, the reed does not draw tense at once; it is a little slow.

2. Later there appears a slight swelling, which, though it does not prevent the cords from approximating, does not permit them to join each other

completely. Some air passes between them; there is a slight loss. If you ask a singer to produce a soft tone under observation with the mirror, you will see very well that the vocal reed closes badly. If, on the contrary, you ask the artist to sing, without putting the laryngeal mirror in place, the vocal difficulty seems almost insignificant, the voice comes out clearer and more vibrant, but also louder, the blast of air being greater. The patient masks his lesion.

3. If one has not recognized these little disorders resulting from misuse, and sought their cause, the artist will soon present more serious manifestations; the redness will increase and there will develop a paresis of the thyro-arytenoid muscles; the approximation of the cords will not be perfect; then there will appear a little swelling, which will not yet be a nodule, but the prelude to that grave alteration. Sometimes a true redness of the entire cord is observed, and under the mirror one sees clearly that the unfortunate singer is absolutely powerless to emit a clear sound, because his vocal cords no longer contract normally. If the misuse is continued he arrives at the stage of complete vocal asynergia, which explains why he can produce a sound one day and not the next.

4. Next, one sees appear on the edge of the two vocal cords those little nodular swellings about which much has been written, and which during inspiration present the appearance of two little sharp-pointed prominences, occupying ordinarily the anterior third of the vocal cords in tenors and sopranos, and the posterior third in basses. There results from them a loss of air, preventing the voice from being clear and of good quality. This is the grave period.

Vocal nodules are not—and it is very important to know that they are not—true laryngeal polyps, for their removal does not cure the patient. They are more grave, because they are the indication of a *muscular asynergia*, most often incurable, or at least very persistent and difficult to cure.

**Treatment.**—Before any treatment the specialist should make a diagnosis; without that, no treatment profitable to the patient is possible. He must recognize not only the condition of the larynx, but also the cause of the pathological alterations found. This is not always easy as artists do not willingly confess their faults; they will tell you “I caught a cold,” “I have rheumatism,” or “I have a laryngitis,” but one has the greatest difficulty to make them admit that they make improper use of their voices, singing in ranges that are not properly theirs, abusing the chest register, etc. If you succeed in finding out that a singer sings all the rôles of a score, it is useless to question further, for a singer who amuses himself by thus changing his voice cannot fail to misuse it! Whatever certain authors may say, it is especially, in my opinion, misplacement in the low register which is harmful to the singing voice, and also to the speaking voice. An orator who speaks constantly in the low register is destined to become hoarse, a victim of vocal misuse.

Treatment should be as follows:

1. Vocal rest, which is the basis of the treatment.
2. Change in range if the artist is wrongly classified.
3. Change of method, especially method of voice production.

Unfortunately, these patients, when the lesions and symptoms are slight, will scarcely listen to the doctor's advice. Generally the artist continues to misuse his voice; those who cease on a simple warning are

few. The majority of the singers arrive in a few years at the third stage of misuse, where one can no longer do anything for them, even by means of surgery, which it is preferable never to use in the larynx of a singer. Removal of a projecting nodule will not cure the nodular laryngitis; it would be much better to suppress its cause, for I have already said, and could not repeat too often, that vocal disorder comes not from the nodule, which would diminish and even disappear after a prolonged rest, but from the asynergia of the cords.

After having considered poor utilization of the reed as an etiological factor, one must also be sure that the resonance cavities are free, that nothing in the air-passages can interfere with the work of the voice, or produce the lesions found.

Must one perform cauterizations in the larynx and apply an ambitious local treatment? No! If by chance the local treatment does not succeed, the doctor will be accused of having damaged an artist's voice. Formerly, singers had great fear of laryngeal cauterizations, and they did not want their vocal cords touched. Perhaps they were right, not that a cauterization with chloride of zinc or silver nitrate 1/50 or 1/30 is dangerous, but that it is useless, and reveals on the part of him who advises it or who performs it, a diagnostic error.

Insufflations of anodyne powder or instillations? If you wish; but advise especially a mineral water cure; send the patient to a hot spring. According to his general condition, one should send him to the sulphurous springs (Cauterets, Luchon, Bagnères-de-Bigorre, Aix-les-Bains, etc.) or to the arsenical springs (Mont-Dore, La Bourboule, even to Royat). The important thing for these people is to take an anodyne treatment, for what really matters is patience and *vocal rest*. I have seen artists rest for five or six years before regaining their voices; I even know of some women singers who let their voices rest for seven or eight years, and who after three or four years of regular work regained the voice they had lost following a nodular laryngitis occurring at the age of twenty years. Of course they had changed method and also range. Formerly classed as contraltos, some became sopranos, others mezzo-sopranos, abstaining from the chest singing which had caused the loss of their singing voice.

E. J. MOURE.

(Translated by Chevalier L. Jackson.)

## ULCEROMEMBRANOUS LARYNGITIS

**Synonyms.**—Vincent's laryngitis, Vincent's angina.

The classic authors considered the faucial pillars a barrier to Vincent's angina. Now, numerous observations justify doing away with this legend, and permit us to assert that the micro-organisms causing this disease do not fear to pass the limits of the pharynx to reach the larynx, the trachea, or even the bronchi and the parenchyma of the lung itself. It was apropos of a very severe case of ulceromembranous laryngitis, that in 1909 I was able to establish the symptomatology of this local infection, until then omitted from the treatises on laryngology.<sup>1</sup> In 1923, one of my pupils,

Dr. Bergot, collected in his inaugural thesis the scattered observations on the subject then to be found in medical literature.<sup>2</sup> (See also Vincent's Angina.)

**Etiology.**—Ulceromembranous laryngitis seems to be rare in children, at least if one judges from the observations thus far published. It is between the ages of twenty and forty years that the condition is most frequent. It develops most often in run-down, overworked, fatigued individuals. Sometimes it follows an angina of the same nature, but it can exist alone. Also, during an attack of grippe, or in the course of the eruption of a wisdom tooth (lower jaw) fusiform bacilli and spirochetes may invade the upper portion of the respiratory tree. The disease seems to be more frequent at certain seasons of the year, particularly in spring and autumn.

Briefly, the etiology of this condition appears to be as confused as that of the angina of the same name, but the soil seems to play an important rôle in its appearance. Fatigued, depressed, or overworked individuals are most frequently attacked.

**Symptomatology.**—At the beginning, the functional disturbances resemble in a general way those observed in an ordinary catarrhal laryngitis. The patient is first a little hoarse; swallowing is scarcely disturbed unless the pharynx is simultaneously the site of an infection of the same order. Respiration is normal, but the patient feels some malaise. There may be a rather pronounced febrile condition, without, however, very great temperature elevation. The patient complains of anorexia, and the tongue is coated. At this time mirror laryngoscopy reveals simply slight reddening and tumefaction of the base of the tongue and the epiglottis, as well as of the vestibular portion of the larynx, the ventricular bands, and the arytenoid region. There is not yet any exudate.

This condition may last three or four days. Then the second period, in the course of which the voice takes on a muffled, almost croupy character, is established. The respiration becomes embarrassed, the patient may even show wheezing or indrawing; in fact, asphyxia may threaten to the extent of requiring a tracheotomy.<sup>3</sup> Swallowing is often difficult and painful; cough is frequent, and when the infection has reached the trachea and bronchi, may be productive of purulent expectoration, often blood-streaked, and sometimes containing grayish gangrenous débris. Ordinarily there does not exist any adenopathy in the region.

At this stage of the disease the general condition of the patient is severely hit as a result of the involvement of various viscera, such as the kidneys, the liver, or even the central nervous system. We are dealing, in fact, with a systemic infection which may be very grave.

Locally, one no longer observes only swelling and redness, but erosions, and even diffuse ulcerations, irregular in form and covered with a grayish pultaceous exudate of diphtheroid aspect in the midst of an always abundant purulent secretion. In brief, lesions are found in the larynx resembling in all points those of ulceromembranous angina of the Vincent type.

Bacteriological examination reveals the existence of numerous fusiform bacilli and spirilla associated with other microbes—staphylococci and even streptococci.

In some severe forms one may observe a diffuse infiltration of the entire laryngeal mucosa with necrosing ulceration, resulting in a serious

stenosis and at times a perforation in the cartilages due to perichondritis, analogous to that which occurs in typhoid fever, though the bacterial flora remain characteristic.

**Course.**—The course of this laryngitis is quite variable, depending especially upon the virulence of the infection and the resistance of the patient. Generally the first stage lasts three to four days; then the second period begins, only to disappear without bringing any striking manifestations. *This is the benign form.*

*In the severe cases*, on the contrary, the general condition is seriously affected from the beginning; fever develops and persists, functional disorders are marked, and the disease often lasts for several weeks, having sometimes a tendency to extend to the deep portions of the tracheobronchial tree. Sometimes, even at the moment when the patient seems cured, one may see appear without appreciable cause a repullulation of the fusospirillar symbiosis, with recrudescence of the functional disorders and the local symptoms. In a case reported by Arrowsmith the disease lasted several months; in one of my patients<sup>4</sup> it persisted for eighteen months, with alternating periods of improvement and relapse.

Thus we see how variable the course of ulceromembranous laryngitis may be, according to the diverse clinical forms of the disease.

**Pathological Anatomy.**—Examination of the secretions and exudates removed shows various cellular elements (endothelial cells and leukocytes) and the presence of numerous fusiform bacilli and spirilla, which are characteristic of this infection, associated, of course, with the bacteria usually encountered in the region.

**Fusiform Bacillus.**—Well known since the description which was made of it in 1896 by Vincent, the fusiform bacillus is an organism having a wide midportion and slender extremities.<sup>5</sup> The bacillus is of variable length; long, medium, and short forms have been described. Its average length is from 8 to 12  $\mu$ , its thickness 1  $\mu$ . The organism contains in its interior clear vacuoles of unequal sizes, 1 to 4  $\mu$ . According to Letulle it is motile when examined in saliva, and must be considered not a bacillus, but a spirillum. Plaut also admits its motility and describes vibrating cilia. Commandon, on examination with the ultramicroscope, confirms this motility. This characteristic has, however, been disputed by Vincent.

The organism stains well with the basic aniline dyes, especially with Ziehl's red (1 : 10); it does not take the Gram stain.

It is difficult to cultivate in pure culture, but according to Vincent, if a bit of exudate is planted in peptone bouillon, it multiplies in this medium; cerebrospinal fluid enriched with human blood or pleural fluid may likewise be used. Lew Kowicz succeeded in obtaining the fusiform bacillus in pure culture by cultivating it anaerobically, in Veillon tubes, on sugar-agar enriched with one-third of infantile peritoneal fluid. It forms fine gray opaque colonies of a medusa-like aspect. All the cultures give off a putrid odor.

The organism is not pathogenic for animals, though by injecting pure cultures under the skin it has been possible, in mice, to produce an abscess and an extensive necrosis resembling the human lesions.

**Spirillum or Spirochete of Vincent.**—This is a very slender organism, difficult to stain, not staining by the method of Gram, but staining fairly well by the silver methods. It is very difficult to cultivate and inoculate.

These spirilla are found in spirals, like corkscrews, and are so delicate, as Nicolle says, that their flexible elastic bodies twist and untwist like a fine spring.

**Relative Importance of the Organisms in Pathogenesis.**—The bacteriological examination of the false membrane shows: the association of the bacillus and the spirillum in manifest predominance over the ordinary bacteria; or the bacillus accompanied only by the spirillum; or the bacillus alone. At the beginning of the disease the fusiform bacillus clearly predominates; as soon as there is ulceration one finds associated numerous spirilla as well as the habitual inhabitants of the region; then one sees the fusiform bacilli and the spirilla diminish in number and finally disappear when the period of convalescence begins. In case of relapse one finds a re-establishment of the symbiosis.

Direct examination permits us to distinguish three zones: A superficial zone where one finds especially the usual micro-organisms of the region; a middle zone where bacilli or spirilla exist alone; and a deep zone where one finds only fusiform bacilli. For Vincent, the bacillus plays the principal rôle, the spirillum and the other germs a secondary rôle. Reiche thinks that "the absence of spirilla has no great significance." Dopter believes likewise in the specificity of the bacillus of Vincent; on the other hand Letulle denies it. However, the spirillum would seem to intervene in ulceration. Delseaux considers that there are two well defined clinical types, according to whether the fusiform bacillus is alone or associated with spirilla; in the first case the lesion is of pseudomembranous aspect, in the second case it is ulcerative. The spirillum would also seem to play an important part in the genesis of general complications, and the same author, commenting on a case of "pseudomembranous angina followed by death from pernicious anemia," has the impression that this anemia is due to the penetration of spirilla into the blood.

Finally, since the appearance of treatment of the spirochetoses with novarsenobenzol, and the results obtained in fusospirillar infections, several authors (Archard, Herlich, Simonin) believe that not the fusiform bacillus, but the spirillum plays the principal rôle. However, a recent case of "ulcerative stomatitis due to fusiform bacilli without spirochetes," observed by Professor Sabrazes (*Gazette Hebdomadaire des Sciences Médicales*, Bordeaux, November 4, 1923), seems to show that the spirochetes play a minor rôle in the symbiosis of Vincent. The author affirms the truly gangrenous action of the fusiform bacillus (which showed itself resistant to bismuth) and, on the other hand, the aggressiveness of the fusiform bacillus without its habitual associate the spirochete, which does not exist in the lesions, having probably been annihilated by the bismuth. He calls attention also to a polynucleosis (without notable anemia) in the course of this invading ulcerative stomatitis, contrasting with the lymphocytoses and monocytoses of high degree attributed to fusospirillar lesions.

But, though the bacteriologists still discuss the respective importance of the bacillus and the spirillum, all the clinicians agree in recognizing the existence of a "fusospirillar infection, whose manifestations may be multiple" as we have just shown in the preceding paragraph.

**Complications.**—These may be local or general.

*Local.*—The most important and most grave immediate complication is asphyxia, which may result either from edematous infiltration of the

glottic and subglottic mucosa or from the immobilization of the vocal cords in the median position by double crico-arytenoid arthritis.<sup>6</sup> Respiratory embarrassment may also result from more or less extensive fibrous synechiæ uniting the vocal cords, or following perichondritis such as one observes after typhoid fever, variola, or syphilis.

Finally, a rather frequent complication is the extension of the fusospirillar infection to the trachea, the bronchi, or even the lungs.

*General.*—There may be phenomena of general intoxication, especially a very grave sort of pernicious anemia. Delseaux<sup>7</sup> even supposed that the spirochetes could penetrate the blood, and he advised, in grave cases, seeking the pathogenic agent in the patient's blood-stream. Let us point out finally among possible complications: albuminuria, gastro-intestinal disorders, and disturbances in the liver, heart, and central nervous system. Even cases of arthralgia<sup>8</sup> have been noted, coming on toward the decline of life.

*Prognosis.*—The prognosis is extremely variable. Entirely benign in its milder forms, and leaving no trace of its presence, this disease is at other times of extreme gravity. Death is rare, but possible: Dr. Texier<sup>9</sup> reported a fatal case in a young man, twenty-one years of age. A fatal termination may be due to cachexia, or come as a result of a visceral complication.

*Diagnosis.*—The differential diagnosis of ulceromembranous laryngitis must be made from a certain number of laryngeal affections.

*Acute catarrhal laryngitis* does not resemble it in any respect, since there exists neither ulceration nor infiltration nor membranous exudate. Likewise, acute serous edema of the laryngeal mucous membrane would not be confused; only laryngitis with ulcerative lesions and membranous secretions need be distinguished from the form which we are discussing. Acute non-diphtheritic inflammation of the larynx with simple membranous exudate is not accompanied by secretions; there exists little or no infiltration of the mucosa, and bacteriological examination does not reveal the presence of staphylococci, pneumococci, or yeasts, nor of fusiform bacilli or spirilla. The development is more rapid, more acute, and the duration is shorter, even if it has been necessary to perform a tracheotomy to prevent the asphyxia which is always possible in such a case on account of the presence of thick and buffy false membranes in the interior of the larynx.

*Laryngeal Diphtheria.*—True membranous croup is very rare. It is a *disease of childhood*; we have seen that the contrary was true of fusospirillar laryngitis. Generally a characteristic angina coexists or precedes. From the beginning the temperature is elevated, the general condition bad; there exists generally some submaxillary and carotid adenopathy, which is rare in the ulceromembranous form; the voice is faint and croupy in character, and the secretion almost none. The false membranes are grayish, not very thick, and discrete, following the form of the mucosa, which is simply eroded, without presenting any very notable infiltration nor true ulceration.

Finally, the bacteriological examination indicates the presence of the Klebs-Löffler bacillus—long, medium, or short, according to the severity of the infection.

The course of this disease is typical, usually going on toward cure if treatment is administered in time, or toward a fatal termination in the

severe cases or when treatment is delayed. The outcome supervenes, therefore, in the space of several days. Albuminuria is frequent in diphtheria, rare in ulceromembranous laryngitis.

*Variola*, *scarlet fever*, or *typhoid fever* may cause ulcerative lesions in the larynx, but the existence of the characteristic eruptions, or the history of a pre-existing typhoid fever will suffice to establish the diagnosis.

*Syphilis*, in the *secondary* period, is much more discrete in its laryngeal manifestations, and the simultaneous appearance of other features of this period (mucous patches in the throat, the anus, the genitalia; alopecia; nocturnal headache, etc.), not to mention the Bordet-Wassermann, is sufficient evidence to establish a definite diagnosis.

In the *tertiary* period the ulcerations are deeper, more anfractuous, with sharply defined, punched-out borders. The base of the ulcer is often angry looking, and it is rare that the entire larynx is invaded, the lesion habitually limiting itself to a part of the organ. The Bordet-Wassermann is often positive.

Finally, *acute tuberculosis* of the larynx is ordinarily a complication of an acid-fast infection of the lungs, and the general condition of the patient bears the mark of it. Difficulties in swallowing are sometimes of such severity that the patient cannot get sufficient nourishment, and the voice is faint. On examination one finds a considerable discoloration of the pharyngeal mucosa, of the soft palate, etc., as well as of the entire larynx, the infiltrated mucosa of which is covered with little ulcerated craters, and often with a sowing of little yellowish granular elevations (like grains of wheat), the whole covered with a purulent but not fetid secretion that the patient expels with difficulty.

The bacteriological examination generally shows Koch's bacilli in more or less abundance.

The slow or chronic forms of tuberculous laryngitis would not confuse us long, since the rapidity of development of the Vincent's infection constitutes a diagnostic point of the first order. Moreover, the local lesions observed do not resemble in any respect those of ulceromembranous laryngitis, an affection of essentially acute type.

**Treatment.**—This will be both local and general.

*Local Treatment.*—Thorough lavage of the pharynx and mouth should be prescribed, preferably with alkaline solutions, such as Vichy water, sodium borate, sodium benzoate, in a strength of a teaspoonful to the pint of solution, to which one may add several drops of spirits of peppermint, tincture of aniseed, or eucalyptus.

In cases with abundant suppuration, where there is a heavy exudate, and ulceration of the mucosa, it may be desirable to prescribe lavages to be given two or three times a day with an enema syringe, using one of the solutions described above, or a sodium perborate solution, freshly prepared in the following way—

Hydrogen peroxide.....	1 part
Boric acid (5 per cent.).....	4 parts

which may also be perfumed as described above. The injection should be administered, if possible, during expiration, in order to avoid the introduction of the liquid into the hypopharynx, and especially the air-passages.

If the injection is not very well tolerated it may be replaced with a spray, using the atomizer of Richardson, in which is placed a solution such as the following—

Sodium salicylate.....	8 grams
Antipyrine.....	4 “
Cherry laurel water.....	50 “
Sterile water.....	450 “

It will also be well to touch the affected parts either with an alkaline solution or with 1 per cent. methylene blue; or to insufflate into the affected parts, even into the larynx, once in twenty-four to forty-eight hours, a small amount of neosalvarsan. This last medicament may be mixed with glycerine and applied with the aid of a swab to the ulcerated areas. The insufflation should be done preferably with the aid of a Lubet-Barbon laryngeal insufflator (Fig. 436).

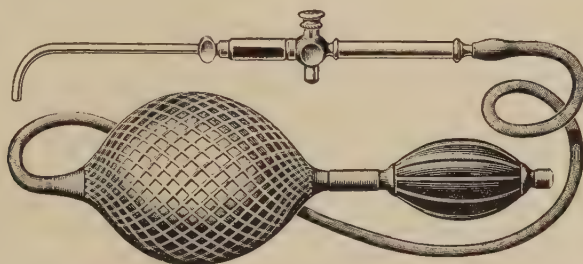


Fig. 436.

*General Treatment.*—It will be well to administer two or three tablets of potassium chlorate in twenty-four hours, the patient allowing them to dissolve in his mouth. This drug has a specific action on the fusiform bacillus. The chlorate absorbed by the gastro-intestinal tract is eliminated by the salivary glands, and for this reason it must not be used to excess; as a troublesome erythema may result. Vichy water (*source des Célestins*) should be prescribed, either alone or with milk. Finally, in severe cases, in order to raise the patient's power of resistance, it may be advisable to use physiological serum in a dosage of 200 to 250 grams in twenty-four hours.

In the stenotic forms, if the respiratory embarrassment is such that the life of the patient is in danger, one must not hesitate to perform a tracheotomy. This should be done, not in the cricothyroid membrane, but in the trachea itself, preferably at the level of the first ring.

As the infection diminishes or disappears, it will be advisable to prescribe arsenical preparations, phosphates, or even strychnine and its derivatives (bitter drops, tincture of nux vomica, etc.). If these drugs cannot be taken by mouth, subcutaneous injections of sodium cacodylate may be substituted.

*Diet.*—Finally, throughout the duration of the disease one must forbid all sorts of shell-fish, fish, white and red meat, and even bouillon, which is very irritating. The patient should be given a soft diet: rice and milk, tapioca and milk, unspeiced pastry, and eggs if well tolerated. It is only in the period of convalescence, when all traces of local infection have

disappeared, that the diet may include meat, commencing with brains, calves' sweetbreads, white meats, etc.

**Treatment of Complications.**—The complications should be treated by appropriate means, concerning which we need not speak at length. We shall simply recall the fact that if it has been necessary to insert a tracheal cannula to prevent asphyxia, the cannula should not be removed until direct examination has shown that air passes into the trachea normally and through a sufficient airway. If the larynx remains stenosed to the extent that the cannula cannot be removed, one should not begin the dilatation until after the local and general phenomena of the disease have completely disappeared. This grave complication may then be treated by methods that are well known today, of which laryngostomy and dilatation by means of some sort of rubber plug will probably give the best results. We shall not discuss this part of the treatment further, but refer the reader to the article on chronic stenosis of the larynx.

E. J. MOURE.

(Translated by Chevalier L. Jackson.)

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#### MEMBRANOUS LARYNGITIS (NON-DIPHTHERITIC)

**Definition.**—A form of laryngitis associated with the formation of a false membrane, but not due to *Bacillus diphtheriæ*. It is sometimes called *false croup* also *fibrinous laryngitis*. It is rare compared to diphtheria. It may complicate measles or scarlatina, or may occur as a primary disease. The form due to Vincent's infection is considered separately in the article by Professor Moure (p. 823). The inflammatory condition with membrane formation, following trauma, war-gases, caustic burns, and scalds does not constitute a membranous laryngitis as the term is here used.

**Etiology.**—The cause is usually bacterial, though no one kind of organism has been invariably found; perhaps streptococci occur most frequently. At the Bronchoscopic Clinic almost every variety of pyogenic organism has been found to predominate in certain cases, though absent or scarce in others. *Bacillus coli communis* and *B. pyocyaneus* have been found in almost pure culture in a number of instances. Mycotic organisms

were present in the false membrane and in the tissues in a number of patients. Measles and scarlatina may be factors in producing vulnerability. Membranous laryngitis is less common in children than in adults.<sup>1</sup>

**Symptoms.**—The chief symptoms are hoarseness, croupy cough, and a feeling of discomfort in the region of the larynx. A little mucoid secretion may be raised; rarely, and only in the later stages, a bit of membrane may be coughed out. The patient, if an adult, does not complain of feeling ill; the temperature is not elevated, nor is the appetite impaired. Children usually manifest systemic disturbance; severe prostration, and other toxic symptoms come on early. Cardiac failure and pulmonary complications are common.

**Laryngoscopic Appearances.**—In adults and older children mirror examination will reveal an inflammatory larynx with large patches of grayish-white exudate. Direct laryngoscopy without anesthesia, general or local, is called for in children too young to co-operate in mirror examination. It not only reveals the exudate, but enables the examiner to remove portions of the membrane and to obtain swab-specimens for smears and cultures. The membranous exudate will be found tightly adherent.

**Diagnosis.**—A membranous laryngitis should always be regarded as diphtheritic until proved otherwise. The diagnosis can be made only by the failure to find diphtheritic bacilli by smears and cultures made from specimens removed directly from the larynx. Under no circumstances should we be misled by reports on specimens taken from faucial secretions. Direct laryngoscopy affords the only practical way of obtaining uncontaminated swab-specimens from the larynx. Trauma, caustic burns, scalds, and war gases usually show their results on the pharynx and fauces.

**Prognosis.**—The prognosis is serious in children, especially when associated with measles or scarlatina. As a primary disease it is less serious. In adults recovery is the rule. Neglect of early tracheotomy for obstructive laryngeal dyspnea is probably the cause of most deaths.

**Treatment.**—The most important thing is to watch for signs of obstructive laryngeal dyspnea, and to do a tracheotomy early. It must be done low. If in emergency a high tracheotomy be done, a low one should follow as soon as the danger of impending asphyxia is past. Wearing a cannula in a high tracheotomy wound will certainly result in difficult decannulation and chronic laryngeal stenosis. Next in importance to the prevention of asphyxia is the prevention of cardiac failure. Judicious use of cardiac stimulants requires the good judgment of the pediatrician or internist. The disease is self-limited. As it is difficult to be certain in the early stages that the disease is not diphtheria, administration of antitoxin is usually justified, and quarantine is a wise precaution.

Local treatment is useless; but vaporization of compound tincture of benzoin from hot water, and menthol similarly used, are standard remedies.

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## HERPES OF THE LARYNX

**Definition.**—An eruption of one or more vesicles on an erythematous base.

**Incidence.**—Herpetic eruptions are not so uncommon in the larynx as the paucity of literature would seem to indicate. Usually they are accompanied by a similar eruption on the posterior pharyngeal wall, the pillars of the fauces, the velum, and uvula.

**Etiology.**—The etiology of herpes is explained rather unsatisfactorily on theoretic bases in the general text-books. There is no known reason why it should localize in the larynx.

**Herpes simplex** occurs in the larynx in association with herpes of the lips and mouth at the onset of acute infective diseases, especially those of the type called "colds." This is the so-called "fever-blister." In the larynx it is seen as a vesicle which leaves a superficial ulcer that quickly heals. The vesicles may be in the intrinsic or extrinsic area. It is usually painless, though sometimes a subjective sense of irritation or even pain may be felt. The differential diagnosis from Vincent's infection and aphthous ulcer is based upon the evanescent character of herpes simplex. Vincent's angina is a severe local lesion, whereas herpes simplex is merely an incident in a systemic disturbance. In pemphigus of the larynx the associated lesions of the skin are diagnostic. Vincent's infection is easily diagnosed by microscopic examination of fresh secretion. It usually does not require treatment; but if any be used, it should be of bland and unirritating character, such as an oily spray. Nitrate of silver is very objectionable as an application in such cases. It will sometimes cause a new crop of vesicles. Any application on a swab is bad for this, as for most other laryngeal diseases.

**Recurrent herpes of the larynx** is characterized by successive crops of vesicles which leave chronic, superficial, sometimes painful ulcers. They may last for weeks, and may leave a thickening, but usually do not leave a visible cicatrix. New vesicles appear at the same or a new site. Chronic stenosis of the larynx may result, and in a few cases tracheotomy is required. The bacteria vary in different cases. The diagnosis requires the exclusion of aphthous ulcer, syphilis, tuberculosis, Vincent's infection, blastomycosis, and actinomycosis. Local applications are of little use; silver nitrate solutions will, in some cases, produce a fresh crop of vesicles. Arsenic internally and bismuth subnitrate dry on the tongue in small doses for local effect, are the best remedies.

**Herpes Zoster.**—Though rare and usually self-limited this is a very important disease, because of the distress it causes and the difficulty in some cases of affording permanent relief. The eruption may affect any part of the larynx, and even the subglottic area. Fever and pain are early symptoms, then the vesicles appear and soon rupture, leaving superficial erosions; these heal in a week or two, and sometimes leave a scar. In a small percentage of cases the disease assumes a chronic form, the ulcers become sluggish, healing is tardy, and successive crops of vesicles maintain the constant presence of the superficial ulcers. Dysphagia and odynphagia are usual, and often marked. The diagnosis rests chiefly on the presence of herpes zoster in other parts of the body, the mixed, non-specific character of the bacteriological findings, the exclusion of Vincent's infection, aphthous ulcers, syphilis, tuberculosis, lupus, pemphigus, contact ulcer,

and varicella. Pain and high fever, 103° to 104° F., even a rigor, are characteristic of herpes zoster at the onset. Microscopic examination of fresh scrapings from the ulcer, with dark-field illumination if desired, is important to exclude spirochetosis. The other points in differential diagnosis will be found under the respective headings. Dr. Moure's classic chapter on "Ulceromembranous Laryngitis" should be carefully studied in this connection. The *prognosis* is usually favorable. The rare chronic cases may drag along for months; but if asphyxia be guarded against by prompt tracheotomy, there is little danger to life.

**Treatment** is directed toward relief of distress in the self-limited cases. Orthoform insufflations and bismuth subnitrate dry on the tongue are two of the most efficient local remedies. Silver nitrate usually provokes new lesions. If the disease tends toward the chronic form the systemic treatment according to indications revealed by thorough and complete study by the internist becomes of the utmost importance. Rest in bed and increased elimination constantly maintained by bowels, skin, and kidneys are helpful measures in any case.

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### APHTHOUS ULCER OF THE LARYNX

This disease occurs in two forms. It may occur in association with aphthous stomatitis of children, or as an intermittent disease associated or alternating with aphthous ulcer of the mouth, palate, fauces, pharynx, tongue, or lip. While it may be due to local infection, intestinal toxemia is a basic factor, recognition of which fact is of utmost importance in treatment. For diagnosis, Vincent's infection, herpes, contact ulcer, and pemphigus must be excluded. For treatment and prophylaxis, all that is necessary is to give a small dose of compound cathartic pill twice weekly. Hypercatharsis must be avoided by reducing the dose to the point where only one free movement is produced.

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### ANGIONEUROTIC EDEMA OF THE LARYNX

**Definition.**—Angioneurotic edema is the name given to a disease characterized by transient, circumscribed, edematous swellings appearing on mucosal or epidermal surfaces or both. The larynx alone may be involved, but more commonly there are associated lesions in the gastro-intestinal tract, esophagus, mouth, tongue, pharynx, lips, eyelids, skin, or genitalia.

**Synonyms.**—Quincke's disease; blue edema (Charcot); urticaria of the larynx.

**Etiology.**—Heredity is certainly a factor in many cases, though it cannot be traced in all. Of 170 cases of hereditary angioneurotic edema, 36 died of edema of the larynx.<sup>1</sup> Sensitiveness, congenital or acquired, to foreign proteins has been clearly demonstrated in many cases. I have observed a case following injection of antitoxin. Anaphylaxis to unknown

substances has been assumed to be a cause. The type of individual in which the disease usually occurs and its association with allergic, neurotic, and hysteric manifestations suggest a neurotic etiologic factor. Worry, grief, exhausting work, excessive social activities are contributing causes in some instances.

**Pathology.**—The transudation of serum implies a dilatation of blood-vessels in the area involved and the inference is that it is a vasomotor disturbance with temporary increase in the permeability of the walls of the vessels in the affected area. Local hemorrhage into the tissues may occur giving rise to purpuric spots on the dermal or mucosal surfaces; the mucosal lesion may be decidedly hemorrhagic in character. The swelling may disappear in a few hours or days and leave no trace. In some cases, however, prolonged chronic edema may result from many attacks in the same location, each attack leaving a small amount of residual swelling; or a single attack may leave a chronic edema, lymphangiectasis or a lymphangioma. This may be the case in the larynx. Postmortem findings in a case reported by Koenig are typical. The patient was a physician aged sixty-five years, and hoarseness and dyspnea came on suddenly while he was lecturing. He was rushed to a hospital for tracheotomy, but died while the operation was being done. Autopsy revealed no edema except in the laryngeal region. Marked edematous swelling began at the base of the tongue and extended on the left side into the pharyngo-epiglottic ligament, on both sides of which the valliculæ were changed to pear-like sacs. The ary-epiglottic folds were enormously swollen gelatinous appearing masses, flattened on their median contactual surfaces, and occluding the upper orifice of the larynx. There was no edema of the cords. Microscopic sections of the edematous areas showed edema of connective muscular tissues. There was marked infiltration with leukocytes, lymphocytes, and plasma-cells. There was a great preponderance of eosinophils in the edematous tissue and in the blood-vessels. Very many mast-cells were noted.

**Prognosis.**—The prognosis of laryngeal angioneurotic edema is bad unless tracheotomy be done or an expert tracheotomist be kept in the house. Many patients have died on the way to a hospital. Statistics show that in about 33 per cent. of the cases the patient asphyxiates, obviously for want of a prompt tracheotomy. The disease is rarely if ever fatal, otherwise than by asphyxia from laryngeal obstruction. Recurrences are frequent unless allergic, general, or neurotic causes can be discovered and successfully treated.

**Laryngoscopic Appearances.**—In a typical case mirror examination shows the laryngeal image to be replaced by two globular masses one on each side of the base of the tongue (Plate VIII). If the epiglottis is uninvolved its tip may be seen pressed against the tongue by the globular masses; if the epiglottis is involved it shows as an edematous transverse roll pressed tightly forward against the base of the tongue (Plate VIII). The color of the swelling in most cases is paler than that of the normal mucosa, and may be white or pale yellowish pink. Rarely it may be reddish, bluish, or purplish in tinge. In all the cases that I have seen the swellings lacked the translucent bluish watery appearance similar to that of edematous nasal polypi, that characterizes the inflammatory and nephritic edemas of the larynx. A small supplementary nodule of violet or bluish color, as shown

## PLATE VIII



Angioneurotic edema of the larynx. The three circles represent the mirror view in the same patient at different times. The nodular mass anteriorly in all three illustrations represents the lymphoid tissue at the base of the tongue; it was in a state of chronic folliculitis.

At the left is shown the larynx when first seen. The epiglottis is thickened. The aryepiglottic folds are thickened posteriorly, the left one more than the right; both overhang forward over the posterior ends of the apparently normal cords. The thickenings were considered the residue of previous attacks of angioneurotic edema affecting the larynx and also the upper lip. The lip also showed a residual thickening in its central portion.

The central illustration shows the condition of the same larynx a few days later. The patient had awakened with dyspnea accompanied by subjective sensations of irritation in the larynx. The epiglottis is a thickened roll; both arytenoids are swollen; a small violet colored nodule resembling an obstructed vein is seen at the right side.

In the circle at the right is illustrated the condition of the larynx three months after the sketch shown in the central illustration was made. The swelling had quickly subsided to a half-way stage; but had remained stationary in the condition here shown. The thickening of the epiglottis and left arytenoid remained stationary. The mass of chronic edema on the left side was probably lymphangiectatic in structure.



in Plate VIII, has been present in a few cases. In only 1 case did the masses give the impression that puncture would liberate water, as is so often the impression given by the acute inflammatory and nephritic edemas. The cords are usually visible in the inflammatory edemas and are seen to participate in the inflammation and usually in the edema also. In angioneurotic edema, on the other hand, the cords appear thin and white if they can be seen. Usually they are hidden from the mirror view by the two huge, edematous, usually symmetrical, masses that bulge upward, one from each arytenoid region.

**Symptomatology.**—Hoarseness may be slight or absent. Other symptoms are cough, local sensations of heat, irritation, and jaggings or pricking, indrawing at the suprasternal notch, wheezing, and stridorous breathing. The dyspnea may increase rapidly until asphyxia supervenes. The disease may possibly be associated with asthmatic symptoms but all of the case reports strongly suggest that the so-called "asthma" was really obstructive laryngeal dyspnea. The general symptoms other than the dyspnea are those of the associated lesions. Gastro-intestinal symptoms—abdominal pain, vomiting, melena—and urethral and cystic irritation are often accompaniments. The general temperature is not elevated as a rule, and the local temperature of the dermal swellings is lowered. The dermal swellings resemble giant-sized urticarial lesions, though the itching is usually less. The lesions may last a few hours or a few days, and usually disappear without leaving a residual lesion. Recurrences may be at the same locations or at other sites; the larynx may be affected in one attack and not in another.

**Diagnosis.**—Anyone familiar with the mirror appearances of the common inflammatory and tuberculous edemas of the larynx will be struck by the different appearance of angioneurotic edema in this region; but it is difficult if not impossible to describe the difference in a way that would be useful to one not familiar with laryngeal edemas. The inflammatory and nephritic edemas, as seen in the mirror, convey an impression of watery softness like that of an edematous nasal polyp. The angioneurotic edema does not look as if a puncture would liberate water; it appears to be of a greater firmness. When palpated it is soft, though not as soft as the acute inflammatory type of edema. It should be emphasized here that palpation in any case should be gentle, and should not be done without having already made due preparation for immediate tracheotomy, should necessity for it be precipitated. This applies with especial force to angioneurotic edema. Diagnosis of angioneurotic edema of the larynx, as distinguished from other laryngeal edemas, rests in some cases on the association with the characteristic transitory edemas elsewhere in the body either in the same or previous attacks. Very searching inquiry will usually elicit a history of significant data. Even in the absence of such a history an acute edema of very sudden onset, associated with pricking sensations, but without involvement of the vocal cords and unaccompanied by the usual symptoms and mirror appearances of acute laryngitis is apt to be of the angioneurotic type. It is especially so when occurring in a patient known to have had a previously normal larynx and in whom there is no nephritis or other organic disease. On the other hand a laryngeal edema developing upon an acute laryngitis with inflamed cords, and accompanied by tracheitis and rhinitis would not be regarded as angioneurotic. The edemas of

tuberculous laryngitis would present no difficulty in differentiation from the acute stage of angioneurotic edema; but the lymphangiomatous or lymphangiectatic conditions that sometimes follow repeated attacks of the acute disease might mislead. Careful study and the application of the usual diagnostic steps in exclusion of tuberculous laryngitis will usually solve the difficulty. One condition, often overlooked, may cause a varying edema of the arytenoid eminences; namely, ulceration of the anterior wall of the hypopharynx. Ulceration in this region, whether of the syphilitic, malignant, or traumatic types may be invisible below the level of the arytenoid and aryepiglottic edematous masses of which they are the primary cause. The normal vocal cords in such cases render these conditions especially misleading. Direct inspection of the hypopharynx with the direct laryngoscope will always determine this point. In a number of such cases I have set aside a diagnosis of angioneurotic edema upon finding an acute inflammatory condition due to an embedded spicule of bone; in other cases a cancer. The non-inflammatory character of angioneurotic lesions furnishes a basis for excluding Ludwig's angina and all other acute infective conditions.

**Treatment.**—The literature of this disease is an appalling record of asphyxia for want of a tracheotomy. It cannot be too strongly urged that the outstanding characteristic of angioneurotic swellings anywhere, as compared to inflammatory edema, is the suddenness of their development. The swelling comes in some cases in a few minutes rather than a few hours. I have seen the larynx almost completely closed in seventeen minutes after the onset of the first symptom. If a skilled tracheotomist with complete outfit in readiness cannot be in constant attendance tracheotomy should be done before the patient is *in extremis*. "We always preach doing tracheotomy early but always do it late" should be in mind in dealing with this dangerous disease. Local sprays of adrenaline and cocaine solutions are helpful. All irritant medicaments and the use of swabs are strongly contraindicated. Puncture seems inadvisable. Absolute physical rest in bed combined with a régime of absolute silence is advisable. The patient should not utter a word and all relatives and visitors should be excluded. Under this plan of unirritative management the spontaneous tendency to recovery will usually result in complete subsidence of the lesion. It then becomes urgently necessary to search for etiological factors with a view to prophylaxis. As soon as the laryngeal swelling has subsided sensitization tests may be started. Signs of reaction in the larynx as seen in the mirror are in some instances a better index than the local dermal reaction. Bacterial sensitization should be considered in etiological studies, and focal infections should be searched for, and dealt with if found; but operations on the nose and throat are contraindicated unless the indications for them are very strong and clear. It is of the utmost importance that the patient's usual environment and habits of life be studied. Nearly always there will be found anxiety, worry, grief, overwork, unsatisfied ambition, social activities, late hours or similar factors tending to undermine the patient's health. I have obtained an immediate and complete cessation of recurrences in every patient whose full co-operation I could obtain in the following of a tranquil life free from fatigue, worry, ambition, late hours, and similar factors as above enumerated. Endocrine studies are important and should be carried out, but my experience justifies the inference that

whatever endocrine imbalance was present was the result of the habits of life just referred to, and that the evidence of imbalance disappeared under the co-operative régime described.

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### SERUM DISEASE OF THE LARYNX

The dermal urticaria, the toxic, and the other phases of the serum reaction are so distressing that the presence of a reaction in the laryngeal mucosa is often overlooked. The laryngeal manifestations vary from a passive engorgement to an edema that may require tracheotomy. The diagnosis is made with the mirror and the treatment is the same as that for angioneurotic edema (*q. v.*).

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### URTICARIA OF THE LARYNX

The indistinct differentiation of this disease from angioneurotic edema has seemed to justify considering the two diseases (if indeed they are two distinct diseases) together under angioneurotic edema of the larynx (*q. v.*).

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### FIBRINOUS CORDITIS

**Definition.**—An inflammatory lesion associated with a thin fibrinous exudate localized on a limited area of the vocal cords. The term is not intended to include all cases of non-diphtheritic fibrinous exudative lesions of the larynx.

**Incidence.**—The disease has been rarely reported; but it may have occurred frequently under conditions preventing laryngoscopic examination.

**Etiology.**—Brown Kelly,<sup>1</sup> to whose observations we are indebted for data, found the disease to follow exposure to irritants such as cold, gas, tobacco; and rarely influenza. Archer Ryland<sup>2</sup> found a number of typical cases during the war in soldiers who had been exposed to mustard gas. The location of the lesions, as Ryland has observed, corresponds to the portion of the cords that never coapt so tightly as to prevent passage of air.

**Symptoms.**—The chief symptom is hoarseness; but even this may be very slight. Some patients are aphonic. Cough may be present, but pain is usually absent.

**Laryngoscopic Appearances.**—Mirror examination reveals, in a typical case, a pair of symmetrical white patches on opposite surfaces of each

cord slightly anterior to the middle of the cords, the two patches together forming an oval. Anterior and posterior to the patches, the cords are pinkish, but not intensely inflammatory. The general endolaryngeal mucosa may be normal. At a later stage the oval patches of exudate fade away and disappear in two or three weeks, the final stage showing a small white spot at the edge of each cord. The disease is not ulcerative, though small temporary erosions have been observed.

**Prognosis.**—If the cause is not repeated complete spontaneous recovery within a month may be expected.

**Treatment.**—The disease seems to be self-limited. All that is necessary is rest of the voice and freedom from air-borne irritants. All irritant local applications are absolutely contraindicated.

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### LARYNGEAL HEMORRHAGE

**Definition.**—Extravasation of blood into the laryngeal mucosa, or escape of blood into the larynx. *Hemorrhagic laryngitis* applies properly only to those cases associated with inflammation. *Hematoma* (*q. v.*) is applied to a tumor-like collection of blood. *Purpura laryngis* is a particular form of that disease.

**Etiology.**—Vocal abuse is usually the exciting cause. Vascular and blood diseases (purpura, leukemia, chlorosis, etc.) may be factors. Adherent crusts and their detachment may cause hemorrhage; it is usually slight if the coagulation time is normal. Some other form of trauma may be a cause. Severe infectious disease, such as typhoid fever, may cause either free bleeding from, or purpuric spots on, the laryngeal mucosa. Hemorrhage is very rarely the result of sloughing abscess. Postoperative hemorrhage is elsewhere considered.

**Symptoms.**—Hoarseness, slight cough, hemoptysis, and subjective discomfort may be present. The blood may be only streaks in the sputum, or a succession of expectorations of blood or clots. The symptoms of laryngitis are usually present, and local inflammation is rarely absent.

**Diagnosis.**—Inspection of the interior of the larynx (with the mirror in adults, with the direct laryngoscope in children) is the only means of determining that the blood comes from the larynx itself. If the hemorrhage is under the epithelium it can be plainly seen. Usually it is on one or both cords.

**Prognosis.**—Hemorrhage from the larynx usually ceases spontaneously; if not, it can be stopped. Extravasated blood is usually absorbed, but may result in a hematoma, requiring operative removal. Recurrences are likely unless the patient ceases vocal abuse. Ulceration and perichondritis (*q. v.*) may be sequelæ.

**Treatment.**—Hemorrhage into the mucosa or from laryngeal vessels usually ceases under strictly enforced silence and rest in bed. The patient

should write every word he has to say. Saline laxatives are helpful. Local applications are usually unnecessary and harmful. A spray of adrenaline with a little cocaine in the solution is usually all that is necessary. Tucker's method of injecting adrenaline with a hypodermic needle has been used with prompt and satisfactory results at the Bronchoscopic Clinic. The needle is inserted through the cricothyroid membrane and guided upward to whichever side the bleeding-point lies. The injection is not into the lumen of the larynx, but into the tissues beneath the bleeding-point. In a few instances we have touched the bleeding-point with the galvanocautery, through the direct laryngoscope, though this is a method always to be used with great caution and precision in the larynx, lest perichondritis and chondrial necrosis follow. Postoperative hemorrhage may require packing the larynx, to which, of course, tracheotomy and the insertion of a tracheotomic cannula would be a preliminary requirement. Laryngeal packs must be with large gauze squares, not tape-like strips.

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### PERICHONDritis OF THE LARYNX

**Definition.**—An inflammation of the perichondrium of any of the cartilages of the larynx.

**Etiology.**—Perichondritis is due to infection, either as a primary focus or as an extension. The commonest causes are syphilis, tuberculosis, cancer, typhoid fever, diphtheria, high tracheotomy, accidental and surgical trauma. In many cases seen at the Bronchoscopic Clinic the trauma was received in coasting and automobiling accidents, falls across a bar, or the edge of a stair-tread, attempted suicide, radium and Roentgen-ray burns, etc., but by far the most common cause was high tracheotomy.<sup>1</sup> Radium and Roentgen-ray burns have in recent years been the cause in many cases. Many a child has come in with the laryngeal cartilages completely destroyed and the whole larynx as effectually obliterated as if a laryngectomy had been done, as a result of excessive radiation for multiple papillomata of the larynx—a benign self-limited disease! Typhoid fever was formerly a very common cause, some hundreds of cases having come under the author's care at the Western Pennsylvania Hospital in Pittsburgh.<sup>2</sup> Cancer does not readily penetrate hyaline cartilage. When it goes through the laryngeal cartilages the way is usually opened through by the mixed infections, perichondritis, and chondrial necrosis. Undoubtedly the galvanocautery point, used with great delicacy and precision, is a valuable remedy in tuberculous edema of the larynx; but anything approaching the technic of the surgeon removing a superior maxilla with a soldering iron will certainly cause perichondritis, necrosis, and obliteration of the larynx. Overlooked foreign body in the larynx is a by no means rare cause of laryngeal perichondritis.

**Pathology.**—Syphilis and tuberculosis and their secondary suppurative infections may reach the perichondrium by extension, or a primary focus may originate in the perichondrium. If the inflammatory process starts as a primary focus of specific infection it soon becomes a mixed infection. The nutrition of the cartilage soon is impaired, necrosis sets in and extends

as the perichondrium becomes separated. The process may be rapid, or it may be prolonged for months. All of the particular cartilage involved may be destroyed (Fig. 437), or healing may leave islands. Usually dense fibrous tissue is formed and its contraction completes the ruin of the lumen of the larynx. All cases are not of this extent or severity. The perichondritis may be only a small spot or it may be limited simply to the edge of a divided cartilage. After a time necrosis of the edge ceases, or a small island of cartilage may be shed and complete and satisfactory healing take place.

**Symptomatology.**—The symptoms in the acute and the chronic forms are quite different. Acute laryngeal perichondritis as in trauma, diphtheria,

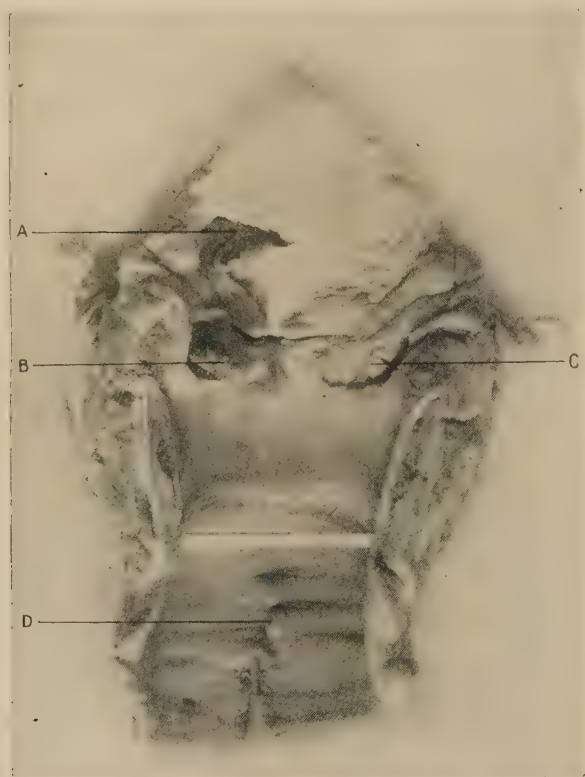


Fig. 437.—Photograph of specimen of larynx acutely stenosed by perichondritis complicating typhoid fever in a man aged forty years. *A*, Gap where specimen was excised postmortem. *B*, Necrotic left arytenoid cartilage hanging by a shred. *C*, Necrotic area from which right arytenoid cartilage necrosed and disappeared before death. *D*, Interior view of tracheotomic wound. Specimen lent by Major Frederick Russell, U. S. A. Patient of Dr. Joseph H. Bryan. (Peroral Endoscopy and Laryngeal Surgery by Chevalier Jackson, Text-book, p. 581, Fig. 437.)

or radium burns, may be accompanied by chilliness, fever, malaise, headache, severe local pain, and marked tenderness. On the other hand the chronic form such as accompanies tuberculous laryngitis or the breaking down of a gumma may show no systemic reaction, no pain, and little tenderness. There are many intermediate cases. The breath is usually fetid, and if the tracheotomic cannula is neglected the odor in the neighborhood of

the patient may be very unpleasant. The voice is usually somewhat altered and is often hoarse. Dyspnea is almost certain to supervene in the acute or very extensive cases or those in which both crico-arytenoid joints are fixed. It is most often the edema that causes the dyspnea.

**Mirror examination or direct laryngoscopy in children** reveals edematous masses meeting in the midline if the case is an acute and severe one. In chronic cases there may be very little amiss in the appearances. In some cases a fistula discharging pus is seen and a tender place can be found with the finger externally, locating the area from which the pus comes. Sequestra may be found in the fistula or protruding elsewhere. Granulations, often quite exuberant, are characteristic of necrosing cartilage. Nipping off what are apparently granulations for histological examination is of great importance apart from the question of perichondritis. A careful probing of the exuberantly granulating area will usually reveal necrotic or exposed cartilage if that is the cause of the granulations.

**Diagnosis.**—The diagnosis is usually made with little difficulty. If in the case of an acute or chronic inflammatory disease of the endolarynx, tenderness of the thyroid cartilage (not of the overlying skin) can be definitely made out, a tentative diagnosis is established; and the patient should be treated on that basis while being studied. The localized thickening at the tender point leaves little doubt as to the presence of perichondritis. When syphilis or tuberculosis or cancer is known to be present in the larynx, the thyroid and cricoid cartilages and first few rings of the trachea should be palpated frequently for evidences of perichondritis. If one or both of the arytenoids be fixed, palpation with a probe to determine passive mobility and consistency will reveal involvement of the perichondrium of this cartilage. Search for foreign body should always be made in every case of otherwise unexplained swollen and edematous larynx.

**Prognosis** depends upon the cause, severity, and extent of the perichondritis. Cases of mild degree and small area of involvement may subside leaving no sequelæ if promptly recognized and treated. Any very extensive loss of cartilage destroys the lumen of the larynx, because no resistance is offered to cicatricial contraction. Unsanitary, unclean, improper care, or rather lack of care of the tracheotomic cannula will subject the patient's life to great danger from sepsis, and mediastinal and pulmonary complications. When a proper daily, or in some cases hourly, toilet of the wound is carried out, there is very little danger to life. In the acute stage, with sudden onset of edema, many patients asphyxiate for want of a tracheotomy. After tracheotomy, with a good tracheotomic nurse in charge, the deaths will be very few. The prognosis as to decannulation and cure of chronic stenosis depends on the amount of cartilage lost. If a good cartilaginous framework remains, the cicatricial part of the stenosis can be cured in almost every case, but the treatment requires many months. The prognosis of perichondritis complicating laryngeal tuberculosis depends upon the general prognosis. In syphilitic perichondritis recovery is the rule under treatment.

**Prophylaxis.**—The most important fact revealed by all the author's studies of this condition is that in many if not in most of the cases perichondritis could have been prevented or could have been arrested in the early stage. High tracheotomy should not be taught.<sup>1</sup> If anyone has

done a high tracheotomy and has saved a human being from asphyxia he should not be criticized. But after the patient has sufficiently rallied to be trusted to do his own breathing, before the surgeon leaves him, a second tracheotomy should be done low down so that it will not be necessary to wear the cannula in the high position, in contact with the laryngeal cartilages. A careful toilet of the wound, as outlined in the chapter on Tracheotomy should be instituted. It is rare to see a patient come to the clinic wearing a cannula but that we find it has not been changed for days or weeks or even months! Often the cannula is corroded, covered with verdigris, and crusted with foul secretions. Needless to say such conditions favor the development of perichondritis. Soft rubber cannulæ are irritating, hard rubber cannulæ cannot be boiled, those of aluminum are roughened by boiling, plating soon wears off and becomes roughened. The wearing only of sterling silver cannulæ as made for us by Pilling, is a prophylactic measure of the utmost importance. In typhoid fever<sup>2, 3</sup> I found the most important prophylactic measure was oral cleanliness and oral antisepsis. Besides this, in diphtheria, of course early diagnosis and the early administration of antitoxin would probably prevent nearly all patients from developing perichondritis. In syphilitic patients with laryngeal lesions the administration of potassium iodide seems to favor the development of perichondritis; other antisiphilitics should be used, and especially the protiodide of mercury, keeping the patient barely under the salivation point for weeks. In tuberculous patients, absolute silence, the patient writing every word he has to say, and rest in bed outdoors for twenty hours out of the twenty-four are very important in the prophylaxis of the extension of a tuberculous lesion to the perichondrium.

**Treatment.**—All of the prophylactic measures mentioned in the foregoing section are valuable in treating the early stages of perichondritis. *Early tracheotomy*, done low and properly cared for (see the chapter on Tracheotomy), is a measure of the utmost importance. It puts the larynx at rest and drains away pus and purulent secretions that otherwise would accumulate in the subglottic space. If pus should accumulate under the external perichondrium it should be evacuated. Usually the pus is on the inner surface. If it can be located, evacuation is done with the long laryngeal knife through the direct laryngoscope, the aspirator being kept constantly in active operation. After that, with a good tracheotomic nurse in charge, properly equipped with a mechanical aspirator and proper aspirating tubes, there will be little or no danger of inspirated infections. (See After-care of Tracheotomy.)

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## ABSCESS OF THE LARYNX

**Definition.**—A localized collection of pus beneath the surface in any portion of the laryngeal structure. The older authors loosely called the condition *phlegmonous laryngitis*, and also *suppurative laryngitis*.

**Etiology and Pathology.**—As with abscess anywhere, the prime factor is the presence of pyogenic organisms. These may be any one or more of the pathogenic organisms encountered elsewhere. They may have been preceded by other organisms such as those of typhoidal, diphtheritic, syphilitic, mycotic, tuberculous, influenzal, and other infections. Thrombotic and embolic processes may be initial factors. The abscess is quite commonly associated with perichondritis (*q. v.*) and chondrial necrosis. The perichondritis may be the primary lesion, or the perichondrium may be reached by continuity or by vascular or lymphoid channels. The peculiar histological structure of the subglottic tissues in very young children, as demonstrated by A. Logan Turner, predisposes to edema and abscess. The areolar tissue between the epiglottis and the hyoid bone seems particularly vulnerable. Radium and Roentgen-ray applications for laryngeal papillomata in children and for cancer in adults are today one of the most common causes of laryngeal abscess, perichondritis, and chondrial necrosis. Abscess of the larynx may follow an acute tonsillitis, Vincent's angina, cervical cellulitis, etc. The disease may occur at any age; it is not uncommon in infancy.

**Symptomatology.**—Dyspnea, dysphonia, odynphonia, dysphagia, odynphagia, pain, tenderness, hoarseness, croupy cough, semicough, wheezing, or cyanosis may be present and indicate obstruction to the air- or food-passages. The dyspnea may be inspiratory or expiratory, or both. Swelling may be visible in the mirror or palpable externally, or both. Fever, malaise, anxiety, and sleeplessness are usually present in severe cases.

All of the symptoms may be masked by spreading infection.

*Laryngoscopic appearances* are those of intense acute inflammation plus a reddish swelling at the site of the pus collection. The swelling may be monolateral, bilateral, or central. The pus collection may be outside the wings of the thyroid, inside, or both outside and inside. The pus may be under the anterior or the posterior mucosal covering of the epiglottis, or under both anterior and posterior coverings. If it is under the anterior covering the epiglottis may be crowded down on the upper orifice of the larynx.

**Diagnosis** can be made only by laryngoscopy, direct, or indirect. The mirror cannot be used in very young children, but in these, direct laryngoscopy will promptly reveal the lesion. The direct method is usually necessary in adults also, for verification of the suspicions, and for accurate localization. Edema without abscess is usually bluish gray and semitranslucent, "watery" it is usually called, resembling an edematous nasal polyp. Inferential diagnosis is misleading and dangerous. All of the symptoms may be produced by foreign body, diphtheria, a neoplasm, influenza, and many other diseases, without a pus collection. In all laryngeal diseases diphtheria and syphilis should be excluded by laboratory methods as a matter of routine. Thymic enlargement may be revealed by the Roentgen ray, and this means should never be neglected; but the bronchoscopic method is the only way to determine whether or not the thymus

is obstructing the airway. Palpation with the finger through the mouth will reveal a laryngeal abscess if extrinsic. Palpation externally will reveal an abscess on the outside of the thyroid cartilage. If fluctuation is felt the diagnosis is almost certain, though a mucous, bronchial, or other cyst may require the needle for exclusion. Cysts are not usually accompanied by fever as are abscesses, but other causes of temperature elevation may coexist.

**Prognosis.**—If asphyxia be avoided by tracheotomy there is little danger to life. Recovery is the rule after surgical drainage. Laryngeal stenosis may follow, however, as a result of cicatricial contraction or, especially, necrosis of cartilage.

**Treatment.**—Evacuation of the pus is a prime surgical requirement, unless impending asphyxia calls for tracheotomy first. The abscess should be incised by the direct laryngoscopic method with the patient in the proximally inclined position. Incision by mirror guidance is not only inaccurate but is difficult with the patient in the recumbent low-head position required to prevent flow and inspiration of the liberated pus through the glottis into the deeper air-passages. External drainage is called for in externally palpable abscesses. The exploratory needle may be used first for confirmation if desired. If pus has not yet formed, the icebag externally and ice dissolved in the mouth are helpful. A laxative should always be given. The diet should be liquid.

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### BURNS OF THE LARYNX; RADIONECROSIS OF THE LARYNGEAL CARTILAGES

**Etiology.**—Burns of the larynx may occur by the penetration through the skin surface of hot irons. Quite a number of such cases occurred in the iron and steel mills of Pittsburgh; but such accidents have been eliminated by newer industrial methods. *Endolaryngeal* burns may be caused by the galvanocautery, molten metal, or other hot liquids, hot vapors, such as steam, caustic acids and alkalies, radium, the Roentgen ray, etc. Lye is sometimes swallowed with suicidal intent by melancholiacs; but most of the cases occur in children, who mistake the lye for sugar, or drink from a vessel whose dregs contain lye. *Silver nitrate* is a not infrequent cause of burns of the larynx. Strong solutions cause blisters followed by slowly healing erosions. The fused silver nitrate will cause ulceration that may take months to heal, and may end in stricture. Weak solutions are not only useless, but even they are actually harmful in the larynx. Solutions intended for the pharynx should not be allowed to drip into the larynx. Burns by war gases are considered in a separate chapter by my esteemed colleague, Professor Fernand Lemaitre. Sex is an etiological factor only in radionecrosis of the laryngeal cartilages, which is exceeding rare in women.

**Pathology.**—The first effect of a burn of the mucosal surface of the larynx is an acute inflammation in the area affected. If the burn has killed a portion of the tissue the inflammation is in the region surrounding the necrotic area. If the burn is a small one, as that produced by a skilful momentary touch of the cautery point, there is no visible edema. After more extensive burns of any kind, primary acute edema is always a marked feature, and clinically one of the most important. In addition to the primary pathological changes noted above there are other changes that though of the utmost importance clinically are in most instances so long in developing that they might be considered secondary. These pathological changes are perichondritis and necrosis. They may not be discoverable until weeks or even months after the burn. This is particularly true of the burns by the Roentgen ray and radium. The laryngeal cartilages are particularly sensitive to radiation, and ultimate total destruction of the laryngeal carapace may follow overdosage (See Perichondritis of the Larynx). As shown by Hautant,<sup>2</sup> the sensitiveness of the cartilages is chiefly at the site of the greatest calcification; namely, the inferior half of the median anterior angular part of the thyroid cartilages. This applies especially to men. The edema practically always associated with this late perichondritis and *radionecrosis* may be a continuation of the edema associated with the early mucosal reaction; or it may be a late development coming on weeks or months after the exposure. We may have then an early acute edema or a late chronic edema. In the late stage of these lesions there may be extensive sloughing of the soft tissues as well as of the cartilages; but in the early stages sloughing does not occur. In burns caused by boiling water, molten metal, household lye, and other caustic poisons there is usually sloughing if the burns are severe. Ulceration follows sloughing, and healing is slow. In the severe burns by irritation healing may not take place for years, if ever. As elsewhere in the mucosal surfaces of the body, slowly healing ulcers following burns of any kind build up fibrous tissue, the contraction of which leads to stricture. Cicatricial stenosis due to burns by radium or the Roentgen ray usually results from the late chondrial radionecrosis; only rarely from the early reaction.

**Prognosis.**—Obviously the prognosis is influenced by the additional presence of burns other than those of the larynx; but it may be said that the prognosis in cases of burns due to causes other than radium and the Roentgen ray is usually good as to life, if the patient is in a hospital in which obstructive laryngeal dyspnea (*q. v.*) is promptly recognized and promptly treated by tracheotomy. Under routine home care most of the patients die for lack of this procedure. The secondary cicatrices of the larynx may require months of treatment, but the prognosis as to ultimate recovery is good if there is no chondrial necrosis. If the laryngeal carapace is gone it is rare that an ample laryngeal airway can be obtained. The prognosis after radium burns is generally unfavorable and especially so if the burn is severe. Sloughing, necrosis, indefinitely retarded healing, gaping wounds, etc., are not uncommon sequelæ, and in many such cases the outcome is fatal.

**Symptoms.**—If the burn is altogether intrinsic there is seldom pain or much discomfort. Hoarseness, cough, and dyspnea are the usual symptoms. Extrinsic burns are, however, almost always present, and give rise to pain, dysphagia, odynphagia, and drooling.

**Prophylaxis.**—Burns from household lye can be largely prevented if mothers, housewives, teachers, nurses, nursery maids, and scrubwomen are taught that lye is a poison that must never be left in the reach of children. Burns by radium and the Roentgen ray can be avoided if the extreme sensitiveness of the laryngeal cartilages to radiation be realized. Disastrous obliteration of the larynx in children with a benign self-limited disease like multiple papillomata is deplorable. In adults with inoperable cancer anything that offers hope of arresting the life-destroying growth is justifiable. Window resection of the thyroid cartilage may permit of sufficient dosage without perichondritis and radionecrosis.<sup>2</sup> G. E. Pfahler has developed a technic that does not burn the cartilages or even the skin. As pointed out by Hautant it is the midline zone of the thyroid cartilage that is most sensitive to radium and Roentgen-ray burns. Douglas Harmer has made important studies on this subject.

**Treatment.**—The treatment of burns by war gases is masterfully presented by my colleague, Professor Fernand Lemaitre, in another section of this book. The most important consideration in all kinds of burns of the larynx is the possibility of edema. The patient's larynx should be examined with the mirror a number of times daily, and two alternate nurses thoroughly familiar with the cardinal signs of obstructive laryngeal dyspnea should be in attendance. Tracheotomy (*q. v.*) should be done low and early. If in the case of unfortunate haste a high operation is done, a low operation should follow the re-establishment of natural respiration. Local applications to laryngeal burns, if used at all, should be in the form of mild unirritating sprays. Applications on swabs aggravate the trouble. Rest of the larynx is the best remedy. In the early stages of laryngeal burns associated with extrinsic mucosal burns the intense odynphagia, with the increased pouring out of thick salivary secretions, calls for frequent use of the mechanical aspirator. After the aspiration of secretions from his mouth and pharynx the patient should be forced to swallow water at room temperature in large enough quantities to prevent dehydration. Liquid foods may be given also, but if the water is in sufficient quantity a few days without food will be of small consequence. Vaporization of compound tincture of benzoin in the room will palliate the patient's discomfort.

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### CICATRICIAL STENOSIS OF THE LARYNX; TREATMENT

**Preliminary Study of the Case.**—Before any attempt is made to treat a case of laryngeal stenosis, a thorough study of the patient's general as well as local condition should be made. The treatment is prolonged, therefore, as urged by Chevalier Jackson, and the patient should be in the best possible general condition before treatment is started. Active lues or tuberculosis

is a contraindication to local treatment. The presence of fever from any cause is a contraindication to dilatation or operative treatment. Prior to operation all foci of infection in the mouth or upper air-passages should be removed, if possible. Teeth should be put in good condition; diseased tonsils should be removed and sufficient time allowed for healing.

The local condition of the larynx and upper trachea should be thoroughly studied. The great majority of patients with chronic stenosis of the larynx present themselves wearing a tracheotomy tube. The exact position of the tracheotomic fistula with reference to the cricoid cartilage should be determined both by external examination and by direct laryngoscopic inspection of the interior of the larynx. If necessary, a small bronchoscope may be used through the laryngoscope to examine the cords and subglottic



Fig. 438.—Photograph showing the technic of direct laryngoscopic dilatation. No anesthetic is required. The nurse is holding three sizes of dilators. Three sizes are used at each sitting. The largest one used should engage snugly in the stricture. When the largest dilator passes through the larynx without resistance the sizes are stepped up. Stage 1, exposure of the upper orifice of the larynx, is here illustrated.

larynx. If the atresia is complete, retrograde examination through the tracheotomic fistula with a retrograde laryngoscope will be a valuable aid. Jackson in his writings and teachings has very strongly emphasized during the past forty years the fact that faulty tracheotomy is one of the chief causes of chronic laryngeal stenosis. While today high tracheotomy is less frequently performed, largely because of this teaching, the tube is still frequently placed too high in emergency. When the tracheotomic fistula is found to be above the second tracheal ring, the first step in the treatment of such a patient should be a low tracheotomy. In placing the tube low, local anesthesia should be used, and if possible two or three cartilaginous

rings should be preserved between the old and new openings. When the tube is placed at this lower level it is well below the conus elasticus, the larynx is put at rest, and when the tracheotomy is properly performed, the tube may be worn until the stenosis is fully dilated without fear of its presence producing further cicatrices.

**Treatment.**—The treatment of chronic stenosis of the larynx attempts to restore a normal, or at least a functionally normal airway through the stenosed area. This is accomplished by gradual dilatation over a prolonged period during which absorption of the cicatricial tissue occurs. In some cases it is necessary to excise the scar tissue and follow this with gradual dilatation until complete healing with epithelization occurs. The final result will depend upon the integrity of the cartilaginous framework



Fig. 439.—Direct laryngoscopic dilation of cicatricial laryngeal stenosis. Stage 2, inserting the bougie.

of the larynx and upper trachea, particularly the cricoid cartilage. If the cricoid cartilage, the only complete ring in the tracheobronchial tree, has been destroyed by previous inflammatory conditions or high tracheotomy, failure to obtain a satisfactory airway through the larynx will result. In children the normal growth of the cartilage will assist in obtaining an adequate lumen. Bearing this in mind, every effort should be made to avoid injury to the cartilage. The presence of an active perichondritis is a contraindication to all methods of dilatation. The affected parts should be placed as nearly at rest as possible, until the acute inflammation has subsided.

**Methods of dilatation** may be considered under two heads: one, peroral methods; two, operative methods.

*Peroral methods* of dilatation are applicable in all cases in which there is not complete atresia of the airway. They should be given a thorough trial in practically all cases before the more radical external methods are attempted.

(a) *Direct Laryngoscopic Dilatation*.—The larynx is exposed with a Jackson laryngoscope, metal dilators of one-piece construction are passed at weekly intervals. The largest size that is used at each treatment should engage with only slight resistance in the lumen. As the opening through the stenosed area increases, the size of the dilators is stepped up until the laryngeal lumen is brought to normal. This usually requires a period of three months to two years. As the airway opens up a partial cork is placed in the tracheotomy tube so that breathing will be partially through the mouth and will thus exercise the larynx. During the course of treatment great care must be used that overdilatation does not cause perichondritis. If an acute laryngotracheitis should develop at any time, dilatation should be discontinued while it persists, and the cork should be removed from the tube in order to put the larynx at rest. This method, devised and perfected by Chevalier Jackson, is carried out without anesthesia, general or local, and is the most valuable method we have for treatment of stenosis of the larynx. It is particularly valuable in treating laryngeal stenosis in children under five years of age where the after-care of laryngostomy is so difficult that it makes the operation impracticable.



Fig. 440.—Photograph of a child wearing an endless string through the larynx, pharynx, and nose. The string is used to pull through increasing sizes of dilators.

In some cases where there appears to be a complete closure of the lumen of the larynx it may be possible to pass an eyed probe or filiform retrograde through the tracheotomic fistula from below. A string may be attached to the probe or filiform and carried through the larynx. The upper end of the string may then be passed through the nasopharynx from the mouth and out through the anterior nares. If the upper and lower ends then be tied together we have in position a continuous string through the larynx, pharynx, and nose (Fig. 440). To a loop of this string brought from the pharynx through the mouth a dilator can be attached and pulled downward into the stenosed larynx by traction on the string through the tracheotomic fistula. After each dilatation the dilator is removed through the mouth and the new string should be allowed to remain through the mouth and nasopharynx. When the lumen is thus established further treatment may be continued until a cure is obtained by the direct laryngoscopic method of Jackson as described above.

(b) *Intubational Treatment of Chronic Laryngeal Stenosis*.—Various types of apparatus have been devised for intubational treatment. The

intubational tubes patterned after the O'Dwyer apparatus have been successfully used. They are best placed in position by the direct method with a Jackson laryngoscope and the Mosher direct intubation apparatus. A great many ingenious tubes have been devised but the space allotted to this chapter will not permit of their description. By this method of treatment two difficulties are encountered: First, the adaptation of a standard set of intubation tubes to the particular case of laryngeal stenosis so that an improperly fitted tube may not produce a perichondritis; second, the retention of the dilating apparatus in the larynx. This latter difficulty is overcome in the Jackson self-retaining tubes by screwing a post through the tracheotomic fistula. A block placed over the post prevents its becoming unscrewed and maintains the fistula (Fig. 441, Nos. 2 and 3).

The tube may also be held in position by means of a string fastened to its lower end and secured around the tracheotomy tube below. In Iglauer's

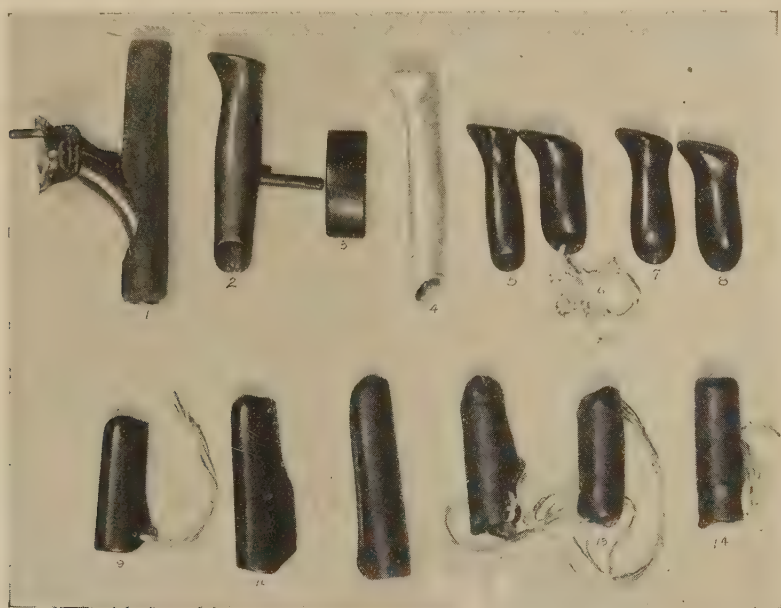


Fig. 441.—Laryngostomy apparatus. 1, Jackson laryngostomy tube fitted with rubber dilating tube. 2, Jackson self-retaining intubation tube showing retaining post and block (3). 4, Jackson intubation tube. 5 and 6, Closed hard-rubber plugs. 7 and 8, Open hard-rubber plugs pulled in from above by means of a string carried up through the larynx from the laryngostomy opening, and worn above a tracheotomic cannula. 9, Closed plug. 10, Open hard-rubber plugs put into larynx from below and worn above tracheotomic cannula. 11, Surgeon's rubber glove finger used for covering rubber tubes used for plugs. 12, 13, and 14, Complete plug with string for attachment to tracheotomy tube.

method dilatation is accomplished by means of a doubled soft-rubber tube pulled into the larynx from above. The tube is held in position by attaching the string to the tracheotomy tube below.

The author has obtained the most satisfactory results from the intubational method by making a plug of folded and inverted cigarette-drain, (Penrose drain) adapting the size and length properly to the individual case. The plug is placed in the larynx by means of the continuous string through the larynx and nasopharynx as described above. It is held in

position by a string attached to its lower end and to the tracheotomy tube below and is suspended from above by a supraglottic swell and the string attached to the upper end and out through the anterior nares. This apparatus is elastic and is placed in the larynx "on the stretch," and has been found very effective in securing a wide and rapid dilatation without undue trauma.

**External Operative Methods of Dilatation.**—(a) *Schmiegelow's Method.*—The method of dilatation as recently described by Professor Schmiegelow of Copenhagen at the 1927 session of the Inter-State Post-Graduate Assembly at Kansas City consists, first, in low tracheotomy, second, of thyrotomy with complete exposure of the interior of the larynx and trachea above the tracheotomy fistula. Scar tissue is then excised and a rubber tube of the proper size and length is selected to fill the laryngeal cavity snugly, extending upward to the level of the arytenoids and downward to the level of the stenosis. The rubber tube is placed in position and is held there by transfixing it with a silver wire which is passed through both thyroid alæ. The thyrotomy wound is closed, leaving the tube in position. The silver wire is then cut off at the level of the skin on each side and the skin is pulled over the end of the wire (Fig. 442). The tracheotomy tube is replaced. The dilating tube is left in place for a period of several months, the silver wire acting as an anchor to the tube. On withdrawing the tube through the mouth, traction on the tube bends the silver wire, allowing it to come out. In some of the reported cases a second thyrotomy and replacement of a tube of larger size was necessary to secure sufficient dilatation. Schmiegelow reports excellent results in his series of cases by this method.

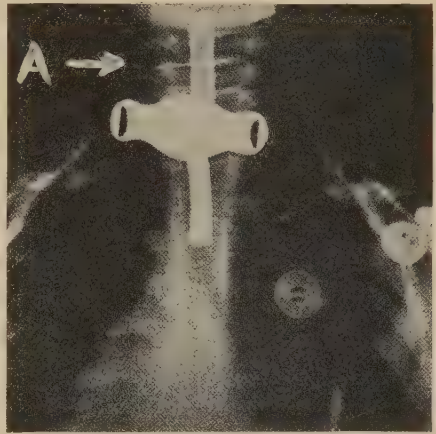


Fig. 442.—Method of Schmiegelow. The silver wire (A) passes through the tube and both wings of the thyroid cartilage.

(b) *Laryngostomy* is defined by Jackson as "the surgical procedure of laying open the larynx and keeping it open for a long period of treatment. More or less of the trachea is included in the opening and the procedure is then a laryngotracheostomy. The purpose of the operation is to get rid of the cicatricial tissue and to cover the newly formed lumen of the larynx, first with small, firm granulations, and then with epidermal epithelium."

**HISTORY.**—The reader is referred for notes on the history of laryngostomy to *Peroral Endoscopy and Laryngeal Surgery*.<sup>2</sup>

**INDICATIONS.**—The operation of laryngostomy is indicated in cases of cicatricial stenosis of the larynx when all other methods of treatment fail. If sufficient cartilaginous framework remains to give support to the airway a cure can be obtained. In cases where sufficient airway through the larynx to enable to closure of the laryngostomy fistula is not obtained, a large fistulous opening in the neck communicating with the mouth through the larynx may enable the patient to develop a good voice, and the laryngostomy opening is safer and more desirable than wearing a tracheotomy tube.

After an epithelized trough is secured following laryngostomy the framework of the larynx and trachea may, where deficient, be built up by the transplantation of cartilage and bone. In this way the narrowed lumen may be increased to such an extent that the fistula may be closed. The author had the privilege of examining a patient of Liebault's in Paris in whom the laryngeal framework had been deficient and a new framework had been built up by Liebault by transplanting bone and cartilage in this way. Moure's method of laryngostomy (Fig. 443) had produced a good lumen through the larynx. The patient had a normal airway and an excellent voice following plastic closure of the fistula.

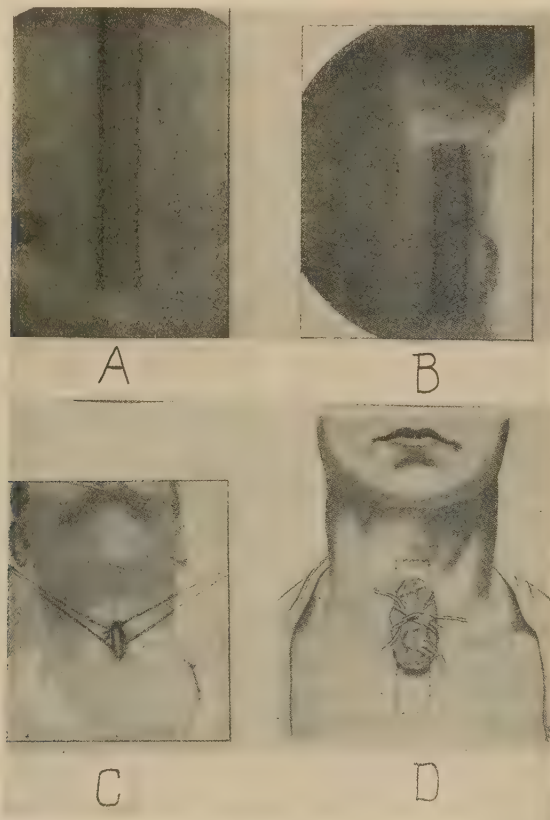


Fig. 443.—Method of Canuyt. A, Radiograph of rubber dilating tube in place, in the patient, as seen from the front. B, Radiograph of the rubber dilating tube as seen laterally. C, Rubber dilating tube in place showing the retaining strings. D, The rubber dilating tube is indicated by the dotted line, the opening is packed with gauze to maintain the opening and to fix the tube when the knots are tied down tightly on it.

**INSTRUMENTS.**—Besides the general operating instruments there should be a blunt-pointed bistoury, Jackson's turbinotome, Jackson's laryngeal-grasping forceps, Jackson's thyrotomy scissors (right and left), small retractors, silk for suturing the mucosa of the skin, and a small electric headlight. An electrical aspirating apparatus of the type used for tonsillectomy, manufactured by Pilling or Sorenson, will be found satisfactory. For the first dressing an iodoform gauze roll, well vaselized, and held in posi-

tion by a hammock of plain gauze is used. Later, special rubber tubing, Penrose drain, finger cots, etc., are necessary for construction of the dilating unit, depending upon the type of dilating apparatus used.

**TECHNIC.**—The technic is essentially that of Jackson (Fig. 444).

**POSITION OF PATIENT.**—The patient is placed in the combined Trendelenburg-Rose position to prevent aspiration of blood and secretion. The wound is not allowed to close during the operation, being kept open with retractors. A sand-pillow is placed under the shoulders.

**ANESTHESIA.**—Local infiltration anesthesia,  $\frac{1}{2}$  per cent. novocaine, is best. In adults the interior of the larynx may be swabbed with 10 per cent. cocaine.

**OPERATION.**—The operation may be described in four steps: First, opening the larynx; second, incision of the posterior wall; third, suture of the mucosa to the skin; fourth, placing of the dilating tube and dressings.

1. Laryngotomy. The simplest method of opening the larynx, requiring but a second or two, is to insert the lower blade of the inverted Jackson turbinotome in the tracheal fistula upward to the level of the thyroid notch, and divide all the tissues, including the skin at one clip. If the

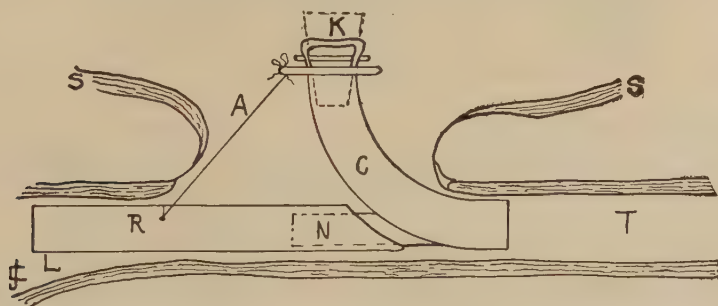


Fig. 444.—Schema showing Chevalier Jackson's method of laryngostomy. The hollow upward metallic branch (N) of the cannula (C) holds the rubber tube (R) back firmly against the spur usually found on the back wall of the trachea. Moreover, the air passing up through the rubber tube (R) permits the patient to talk in a loud whisper, the external orifice of the cannula being occluded most of the time with the cork (K). The rubber tubing, when large sizes are reached may extend down to the lower end of the cannula, the part C coming out through a large hole cut in the tubing at the proper distance from the lower end. (From *Peroral Endoscopy and Laryngeal Surgery* by Chevalier Jackson, Text-book, 1914; also *Endoscopie*, Gaston Doin, Paris; also *Bronchoscopy and Esophagoscopy*, by Chevalier Jackson, Text-book, 2 ed., 1927.)

thyroid cartilage has been divided before, care should be taken that the clip be made through the line of fibrous union so as to prevent any loss of cartilage that might occur if the incision were made elsewhere.

2. Incision of the posterior wall. This is best done with a sharp scalpel, the cut is made vertically, exactly in the median line, entirely through the scar tissue, care being used not to incise the esophageal wall. This will give a trench in which the dilating apparatus will be placed. Web-like cicatrices may be excised.

3. Suture of the mucosa to the skin. If there is sufficient tissue to hold the sutures at the outer edge of the skin and the mucosa, the edges may be joined by three or more deeply placed sutures. Care should be taken not to injure the cartilage. If there is not sufficient tissue to hold the sutures they may be dispensed with.

4. Placing of dilating tubes and dressings. The laryngotomy wound is kept open with retractors, secretions are aspirated from the trachea and larynx and a firm roll of iodoform gauze, large enough to fill the cavity



Fig. 445.—Iodoform gauze roll in sling ready for insertion into laryngostomy trough.

of the larynx, and long enough to extend from the tracheotomy tube upward, to the level of the top of the arytenoids, is prepared by placing it in the hammock of plain gauze, and coating it with vaseline. The ends



Fig. 446.—Ends of gauze sling separated above tracheotomy tube ready for application of central roll of dressing. This is the Chevalier Jackson method of preventing accidental occlusion of the lower trachea as might happen if a tape-like packing were used.

of the hammock-like swing are then brought together, and the roll of gauze is introduced deeply into the widely opened laryngotomy wound (Fig. 445). The ends of the gauze-sling are then separated and a pack of folded gauze is placed directly over the central roll. The tracheotomy tube is placed in contact with the lower end of the pack and adhesive strips are used to hold the dressing in position, and a bandage covers the entire dressing, the ordinary dressing being used around the tracheotomy tube (Fig. 447).

AFTER-CARE is the most important part of the laryngostomy operation. It should be directed by the surgeon himself and carried out by him or an assistant or nurse who is experienced with this particular operation. The tracheotomy tube

should have the routine care that should be given to every tracheotomy case; that is, the inner tube should be cleaned as frequently as is necessary, every ten minutes if needed. The tracheotomy tube and gauze

roll should be changed every twenty-four hours. The roll of gauze that is used in the pack is increased in size until the desired lumen is obtained, providing no undue reaction occurs in the tissues of the larynx.



Fig. 447.—Photograph of the same patient and apparatus as in Fig. 446 with completed dressing.

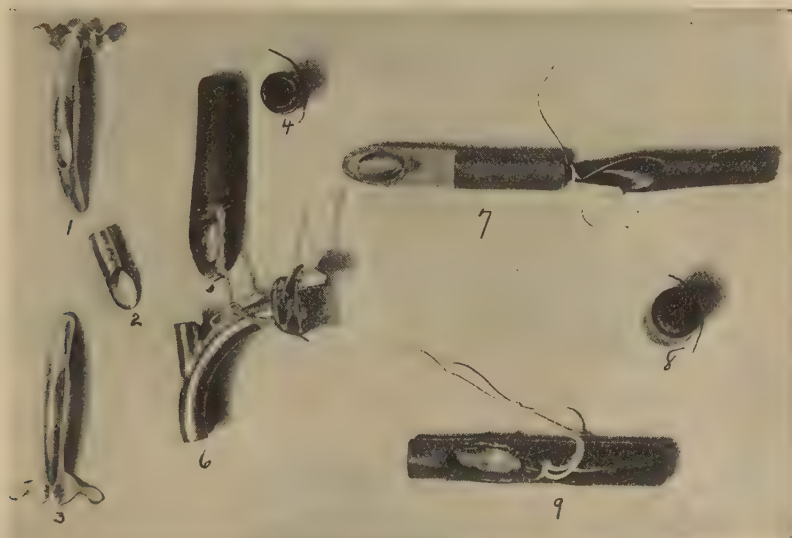


Fig. 448.—Author's laryngostomy apparatus: 1, Open inner cannula. 2, Collar for attaching rubber dilating unit. 3, Closed inner cannula. 4, Open end of (5) cigarette-drain covered rubber dilating tube. 6, Laryngostomy cannula of the author. 7, Extra thickness of cigarette drain cover added to tubing to increase size, and string tied around cigarette drain cover before it is pulled over to close the upper end. 8, Closed end of finger cot tube (9). Closed cannula (3) is used for closed tube (9). Open cannula (1) used for open tube (5).

Sloughing is to be avoided. After the interior of the larynx is smooth and filled with firm granulations the rubber tube dilator is then used. The laryngostomy tube of Jackson (Fig. 442) or the modified tube with the rub-

ber dilating unit may be selected (Fig. 448). We have found in the use of rubber tubing for dilating that there is frequently a reaction in the tissues from even the best grade of rubber tubing that can be secured. By covering the rubber tubing after it is properly fitted to the larynx with a finger-cot or Penrose drain, there is much less reaction in the tissues of the larynx and the dilating is much more rapid. The dilating unit of the laryngostomy apparatus is increased in size as the local condition of the larynx warrants by an extra thickness of finger-cot or Penrose drain until the desire increase in lumen is secured. The dilating apparatus and dressings are changed daily, the larynx being watched very carefully that there is no point of undue pressure.



Fig. 449. Having an epithelialized trough this child is ready for a test period.

In cases where the obstruction is found to be very high in the larynx without such tendency to spur formation on the posterior tracheal wall which as usually occurs in long-standing tracheotomy cases, satisfactory dilatation can be secured by the use of a gauze plug covered with rubber tissue or a plug made of Penrose drain covered with a finger-cot used as the dilating unit. This apparatus can be best held in position by suturing a folded strip of gauze on to the end of the anterior surface of the plug. The fastening of the plug in the middle of the strip of gauze allows the end of the strip of gauze to be carried across the edges of the wound on either side and keeps the wound open. The outer dressing is held to this gauze strip with adhesive and the bandage is applied over the entire dressing. The tracheotomy tube being placed below, helps to maintain the position. This type dressing makes the dilating unit a portion of the external dressing,



Fig. 450.—Roentgenogram of laryngostomy apparatus of Jackson in position, in boy, aged nine years.

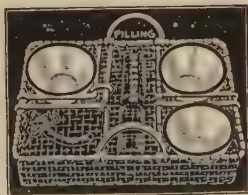


Fig. 451. —Where there are many cases of laryngeal stenosis under treatment change of dressings required for proper care will be greatly simplified by having a wire compartment tray such as that here illustrated, for the sterilization and care of the needed apparatus and utensils.

and if in emergency the external dressing should be removed, the plug comes out of the larynx along with the dressing, thus preventing the possi-

bility of its slipping downward into the trachea. When a completely epithelialized trough is obtained by means of the dilating apparatus, and the lumen is thought to be adequate, the dilating apparatus is then removed for a test period (Fig. 449). If the wound edges approximate closely, a completely corked tracheotomy tube is worn for emergency, and the patient breathes through the mouth. If the sides of the trough stand well open, adhesive is used to close the front of the neck so that the patient will breathe through the mouth. A test period of several months is usually allowed before plastic closure of the laryngostomy fistula is done. The plastic closure method of Nassau has given excellent results in a number of cases at the Bronchoscopic Clinic.

GABRIEL TUCKER.

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### CLOSURE OF LARYNGOSTOMIC FISTULÆ

**Problems Presented by Laryngotracheal Fistulæ.**—Operation for the closure of fistulæ of the larynx and trachea is indicated when the laryngologist has completed his treatment. Patients are referred back to the general surgeon for the performance of plastic operations only after the laryngologist has succeeded in creating a wide open airway.

This problem presents itself more often to the laryngologist than to the general surgeon. Occasionally, of course, the general practitioner will have in his care a patient who has had a tracheotomy performed, and who has found the wearing of the tube to be indispensable. At times the general surgeon may contribute to the number of these patients by injuring the recurrent laryngeal nerve. It is of academic interest only whether the obstruction which preceded the laryngostomy was caused by scar contraction, bilateral recurrent nerve paralysis, gunshot injury, or stab wound; closure of the fistula, independent of the cause, is carried out in the same way.

Heretofore, the obliteration of these fistulæ (Fig. 452) has been a very uncertain surgical procedure, and in about 50 per cent. of cases the closure has not been permanent.

A number of points must be given careful consideration if the end-result is to be satisfactory. The theoretically perfect operation demands that no narrowing of the air-passage be produced, that the new lining shall consist of skin, that the closure shall be made up of more than a

single layer, that an adequate blood-supply shall be present to guarantee the vitality of the reconstructed part, and that absolutely no tension on the sutures shall exist after the operation has been finished.

Encircling the fistulous area and inversion of the tract were not considered, as this procedure would narrow the air-passage and the tissues utilized would have a poor blood-supply because they would consist chiefly of scar. To turn back a flap from one side across the fistula would necessitate sutures on three sides of the flap, and the raw area left by the turning over of the flap from the side is not so easily covered as one taken from the median line. Moreover, this method might leave a line of raw surface on the newly formed anterior wall of the larynx and trachea.

I planned, therefore, to bring the flap from below the opening, with its base attached to the lower edge of the fistula. By doing this, skin and fat



Fig. 452.—Laryngostomic fistula.

which have a fair circulation can be secured, and the narrowness of the flap obviates any difficulty in bringing the superficial wound together in the median line.

**Indications.**—When the patient is able to breathe with perfect comfort under all circumstances, day and night, after the fistulous opening has been experimentally occluded by adhesive plaster, the fistula is ready to be closed.

**Contraindications.**—Operation upon patients who are suffering with the slightest degree of bronchitis or coryza must be postponed.

**Preparation of Patient.**—No special preoperative preparation is necessary. If the patient is at all nervous, a hypodermic injection of morphine sulphate,  $\frac{1}{8}$  to  $\frac{1}{4}$  grain, together with  $\frac{1}{200}$  grain of scopolamine may be given one and one-half hours before operation. As a rule, however, this is not necessary and the patients in this series had no preoperative opiate.

Any hair existing on the surface of the flap should be destroyed by Roentgen ray previous to operation.

Great care must be used in disinfecting the skin, whether it be with alcohol or iodine, as the dropping of any irritating solution into the trachea produces violent paroxysms of coughing.

**Anesthesia.**—Consideration must be given to the method of anesthesia which is of vital importance in these cases. A general anesthetic is difficult to administer; furthermore, the respiratory excursions of the larynx during general anesthesia would be embarrassing while introducing the necessarily large number of fine sutures. Coughing, vomiting, and the secretion of mucus in the trachea are other disadvantages.

Under local anesthesia there is no limitation of the amount of time that may be consumed in operation, and the patient is breathing normally throughout. The seven patients in this series were operated upon under local anesthesia and only one patient complained of pain. The youngest

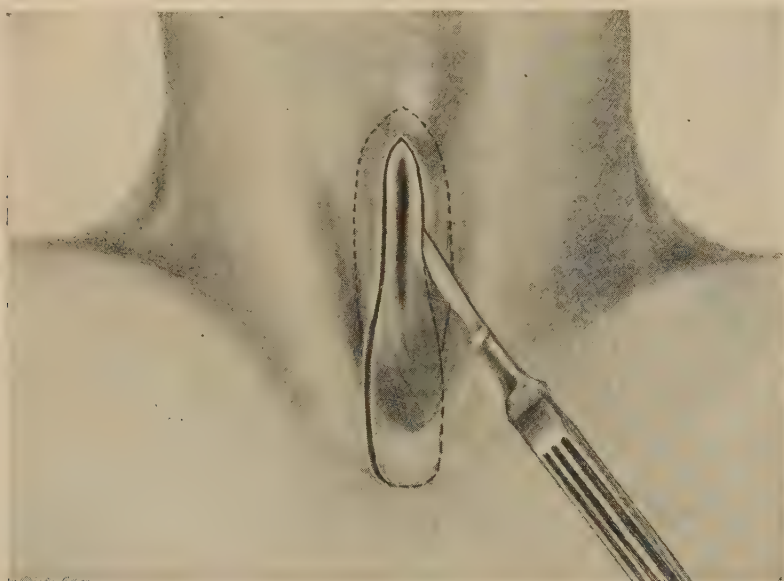


Fig. 453.—Outlining of flap. Dotted lines indicate the area to be undercut.

patient in the series, aged eleven years, gave just as full co-operation as the adults.

Field-block is the method of choice, and novocaine in  $\frac{1}{2}$  per cent. solution, with the addition of 6 minims of adrenaline chloride to each ounce of solution, is the preferred anesthetic. Infiltration of the flap itself is not necessary and should be avoided on account of possible interference with healing.

**Technic.**—At the request of Dr. Chevalier Jackson I devised an operation for the closure of laryngotracheal fistulæ which has given excellent results in the cases presented in this series.

The method employed consists in raising a skin flap with its base attached to the lower end of the laryngostomic fissure (Fig. 453). The width of this flap is outlined in such a manner that the upward prolongation of the incision around the opening is not less than 6 mm. from the edge of the opening. This is a very important point, because if too narrow a flap be cut and the

incised edge, which is afterward undercut laterally as shown by the dotted lines on Fig. 453, is made too close to the edge of the opening, the mucous membrane which lines the air-passage will have retracted to such an extent that introduction of the sutures will be a difficult matter.

The length of the flap downward passes beyond the insertion of the tendinous portion of the sternocleidomastoid muscle and at times extends over the manubrium. The width of the flap should be almost the same at the top as at the base, and the flap should not be made pointed. Measure the length of the fissure and add one-third to this measurement to allow for shrinkage. The width of the flap should be  $2\frac{1}{2}$  cm. as a rule, although it may be as narrow as 2 cm. Skin and underlying fat are lifted in one piece and the lower end of the flap later corresponds to the upper end of the laryngostomic opening. Ascertain that the flap is sufficiently long to lie perfectly loose over the opening without tension. Undercut the area

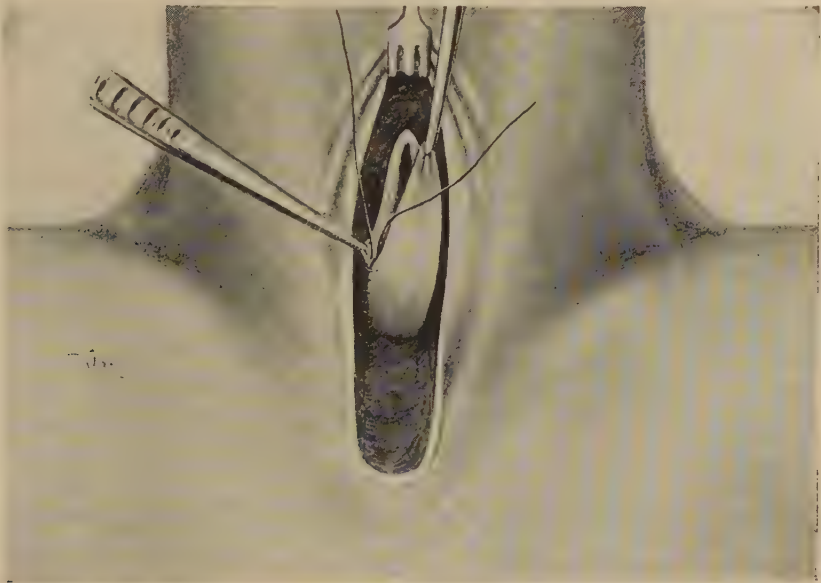


Fig. 454.—Flap turned up and method of suturing.

laterally along the sides of the opening and above it so that the remaining skin of the neck can be brought together in the median line over the flap. The upturned flap is then sutured to the edge of the lining of the opening in such a way that the sutures are invisible from the outside when they are tied, and the knots come to lie on the inside of the air-passage (Fig. 454).

This method of tying the knots is analogous to the end-to-end suture of the small bowel by the Connell method of using interrupted mattress-sutures, or to the repair of a complete tear of the perineum where the sutures are tied on the inside of the bowel.

The first suture is introduced 3 mm. from the point of the folding of the flap at its base against the edge of the lining of the area. This suture and its fellow of the opposite side and the last three deep sutures used in the operation are the most difficult ones to insert and tie. Introduce the needle slightly less than 3 mm. from the edge of the skin-flap at the point

where it is folded up over the opening, from within outward and then reintroduce it from without inward; this brings the knot on the inside.

After inserting and tying two or three sutures on one side, the next two or three sutures should be placed on the opposite side of the flap, proceeding from the base to the tip of the flap. This method will keep the flap straightened out in the position in which it will ultimately lie. In inserting and tying these sutures care must be used to bring the skin edge of the flap into perfect epithelial contact with the edge of the opening. The space between sutures should be less than 3 mm.

As the suture lines approach completion a very little opening is left at the upper end, and sharp scissors should be used to cut off the slightly redundant tip of the flap. This does away with too long a flap and reduces the possibility of gangrene from lack of circulation and, even if it were not



Fig. 455.—Flap completely sutured and superficial wound ready for closure.

necessary for that reason, is a wise procedure because it obviates the utilization of tissue that has been held by forceps and possibly devitalized.

The final problem is that of closing a small tube. The last three or four sutures are left untied until all the sutures are introduced. One interrupted suture running from the fat on the top of the flap to the tissues up and beyond the last point of suture will give added security. Interrupted silkworm-gut sutures close the skin wound (Fig. 455). Be careful not to make too perfect a closure because there may be some infection of the wound and easy exit for secretion should be provided in advance. The space between the sternocleidomastoid tendons is unyielding and the obliteration of a dead space difficult, and a small drain should be introduced at this point.

Ordinary black spool silk, size A, is used for the deep sutures. Catgut should never be used, because the large number of sutures of catgut would

make a bulky mass of foreign matter which might prove to be decidedly detrimental. The best needle is a milliner's straw needle, No. 9, sharps; it may be necessary to use a very fine curved needle if the skin has been cut too close to the opening, on account of the retraction of tissue.

The principle upon which the success of this operation depends is the security with which the flap is held by the non-absorbable sutures. The ultimate fate of these sutures need cause no anxiety; they are cast off into the trachea and larynx during the course of healing.

Successful closure depends also upon anticipation of infection. Operation is carried out under extreme difficulties in the observance of thorough antisepsis. Three of our patients developed some wound infection. On the appearance of inflammation hot boric-acid compresses are used over a period of twenty-four to forty-eight hours. This treatment saves these wounds from a complete breakdown in the same way that it saves abdominal wounds that are on the verge of infection, as recommended by Watkins of Chicago.

Even if the wound should be very red or should be discharging between the sutures, the skin sutures should never under any circumstances be removed.

The trifling infection in our patients had no bearing on the final result. Six of the patients in the series were referred to me by Dr. Chevalier Jackson. Five of these patients made complete and permanent recoveries. A second tracheotomy was performed by Dr. Louis H. Clerf on the sixth patient on account of scar contraction within the larynx. This contraction occurred six months after the obliteration of the fissure and was not the result of the closure of the laryngostomic fistula. The seventh patient, a lad of eleven, who was referred to me from the service of Dr. Fielding O. Lewis, had primary healing and all skin sutures had been removed when, on the eighth day after operation, after having been up and around the ward and apparently in perfect health, he died in a few moments of an edema of the lungs, probably attributable to cardiac disease.

CHARLES F. NASSAU.

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## SYPHILIS OF THE LARYNX

THE frequency with which the larynx is affected in syphilis is somewhat difficult to estimate. Statistics on this point are subject to variation. As regards the comparative incidence of syphilis, tuberculosis, and malignant disease in the larynx, the following figures have been obtained from the statistical tables of the Ear and Throat Department of the Royal Infirmary, Edinburgh, during the period 1907-1922, inclusive. Of 2396 affections of the larynx, syphilis was diagnosed in 144 cases, 6 per cent.; tuberculosis in 389, 16 per cent.; and malignant disease in 136 cases, 5 per cent. Since the introduction of the Wassermann test in 1902, it has been possible to establish a more accurate diagnosis of syphilitic affections.

**Pathology.**—Syphilis affects the larynx both in the inherited and in the acquired form.

In the *inherited* or *congenital type*, it may manifest itself shortly after birth as a laryngeal catarrh. In late hereditary syphilis, on the other hand, the lesion may assume the form of a hyperplasia. As the incidence may be delayed until young adult life is reached—and the same is true of the pharynx—there is a danger of mistaking the condition for a manifestation of the acquired form.

Brown Kelly has classified the varieties of late hereditary syphilis in the larynx as follows:

1. Hyperplasia associated with ulceration; frequently seen as thickening at the periphery of ulcers or scars.
2. Hypertrophic granulations and papillary excrescences, which may or may not be preceded or followed by ulceration; the condition is most often seen on the epiglottis and resembles lupus.
3. Tumor-like hyperplasia.
4. Diffuse hyperplastic infiltration.

In the *acquired type* of syphilis of the larynx, the primary sore is rare. The secondary and tertiary manifestations present no definite reference to the period of time which elapses after the appearance of the primary inoculation. Thus, the secondary phenomena may appear and reappear for a number of years after the initial infection; the tertiary forms may be met with within a few months of the primary lesion, or after an interval of many years. Even the co-existence of a secondary and tertiary lesion is not unknown. A knowledge of these facts has a bearing upon the question of treatment.

The primary sore, when met with, is usually found on the epiglottis. The secondary manifestation is of the nature of a hyperemia of the mucous membrane, occasionally associated with a localized hyperplasia of the epithelium resulting in the formation of the mucous patch; superficial ulceration supervenes. The mucous patches occur on the vocal cords and on the lingual aspect of the epiglottis.

The tertiary lesions take the form of infiltrations due to small-cell proliferation. The infiltration may be localized or circumscribed, producing the characteristic gumma which is found on the epiglottis, the aryepiglottic fold, the ventricular band, the posterior wall of the larynx or in the subglottic area. On the other hand, the infiltration has a more diffuse character, causing a more generalized tumefaction and involving the vocal cords or those parts already named.

Ulceration is a subsequent stage of infiltration, whether circumscribed or diffuse and, if the process continues, the destruction passes more deeply and may involve the perichondrium, causing perichondritis and subsequent necrosis and exfoliation of cartilage. In the more diffuse form of infiltration, in contradistinction to the gumma, an organized connective-tissue process may take place instead of the ulcerative changes. In consequence of the destructive and fibroid processes, if the stage of healing is reached, cicatrization, membranous adhesions, stenosis, and deformity of the larynx are found as sequelæ. As a result of ankylosis of the crico-arytenoid articulation, or from the contraction of the soft tissues round the joint, one or the other vocal cord will become fixed.

Papillary excrescences or neoplasms are sometimes found as rarer mani-

festations of the disease. When present, they are most commonly situated in the posterior commissure.

True paralysis of the vocal cord may be due to peripheral neuritis of syphilitic origin and unassociated with any obvious pathological change in the larynx. Nor should it be forgotten that vocal cord paralysis may occur in the subjects of syphilis, consequent upon the existence of a syphilitic lesion in the medulla or from implication of the motor fibers of the vagus at the base of the skull from a pachymeningitis or a periosteal gumma.

**Symptoms.**—In the secondary catarrhal manifestation, hoarseness is the chief or only symptom complained of. The voice may have a peculiar raucous tone; this, however, may be more pronounced in the tertiary stage when the vocal cords are affected. It offers a contrast to the softer character of the voice observed in tuberculous lesions of the cords. Pain is not usually complained of, and cough, as a rule, is absent.

The symptoms in tertiary syphilis will obviously depend upon the nature and situation of the lesion. Cough, it may again be stated, is rarely troublesome. Pain and dysphagia are associated with ulceration of a gumma affecting the free edge or lingual surface of the epiglottis, the aryepiglottic fold, or the posterior surface of the cricoid lamina. Dyspnea will vary both in degree and in the rapidity of onset. Its occurrence may be expected when the infiltration is subglottic, when the free movements of the vocal cords are interfered with, or in the event of the development of perichondritis and abscess. There may be a sudden and dangerous exacerbation of dyspnea associated with the onset of acute edema. Subsequent cicatrization and deformity may lead to a permanent interference in the natural air-way.

Fetid expectoration may characterize the later stages of tertiary syphilis.

**Diagnosis.**—As the above symptoms are referable to various affections of the larynx, a correct diagnosis cannot be made without laryngeal examination. Even then it is not always possible to arrive at a certain conclusion without seeking for confirmatory evidence in other parts of the body and by a more extended examination. It must be borne in mind, also, that the diagnosis may be further complicated by the co-existence of tuberculosis in the larynx, and a patient with syphilis may be predisposed to the development of malignant disease.

*Syphilitic catarrh* of the larynx may resemble in appearance a simple catarrhal laryngitis. Its resistance to the ordinary therapeutic measures recommended in catarrhal laryngitis should raise the suspicion of its possible syphilitic nature. The same statement, however, is true in regard to the laryngeal catarrh which may be met with in a case of pulmonary tuberculosis. On the other hand the laryngeal mucous membrane in the syphilitic case may present a dusky discoloration and, if mucous patches have developed, the mucosa assumes a certain mottled appearance.

It may be necessary, however, in some cases, before the diagnosis can be arrived at, to depend upon collateral evidence. The primary sore may still be in existence; but a careful examination of the buccal and faucial mucous membrane for the detection of mucous patches, and an investigation of the skin for evidence of roseola or a papular eruption, will probably lead to the correct diagnosis. If further confirmation is still deemed necessary, the blood Wassermann reaction should be tested.

In the diagnosis of *tertiary lesions* the laryngeal picture may in one case prove sufficiently convincing of the syphilitic nature of the condition, in

another case it may fail to provide the information necessary for a correct diagnosis. Certain local appearances are strongly suggestive of syphilis. The disease is more prone to attack the anterior part of the larynx; the affected mucosa presents a hyperemic, congested appearance. The infiltration produced by the gumma, which is usually single, is fairly rapid in its evolution and when it breaks down, the edges of the ulcer are defined, punched out, and undermined. Its surface is covered with a yellowish-gray slough, and destruction extends to the deeper parts rather than along the surface.

Tuberculous infiltrations frequently involve the posterior region of the larynx; they develop more slowly; the mucosa is anemic and pale in color; more than one area of ulceration may be present; the ulcers are shallow, with inactive, irregular, mouse-nibbled edges. The lesions of lupus may in some cases simulate the syphilitic condition, superficial ulceration in one accompanying cicatrization in another.

Malignant infiltration may take the form of a definite tumor, which is slowly progressive and frequently presents an irregular appearance on the surface. Its infiltrative character tends to affect the mobility of that part of the larynx to which it is attached. When ulceration occurs, the ulcer has a somewhat excavated surface surrounded by slightly prominent everted edges.

To the experienced as well as to the inexperienced observer, a correct diagnosis may be impossible merely from laryngoscopic appearances. Other factors must be carefully considered and their proper significance duly weighed. Syphilis of the larynx is more common in males, but the same is true of malignant disease. The age of the patient is a factor on which too much importance should not be placed. It is well to remember that no individual is too old to develop tuberculosis of the larynx, or too young to be the subject of malignant disease. Tertiary syphilis may manifest itself in advanced life.

The history of infection may be readily obtained, but, on the other hand, from ignorance or from a desire on the part of the patient to conceal the fact, a negative statement may be of little value. Confirmatory evidence of syphilis derived from a careful general examination of the patient is of great importance. The laryngologist may obtain highly suggestive evidence in the nasal cavities in the form of an old destructive lesion of the nasal septum, or the palate, fauces, or pharynx may reveal evidence of scarring or of actual loss of tissue. In the absence of such, a very careful general examination of the surface of the body may disclose old pigmentation, scarring, or periosteal thickening. Glands in the neck are very rarely enlarged in syphilitic disease of the larynx; they are a frequent concomitant of extrinsic laryngeal cancer; they are only occasionally found in a tuberculous laryngeal affection.

The Wassermann reaction, when positive, is of undoubted assistance, but in tertiary lesions a negative result may be obtained and the test, in these circumstances, is not a criterion of the absence of syphilis. The exhibition of iodide of potassium may be a useful diagnostic measure. The subjective feeling of improvement which may follow its administration must not be relied upon. Actual improvement as observed in the laryngeal mirror is the only true guide. In syphilis this improvement is progressive, whereas in malignant disease it will prove to be of a very temporary character.

While the above measures should be given their due place by the physician, it may still be necessary for him to extend his investigation in other directions. The examination of the chest and sputum, a register of the evening temperature, and information as to loss of weight will assist him in elucidating the possible tuberculous nature of the laryngeal lesion. The employment of the microscope in suitable cases may furnish evidence of the malignant character of the disease.

**Prognosis.**—When the disease is seen in its early stages the prognosis, as a rule, is good, provided that immediate and energetic treatment is carried out and continued during a considerable period of time. In some cases the disease makes rapid progress and the patient's life may be endangered. The possible development of sudden edema of the larynx during the stage of ulceration is a complication which may make the prognosis more grave. Further, it is wise to express a somewhat guarded opinion as to the effect which laryngeal syphilis may produce upon the future character of the voice. Should cicatrization and stenosis follow a more advanced stage of the disease, the patient may be inconvenienced by a permanent degree of hoarseness or dyspnea on exertion.

**Treatment.**—Once the diagnosis has been made, treatment should not be delayed. The general health of the patient must be attended to and a nourishing but light diet, an open-air life, freedom from work and worry, and tonics should have their place in the daily régime. The teeth should receive attention, and tobacco and spirits should be forbidden; vocal rest must be insisted upon.

For the employment of salvarsan and the other more recent arsenical preparations now in use, information must be sought elsewhere. In syphilitic lesions of the upper air-passages, the rule that mercury should be prescribed in the secondary and the iodides in the tertiary stage requires some modification. Mercury should be given in all stages, and a combination of mercury and potassium iodide will often produce a rapid disappearance of the lesion in cases in which the administration of one or other drug alone has failed to give the desired effect. If there is a tendency to dyspnea, care is necessary in the administration of iodide of potassium, because of its tendency to increase the development of edema. Mercury may be administered by the mouth, by inunction, or by intramuscular injection. Potassium iodide should be given in increasing doses; it should be well diluted with water and taken after meals.

Local treatment will depend upon the nature of the symptom complained of or of the lesion requiring attention. In cases of ulceration, an alkaline spray or one containing peroxide of hydrogen may keep the surface clean. Dysphagia may be relieved by the insufflation of orthoform powder, while nitrate of silver or argyrol solution may be applied to exuberant granulations. The sucking of ice, a spray of adrenaline, or scarification may assist in the reduction of edema, but the necessity of a low tracheotomy in increased stridor or stenosis should be kept in view. The opening of the trachea has the further advantage in that it gives greater rest to the larynx.

The treatment of sequelæ will be considered in the section on Laryngeal Stenosis.

A. LOGAN TURNER.

## LARYNGEAL TUBERCULOSIS

The term "laryngeal tuberculosis" signifies a tuberculous infection of the laryngeal mucosa which, in the vast majority of cases, is secondary to pulmonary tuberculosis and is due directly to an inoculation of the mucous membranes by the bacilli in the lung secretions.

**Etiology.**—Although a true primary tuberculous involvement of the larynx is theoretically possible it is extremely uncommon. A few cases have been reported in which a detailed postmortem examination failed to show any pulmonary lesion, and it is not very uncommon to see cases in which the laryngeal disease so predominates the clinical picture that the lesser pulmonary lesion might reasonably be supposed to have followed the primary involvement of the larynx. Yet we must remember the difficulty of detecting small, and maybe healed foci in the lung or other parts of the body that might have started the laryngeal activity, and one should, therefore, hesitate to assert that such and such a case is a primary laryngeal condition, even though it is hard to conceive how a laryngeal tuberculosis can be started in a person with bacillus free sputum. In these cases, in which the lung condition seems to have followed the laryngeal or in which no lung lesion could be clinically demonstrated, there is a possibility that the primary lesion existed higher up, perhaps in the tonsils, either faucial or pharyngeal, or as has been reported by Lake, in the middle ear. From the tonsil the disease might extend along the mucous membrane to the epiglottis and thence to the rest of the larynx, or bacilli escaping from a tonsillar ulcer might gain entrance into the interior of the larynx. In 2 cases reported by Lake there existed a primary tuberculosis of the middle ear and the discharge pouring from the eustachian tube apparently infected the larynx. In each of these cases the lungs developed signs of phthisis considerably later. However, even taking into consideration all the possible sources of tuberculous laryngeal infection, excepting pulmonary disease, the number is so insignificant that the negative findings of a chest will always cast a doubt on even an extremely characteristic laryngeal lesion.

Therefore, for practical purposes, we should consider laryngeal tuberculosis a complication of a primary pulmonary disease. It is, in fact, one of the most frequent secondary involvements. The frequency of the laryngeal complications varies greatly with the stage of the pulmonary disease. From a study of the reports of a number of different observers it was found that the early stages of lung tuberculosis will show some laryngeal lesion in about 12 per cent.; in the moderately advanced cases in perhaps 26 per cent.; and in the terminal stages of the disease 45 per cent., or even more, will show a laryngeal involvement.

The method of invasion of the tubercle bacilli into the laryngeal structures is, in a great majority of cases, through the surface of the laryngeal mucosa from the secretions coughed up from the lung. Certain authorities place importance upon the possibility of a lymphatic route, but if this ever takes place it is an extremely rare happening, as the laryngeal lymphatics are well isolated from the surrounding structures. It is just within the limits of possibility that in a cervical tuberculous adenopathy there might be a retrograde lymph current which would carry the bacilli up the efferent lymphatics of the larynx.

Hematogenous infection of the larynx can theoretically occur in cases of miliary tuberculosis, but I doubt that a blood infection of the larynx ever takes place in other types of this disease.

The possibility of surface infection from the pulmonary secretions is so plausible that it seems scarcely worth while to look for any other method of invasion. Especially is this true in advanced cases of pulmonary phthisis where large quantities of bacilli-laden mucous stagnates in the larynx for long intervals of time. If the larynx is examined in almost any case, or at any time, where the expectoration is free, a greater or less amount of the purulent secretion from the lung can be seen usually on the posterior commissure and also frequently on the cords and other portions of the larynx.

There are four possible ways by which the tubercle bacilli may gain access to the subepithelial tissues from the surface:

1. Through the unbroken epithelium.
2. Through the ducts of the racemose glands.
3. Through traumatic abrasions.
4. Through pathological epithelium.

1. *Through Unbroken Epithelium.*—It is reasonable to suppose that such takes place with more or less frequency, especially in the region of the subepithelial accumulations of lymphoid tissue. It has been positively demonstrated that the tubercle bacillus passes through the normal epithelium of the tonsillar crypt, and also that it will pass through the intestinal wall without causing any microscopic change. Where we find subepithelial accumulations of adenoid tissue, we are apt to find changes in the basal cells of the epithelium as has been demonstrated in the lateral folds of the pharynx and in the solitary lymph follicles on the posterior pharyngeal wall. At the "cushion" of the epiglottis and in the ventricles and other places in the laryngeal mucosa, such lymphoid accumulations are frequently found and hence we have an opportunity for the penetration of tubercle bacillus similar to that found in the disintegrated epithelium of the crypts of the tonsils.

2. *Through the Ducts of the Racemose Glands.*—These glands occur notably in the ventricles and subglottic regions, and though we have no positive evidence that they act as a gateway of infection, it is entirely within the scope of probability that such may at times take place. Some authors considered it a common route.

3. *Through Traumatic Abrasions.*—Just how far trauma plays a part in the introduction of tubercle bacilli beneath the epithelium of the larynx is uncertain. It is entirely possible that the results of severe coughing may be sufficiently severe to produce lesions, especially of the vocal cords, and by the same force the tubercle bacilli may be driven into the depth of the abrasion. Actual trauma, however, of the vocal cords or other portions of the larynx from coughing is not a common occurrence, though it is seen in such conditions as whooping-cough and the explosive cough of acute tracheitis, where we sometimes find actual hematoma of the vocal cords. It does not seem probable, however, that this method of entrance is a common one.

4. *Through Pathological Epithelium.*—There is little doubt but that the pyogenic bacteria can produce a condition of the laryngeal mucous membrane which makes it as possible for tubercle bacilli to gain entrance as it

is for tuberculosis to provide the gateway for secondary pyogenic infection. We frequently see in acute infections, superficial ulcers, especially of the epithelium covering the vocal cords, and it is probable that the continued action of the sputa from the lungs stagnating, especially in the posterior commissure of the larynx, may lead to various types of erosion and fissuring which open up the deeper structures to the tubercle bacilli. As shown by Lake, it is not uncommon to find small histologic abscesses in the epithelium filled with cocci. The rupture of these epithelial abscesses may easily make a port of entrance for the tubercle bacillus.

The three important factors in surface implantation of tubercle bacillus into the laryngeal mucosa are: First, stagnation of the pulmonary secretions; second, pathogenic changes in the mucous membrane due to pyogenic organisms; and third, the mechanical force of severe coughing.

**Pathology.**—Tuberculosis of the larynx is, of course, similar in its pathology to tuberculosis in other organs, being modified, however, as the result of the peculiar anatomy of the larynx and its functional activities.

Histologically the tuberculosis is characterized by the formation of tubercles surrounded by a zone of inflammatory reaction, which zone has different anatomical characteristics according to the organ affected. The first action of the tubercle bacillus is to stimulate or irritate the fixed connective-tissue elements with the production of the so-called "epithelioid cells." These cells, which form the greater part of the tubercle, are surrounded by a zone of small round mononuclear cells which are probably derived from the leukocytes of the blood. The tubercle thus formed is avascular and as it grows in size the center undergoes a coagulation necrosis due as much to the avascular condition as to the specific action of the tubercle toxin. During the early stages of this necrosis a large irregular cell, called the giant-cell, is formed.

Although all tuberculous lesions in the larynx have the same fundamental histology, in their gross appearance they may vary considerably, due to the stage of their development, to the part of the larynx involved, and to the absence or presence of secondary infections, and also to the chronicity of the particular tuberculous process.

For the purposes of differentiation we speak of infiltration, ulceration, edema, tumor formation, and perichondritis.

Infiltration is the earliest stage of laryngeal tuberculosis. The epithelium covering these areas is usually thickened and in the subepithelial tissue one sees round-cell infiltration and typical tubercles and often more or less edema. The tubercles are found immediately under the epithelium becoming fewer in number the closer to the perichondrium one goes. In fact the subepithelial tissue may at times be practically obliterated with tubercles while there are none near the perichondrium. Sometimes the round cells so predominate that the picture is one of a simple inflammatory infiltration except that typical giant-cells may be seen scattered through it.

According to Manassa, the edema which is so frequently associated with laryngeal tuberculosis may show histologically, either large spaces lined with endothelial cells which are probably dilated lymph spaces or another type in which the bundles of connective tissue have been separated by the fluid. Tubercles are usually not found in the immediate neighbor-

hood of edematous tissue. Besides these two types, a histologic edema is often found on the edges of the tuberculous infiltration. This may be either interstitial or less frequently a true parenchymatous edema. In this latter the connective tissue cells themselves are swollen.

It was formerly supposed that tuberculous ulceration was always due to caseous degeneration of one or more tubercles immediately beneath the epithelium breaking out on the surface, but by a very careful microscopic study Manassa has demonstrated the presence of small ulcers without caseation. The epithelium is apparently destroyed either by pressure or by infiltration from the tuberculous tissue immediately beneath it. This infiltration of the epithelium with cells from the underlying tissue may stimulate the basal cells of the epithelium so that sprouts of epithelium can be seen penetrating into the granulation tissue itself. As the disease advances the connective-tissue elements usually destroy the epithelial cells. When the surface epithelium is of the cylindrical type there is a similar destruction of the epithelium by the underlying tuberculous tissue, but there is no regenerative process such as is seen when the squamous epithelium is attacked unless the cylindrical epithelium has undergone a metaplasia with a change of type to squamous epithelium.

When perichondritis is present there is usually a complete destruction of the superficial structures by ulceration, though occasionally the perichondrium becomes involved while the overlying tissue is still intact. Destruction of the cartilage takes place as the result of disease of the perichondrium which produces either extensive necrosis or the destruction of small isolated portions of the cartilage, but true tuberculous disease of the cartilage itself is exceedingly uncommon, if it ever occurs.

Tuberculoma occurs in two forms: One, the fibrotuberculoma with a preponderating connective-tissue element and the other, the granulotuberculoma in which the tubercles are surrounded by granulation tissue. These tuberculomas may become so extensive that the condition is sometimes confused with malignant neoplasm. While pachydermia may occur in simple chronic laryngitis this type of tumor formation is much greater in tuberculous. This papillomatous hypertrophy of the squamous-epithelium cells may reach an almost unbelievable extent. Histologically the condition resembles papilloma and the tuberculous disease is found only in the underlying connective tissue. Concerning the cartilage, Manassa has shown another very important fact, that cartilaginous tissue may be gradually absorbed and replaced by connective tissue; the connective-tissue elements penetrating the cartilage and replacing the cartilaginous cells without any apparent preliminary necrosis of the cartilage.

Concerning the spread of the disease to contiguous portions of the larynx Manassa believes that this usually occurs by way of the lymph vessels and has described a condition which he calls tuberculous thrombolympangitis. In the inflammatory areas surrounding the active tuberculous disease the lymph spaces were found to be filled with fibrin and round cells, and in many places the contents of these vessels showed true tubercles, caseation, and typical Langhan's giant-cells. Manassa does not mean that the primary infection of the larynx comes through the lymph-stream, but only that the spread of the disease inside of the larynx frequently takes place in this way.

Fetterolf, who recognizes ulceration without infiltration, gives the proportion of the various lesions as follows:

Infiltration.....	20
Infiltration with superficial ulceration.....	19
Infiltration with deep ulceration.....	4
Superficial ulceration.....	10
Deep ulceration.....	4
Tuberculoma.....	2
	<hr/> 59

In the 83 cases of laryngeal tuberculosis examined the epiglottis was involved in 59; the aryteno-epiglottic folds in 58; the ventricular bands in 42; the vocal cords in 49; the arytenoid cartilages in 57; and the inter-arytenoid space in 49. Of course it will be seen from these statistics that in the majority of cases of advanced laryngeal tuberculosis, the greater part of the larynx is usually diseased.

**Prognosis.**—Undoubtedly even extensive tuberculous lesions of the larynx sometimes heal spontaneously, but the untreated laryngeal lesions usually advance until death, the rapidity of advance keeping pace with the progress of the pulmonary condition. Death, in these cases, is usually not due directly to the laryngeal disease, though it is frequently hastened indirectly by the inability of the patient to take proper nourishment. Again where the ulceration is so great that drink and food pass into the trachea, inspiration pneumonia may terminate the patient's suffering. On the other hand, proper local and general treatment, if started early enough should cure 90 per cent. of the cases, provided the lung condition does not terminate the treatment. Even in fairly well advanced cases appropriate local treatment really affords more than an even chance. I have frequently seen the laryngeal lesions heal up under the use of the cautery, while the pulmonary lesion was progressing. This prospect of cure, however, applies only to intrinsic lesions or where the pathology is strictly confined to the laryngeal structures alone. While an isolated tuberculous ulcer on the pharyngeal wall is as a rule easily cured, I have yet to see recovery in a case where the disease starting in the larynx had progressed until more or less of the pharynx was involved.

**Subjective Symptoms.**—Tuberculosis of the larynx produces two types of symptoms, one being the result of interference in the normal physiology of the parts affected, and the other the result of the irritation of the disease which produces a localized peripheral neuritis or an extreme sensitiveness from exposure of the nerve endings in ulceration.

The interference in the physiology produces voice changes from a mild huskiness to complete aphonia, and where the disease has spread beyond the confines of the larynx, there may be distinct dysphagia, which is not due to pain but to a crippling of the normal muscle action. Also, when there is interference with the closure of the glottis, a violent cough may occur during attempts at swallowing, because some of the food enters the larynx.

There are certain types of changes in the voice which are more or less peculiar to laryngeal tuberculosis, though not in a true sense pathognomonic. In advanced disease the voice possesses a peculiar raucous sound, due to the vibration of approximating parts of the swollen laryngeal mucosa other than the vocal cords. Again, the voice sound may vary considerably in its timbre, due to the presence or absence of secretion in the

**larynx.** The general weakness of the patient tends to decrease the amount of sound, producing a condition termed "amblyphonia".

Aphonia may occur in almost any stage of the disease and may be caused either by destruction of the vocal cords directly, by swelling or tumor formation which prevents their proper approximation, by fixation of the arytenoid cartilage from perichondritis of the crico-arytenoid joint, or occasionally from paralysis due to pressure on the recurrent laryngeal nerve by large bronchial or even cervical lymph-nodes.

Recurrent aphonia is frequently seen in pulmonary tuberculosis without active lesions in the larynx. This condition really belongs with the neuroses, though it is usually associated with some catarrhal laryngitis. The patient may lose his voice for several weeks then suddenly recover it completely. Aphonia, unless persistent, is therefore not always an indication of laryngeal involvement.

Dysphonia, or difficulty in talking, is usually found in those cases where there is an infiltration in the posterior commissure of the larynx in which the swelling is just sufficient to prevent an easy approximation of the cords. The patient can talk in a clear voice if he tries hard enough, but it is a distinct effort to phonate and he soon tires.

Although inflammation of the larynx may cause severe cough I doubt whether the secondary involvement of the larynx in cases of pulmonary tuberculosis has much influence on the amount or the severity of the cough. Ulceration in the posterior commissure is more prone to produce cough than lesions in other portions of the larynx. Again where the lesion interferes with the closure of the glottis, violent coughing may result from drink or food entering the larynx.

**Pain** is the most distressing symptom of laryngeal tuberculosis, but is dependent more upon the location of the disease than its extent. Though, at times there may be more or less constant distress, the extreme suffering is usually present only during attempts at swallowing.

Odynphagia, or painful swallowing, may occur at an early stage, when the lesion is extrinsic. It is most marked when the epiglottis is involved, but may be very severe where there is ulceration on the exposed portions of the arytenoid bodies or along the aryepiglottic folds. Usually even advanced intrinsic lesions cause comparatively little pain, though it may be fairly severe when the posterior wall or the crico-arytenoid joint is involved. It must be remembered, therefore, that the absence of pain is no proof that the larynx is not affected. Usually the pain is felt directly in the diseased area, but sometimes is referred to the upper part of the neck behind the angle of the jaw, and especially to the ear. This stabbing, shooting, ear pain is, at times, most distressing. When the soreness is due to ulceration of the epiglottis, arytenoids, or other exposed structures, the swallowing of solid food may become practically impossible, though liquids can be taken with a comparative degree of comfort. On the other hand if the musculature, especially that of the pharynx, is infiltrated the swallowing of liquids becomes almost, if not just, as painful, as the swallowing of solid foods. In these cases even the swallowing of saliva is so distressing that the patient is constantly expectorating.

Tenderness is not usually seen in the earlier cases, but may be elicited by pressure over the thyroid cartilage if the disease is at all advanced. More frequently we find tenderness over the superior laryngeal nerve, especially

as it emerges through the thyrohyoid membrane. This nerve tenderness is somewhat difficult to explain except on the theory of a peripheral neuritis.

In the advanced cases great discomfort and often actual pain is caused by the adherence of partially dried pulmonary secretions to the inflamed laryngeal mucosa, and when the patient, through weakness or for other cause, becomes a mouth-breather, the dryness of the pharynx and larynx may become most distressing.

**Objective Symptoms.**—In spite of the expressed opinions to the contrary, we cannot recognize a so-called “pretubercular stage” or any general appearance of the mucous membranes of the pharynx or larynx that would indicate the presence of a tuberculous implantation. The general pallor of the palate and pharyngeal mucosa that has often been alluded to as a diagnostic indication is the result of a general anemia and not any local tuberculous deposit. In a like manner a general redness of the larynx often seen in phthisical patients is of a catarrhal or, in the more severe cases, of a septic nature and has nothing to do with a possible local tuberculosis, except as a etiological factor. It is possible that an acute or chronic congestion of the mucous membranes may open up a way for the implantation of tubercle bacilli, but this possibility is not the fact of actual disease and, therefore a congested larynx per se, even though occurring in a pulmonary case and causing symptoms, cannot be called tuberculous.

The appearance of a tuberculous larynx varies greatly according to the stage of the disease and to the part involved. This fact makes description difficult and complicated if we attempt to portray the whole picture. There is usually little difficulty in recognizing the condition in the advanced cases while the very early lesions are frequently overlooked, and it is of great importance that these early lesions shall not be overlooked because it is with them that local treatment will accomplish the best results. We shall, therefore, take up the different parts of the larynx and endeavor to describe, first, the earliest departure from normal that should arouse our suspicions and, second, briefly state the subsequent progress of the disease. Also, it must be remembered that the first lesion of tuberculosis is an infiltration and that the ulcer only occurs when the growing tubercle has reached the surface.

*Epiglottis.*—(Plate IX.) The first change from the normal is a unilateral thickening on or near the free edge usually just off the middle line, though sometimes further down near the base. It is distinctly redder than the surrounding mucosa, is not very sharply defined, and is of a firm appearance, not edematous. Generally this spreads until the whole epiglottis is greatly thickened and deformed, sometimes assuming a turban-like shape. Less frequently the involved area breaks down early and forms ulcers before spreading over the rest of the organ. The epiglottic ulcer is cleaner cut than the tuberculous ulcer occurring in less dense tissue and frequently the edges of the cartilage are laid bare. The ulcers become multiple, coalesce and the disease progresses to complete destruction of the whole epiglottis.

*Arytenoid Region.*—(Plate IX.) Here the first evidence of involvement is a smooth edematous swelling of the arytenoid body, usually bilateral though it may be unilateral, and spreading more or less upward along the aryepiglottic fold. While this edema is very similar to that seen associated with septic processes in this region, when seen in a person with pulmonary tuberculosis it is practically pathognomonic of a localized lesion. This

PLATE IX



1. Showing pulmonary secretions stagnating on posterior commissure and left vocal cord with slight congestion of the vocal cords. Condition often seen in pulmonary tuberculosis and the chief etiologic factor in secondary involvement of the larynx.



2. Early tuberculosis of the right side of the epiglottis and of the left arytenoid.



edema may progress until the arytenoid bodies are swollen to such an extent that they almost close the opening of the larynx, and is seldom associated with ulceration. Less commonly perhaps a slightly swollen edematous arytenoid gradually becomes more solid in appearance or reddened and is evidently becoming infiltrated with tubercles. In the infiltrated arytenoid ulceration may take place at a very early period, but it is only in exceptional cases that this ulceration progresses to an extent which destroys the whole arytenoid body. This infiltrated condition of the arytenoid may extend upward along the aryepiglottic fold to the base of the epiglottis, and when this happens closure of the laryngeal opening becomes difficult on account of the immobility of the upper walls of the larynx.

*Posterior Commissure.*—(Plate X.) In this region the earliest departure from the normal is a thickening of the mucous membrane. This thickening is pale in color, seldom smooth in outline, and almost always papillomatous. While we can have papillomatous hypertrophy of the epithelium in this position, in ordinary chronic hypertrophic laryngitis the amount of tumor formation is never so great as seen when it is due to an underlying tuberculosis. The amount of swelling can be better estimated during phonation, as approximation of the arytenoid cartilages pushes the mass prominently into the laryngeal space. When the swelling is very marked accurate approximation of the vocal cords becomes impossible.

*Vocal Processes.*—(Plate X.) One of the most pathognomonic lesions of laryngeal tuberculosis is a smooth, reddened, swollen area, involving the vocal process and the region immediately above it. It is almost always unilateral and the neighboring portion of the cord is usually more or less congested. Later on ulceration may appear here, but generally is not seen except in advanced cases.

*Vocal Cords.*—(Plate X.) The first evidence of a tuberculous implantation on the vocal cords is usually a more or less discrete, reddened, swollen area somewhat resembling a small fibroma, while the whole cord is apt to be more or less discolored. Sometimes minute superficial ulcers occur along the edges of the cord due to the breaking down of small tubercles in the thin adherent mucous membrane. These ulcers give to the cord the so-called "mouse-eaten" appearance. From these beginnings the ulcerative process may go on until a great part or all of the cord is destroyed. The lesion may be either unilateral or bilateral, and at times an ulcer on one cord may apparently infect the approximating portion of the other.

*Ventricle of Morgagni.*—The recognizable lesions of this cavity occur as an infiltrated mass pushing outward below the ventricular band hiding the vocal cord to a greater or less extent. While this infiltration is usually paler than the normal mucosa it frequently appears as a congested area because of the white background of the vocal cord just beneath.

*Ventricular Band.*—Lesions of the ventricular band show no peculiar characteristics. They occur less commonly than in other portions of the larynx, appearing first as elevated, infiltrated areas, but later breaking down and ulcerating. Sometimes the whole side of the larynx, from the edge of the ventricular band upward, appears greatly swollen and red, due to a perichondritis of the thyroid cartilage, with abscess formation.

In the later stages of laryngeal tuberculosis arthritis of the crico-arytenoid joint is not uncommon, and the condition is manifest by a fixa-

tion of the vocal cord on the affected side and is usually associated with a very marked swelling of the arytenoid body.

While the typical appearance of the tuberculous ulcer is often modified by its location and the presence of secondary infection, there are certain features which are more or less characteristic. The edges are ill-defined and wavy, and the base, when it can be seen, appears granular. The surrounding tissue is thickened from infiltration that often hides the real depth and extent of destruction. As a rule the ulcer is comparatively clean and sloughing tissue is not often seen except where cartilage is involved. The healing of the ulcer is not accompanied by much scar formation, so that the parts are seldom deformed by cicatricial contraction. When there is deformity following the healing of extensive ulceration, it is due to destruction of tissue, or occasionally to the healing together of approximated ulcerated areas as is sometimes seen following extensive involvement of the vocal cords.

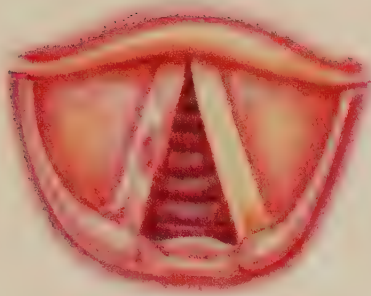
**Diagnosis.**—In advanced cases there is usually little difficulty in making a diagnosis as the condition is always associated with a pulmonary lesion. It is to be remembered, however, that the presence of a pulmonary lesion does not rule out the possibility of syphilis or cancer and that it is entirely possible to have mixed lesions. However, in the large majority of cases, unless the lesion seen in the larynx is very characteristic of some other condition, the presence of pulmonary tuberculosis is very good evidence that a suspicious spot in the larynx is tuberculous. Hence, the importance of thorough chest examination, both by an internist and by the Roentgen ray and laboratory findings, whenever we happen to be confronted with a doubtful though distinct laryngeal pathology. In some cases where the laboratory has failed to show bacilli in the sputum, and the chest lesion is doubtful, diagnosis is exceedingly difficult and only continued observation or a biopsy will settle the doubt.

Acute inflammatory processes in the lower pharynx, impacted foreign bodies, and occasionally severe acute inflammation of the larynx, will produce edema of one or both arytenoid bodies, which in its clinical appearance resembles very closely the edema of tuberculosis. The diagnosis, however, is usually cleared up by the history of the case, the acuteness of the attack, and the appearance of the surrounding structures. Abscess of the epiglottis may resemble a tuberculous infiltration, but here again the history of the case comes to our aid.

Ulcerations that are due to syphilis are not easily confounded with a tuberculous lesion. The syphilitic ulcer is sharply defined with clean-cut edges and surrounded by a highly congested, but smooth mucosa. The Wassermann test or specific therapeusis should clear up the diagnosis, except perhaps in those cases of mixed lesions, when it may become impossible to tell which is the syphilitic lesion and which is the tuberculous.

Malignant disease of the larynx may under certain circumstances become very difficult to distinguish from tuberculous lesions, especially when they occur, as they not infrequently do, in persons with an old tuberculous history or with the evidence of actual pulmonary disease, either active or healed. Quite often a positive diagnosis cannot be made without extensive laboratory examinations, repeated observations, perhaps a trial of therapeutic remedies, and sometimes a biopsy. A word of warning in making biopsies in the tuberculous larynx. The removal of tuberculous tissue from

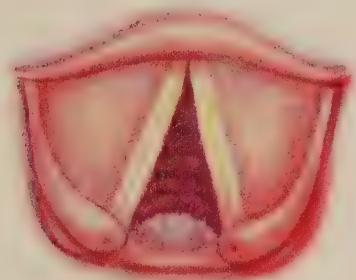
PLATE X



1. Early tuberculosis of the left vocal cord. Thickening in the posterior commissure is a simple hyperplasia and not tuberculous.



2. Early lesion of the left vocal process, one of the most pathognomonic and characteristic early lesions.



3. Beginning tuberculoma of the posterior commissure. Note differences between the appearance in this case and that in illustrations 1 and 2.



the larynx is not attended by any unusual risk as far as the operation itself is concerned, but one should remember, however, that a raw surface is left unprotected against the pulmonary secretion, and that the squeezing effect of the punch-forceps which is usually employed in this operation, has a tendency to force the tubercle bacilli into the surrounding meshwork of lymphatics and hence cause some dissemination of the disease. It is advisable, after removal of the piece of tissue, to gently sear the wound with the actual cautery.

In hyperplastic laryngitis there is sometimes a thickening of the laryngeal mucosa, especially in the posterior commissure, which more or less closely resembles tuberculous infiltration, and is to be differentiated more by the degree of swelling and the presence or absence of other symptoms than by any peculiar appearance of the condition itself.

Among the less common laryngeal diseases, which may at times be mistaken for tuberculosis are pachydermia laryngis, laryngeal scleroma, lupus, and leprosy. They are, as a rule, easily differentiated by careful observation of their clinical characteristics, though sometimes a detailed history and physical examination with all the aids of a well appointed laboratory, and perhaps a continuous study of the case, are necessary before a diagnosis is made.

**The Surgical Treatment of Laryngeal Tuberculosis.**—For the successful handling of tuberculosis of the larynx, it is essential that, along with the local measures, the patient shall receive the best general care and treatment. As a rule the laryngeal lesion must be looked upon as simply an extension of the pulmonary disease, and the healing of the larynx is just one step toward the cure of the patient. Therefore, in the pursuance of our local treatment it is necessary to remember that the topical measures, either surgical or medical, should not be of a nature that tires or exhausts the patient, and it is sometimes wiser to dispense with the local treatment entirely, if it is followed by a rise in temperature and more or less exhaustion of the patient.

Local treatment may be of two types, one palliative and the other curative, but both of these overlap in their application. Palliative measures will, at times, permit the swallowing of more food with the consequent improvement in the nutrition of the patient, while on the other hand the curative methods are frequently, and almost at the moment of their adoption attended with distinct lessening of the pain and soreness. The object of surgery is essentially curative and should not be undertaken in patients where palliative measures alone are advisable. Yet we must recognize that in many cases the healing of ulceration on the edge of the epiglottis or on the top of the arytenoid bodies may be the best palliative procedure, and when this can be effected without exhaustion to the patient, or where the promise of relief is sufficient to warrant a certain amount of fatigue, we are justified in adopting surgical methods even in hopeless cases.

With the exception of tracheotomy, external operations for tuberculosis of the larynx are almost never, if ever, called for. Personally I cannot conceive a case where laryngectomy or even thyrotomy would be justified. Certain authorities have asserted that tracheotomy may at times be a justifiable procedure for the sole purpose of putting the larynx at rest. Unfortunately, in cases of pulmonary tuberculosis, tracheotomy is not a simple affair, as the danger of extensive wound infection of the neck

from the pulmonary secretions is a very real one and should contraindicate this operation entirely except for one condition, and that is laryngeal obstruction. Though laryngeal obstruction from tuberculosis seldom becomes severe enough to demand a tracheotomy, I have had to perform it twice, once because of bilateral paralysis of the recurrent nerves, and once for intense swelling and edema in the subglottic region. In both of these cases the wounds became infected, with extensive sloughing of the neck tissues, and the patients' lives were prolonged only a month or so.

Occasionally the opening and drainage of an external abscess, which has resulted from necrosis of the thyroid cartilage, become necessary.

Internal surgery of the larynx includes both the bloody removal of tuberculous areas and the application of the galvanocautery throughout its extended field of usefulness. The surgical removal of infiltrated areas, and the curettement of tuberculous ulcers, as advocated by Herying, Lake, Krause, and others, are distinct advances in the treatment of this disease, and are frequently attended by very good results. Their use, however, has become distinctly limited because the electric cautery will, in almost every instance, accomplish the same results with less risk of shock to the patient or the spreading of the infection, and further does exceeding more than could ever be hoped for from the older surgical procedures.

Bloody surgery should be limited to the removal of tuberculomata, especially those larger tumor-like formations, which are sometimes found projecting from the posterior commissure, and to the removal of diseased epiglottides. Curettement of tuberculous ulcers and diffuse infiltrations should never be undertaken. A large tuberculoma in the posterior commissure that shows no evidence of ulceration may, at times, be cleanly and wisely removed by a pair of good laryngeal punch-forceps, provided the operator has the skill to remove only the proliferated epithelium, and does not cut into the underlying infiltration. The operation should be done under local anesthesia and by either the direct or indirect method of laryngoscopy. After the removal of the growth the denuded area should be lightly touched with the galvanocautery. This cauterization must be very superficial, as there is a possible danger of disturbing the arytenoid joint, especially when the original removal has been too deep.

When there is complete involvement of the epiglottis the amputation of the whole of the projecting portion is not only justifiable, but is frequently the best way of handling these cases. Its removal is seldom accompanied by any interference with swallowing of liquids or solids, and the relief from pain is sometimes marvelous. The operation is usually accomplished with little shock to the patient and the hemorrhage is insignificant. The only danger is that as a result of the compression by the amputating instrument, tubercle bacilli may be forced along the meshwork of lymphatics with a spreading of the disease down along the aryepiglottic folds. The operation may be done either with punch-forceps, such as those of Lake, or preferably by the cautery snare. When the punch-forceps are used the raw surface should be touched with the electric cautery.

Local anesthesia may be obtained by the direct application of a 10 per cent. solution of cocaine, two or three applications usually being necessary before sensation is completely destroyed. A better position can be obtained with the snare if the wire is made up of platinum-iridium instead of pure platinum, as the former has almost the resiliency of steel wire.

Sometimes the laryngeal mirror is necessary, but usually the epiglottis can be brought into view by firm pressure on the base of the tongue. The snare is then introduced into the mouth with the cannula at the side of the tongue, and the loop made to encircle the epiglottis as close to the base as it can be pushed. The snare wire is then tightened until the base is distinctly constricted. It is wise at this time to grasp the tip of the epiglottis with a pair of tooth-forceps, though this is not necessary as the severed portion usually adheres to the snare wire, and if it should fall into the throat is easily expectorated by the patient. The current is then turned on and the loop gradually constricted until the epiglottis is completely severed. The removal should not be hurried, as the searing of the stump by the heated wire as it cuts through is important as a preventive of the spread of the disease and as an aid in hastening cicatrization.

The electric cautery is undoubtedly the best surgical method of treating laryngeal tuberculosis and the results attending its proper use have taken this disease out of the list of laryngological nightmares. The object of the cauterization is the production of an eschar rather than the destruction of all tuberculous tissue, and this fact makes the procedure a comparatively minor affair. The production of the eschar is followed in about three days by formation of a separating zone of granulation tissue which is necessary for the sloughing off of the heat-destroyed tissue. The development of this granulation tissue is attended by the formation of new blood-vessels which grow from the periphery through the previously avascular tubercle. This brings nutrition to the fixed connective-tissue elements, enabling them to withstand the action of the tubercle toxin, and probably also causing the epithelioid cells themselves to develop into fibroblasts. To repeat then, the object of the cauterization is not so much the destruction of diseased tissue as it is the healing of the tubercle; and because of the limited trauma necessary important organs are not removed or destroyed except by the disease itself. With the possible exception of very large tuberculomata and completely diseased epiglottides, all the clinical types of localized tuberculous lesions are more successfully combated by the cautery than by any other form of treatment.

The application of the cautery may be carried out either by direct or indirect laryngoscopy. In some cases the direct method can be used with advantage, but the ease with which the cauterization of the larynx can be done by the indirect method makes this method the one of choice in the majority of cases. It is, of course, essential that the operator be thoroughly trained in the technic of intralaryngeal manipulation of instruments. In order that the operative field be distinctly seen and the cauterization controlled by sight during the operation, it is sometimes necessary that the patient be trained to tolerate the intralaryngeal manipulation before the cauterization is attempted. Daily swabbing of the larynx with mild antiseptics is often a very useful preoperative measure as it tends to reduce the secondary infection and bring about tolerance on the part of the patient.

Concerning the equipment, it is important that the supply of electricity, whether from battery or from street current, controlled by a rheostat, is of sufficient strength to heat the cautery knives to a white heat almost instantaneously. The platinum point of the knife should be shorter than the usual pattern in order to lessen the danger of burning the normal

mucosa, which is apt to occur at times from spasmodic closure of the larynx. Three types of knives are necessary: The sharp-pointed knife for puncture; the large, flat blade for searing ulcerations; and a small wire loop for treating small infiltrations (Fig. 456).

The operation should always be done under local anesthesia. Two methods are advocated. The drop method has the advantage of being less liable to provoke laryngeal spasm and is somewhat less disagreeable to the patient. This method consists of dropping from a laryngeal syringe, 2 or 3 drops of a 10 per cent. solution of cocaine on the top of the epiglottis and laryngeal aperture. After waiting about five minutes, 4 or 5 drops more of the 10 per cent. solution of cocaine are gently instilled into the larynx during phonation. The cauterization can then be done after a lapse of about fifteen minutes.

The following method of direct application is usually preferable because of the more complete and quicker anesthesia obtained. Two grains of

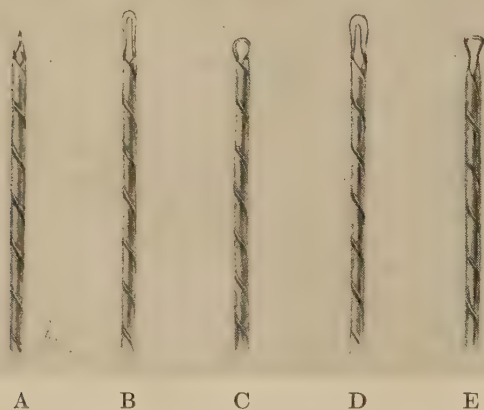


Fig. 456.—Cautery points for treatment of laryngeal tuberculosis. A, Sharp point for ignipuncture. B, Narrow flat blade that can be bent laterally, especially adapted for reaching the ventricle of Morgagni. C, Small wire loop for treating small granulations. D, Large flat knife for searing ulcerated surfaces. E, Flat stirrup-shaped loop especially adapted for treating upper surface of vocal cords.

powdered cocaine are placed in a sterile glass and a firmly wound cotton applicator soaked with sterile water is dipped in the powder. The first application is made into the larynx as far as possible, the patient's reflexes usually preventing the swab being introduced below the ventricular bands. After waiting three minutes a second application is made, using more of the original 2 grains of cocaine; and it will be found that this time the swab can be carried directly to the area to be cauterized. After waiting another three minutes one is generally able to proceed with the cauterization. However, sometimes a third application is necessary, but one should never use more cocaine than the original 2 grains, in order to avoid toxic symptoms.

Experience alone enables the operator to best judge the amount and character of the burn which he should make in each individual case. As a guiding rule edematous swelling and infiltration require ignipuncture, while ulcerations should be seared with a broad knife. True tuberculomata must be more or less completely destroyed, and for this purpose a larger, broad

knife is necessary. In making ignipunctures the point of the knife while cold is pressed firmly into the area to be cauterized. Where there is true infiltration the point of the knife, even when cold, penetrates readily into the diseased tissue, so that the depth of the cauterization can be accurately determined before the current is turned on. Sometimes, however, especially in edema of the arytenoid bodies, the pressure necessary to insert the cold point has a tendency to dislodge the instrument from the point to be attacked. Under these circumstances the current should be turned on while gentle pressure is being made and the knife will then sink easily into the tissue. The burning should cease as soon as the tissue in the immediate neighborhood of the point turns white. As a rule several of these ignipunctures can be made at one sitting, not over one second being required for each burn. In treating ulcerations the whole base of the ulcer should be distinctly seared, but unless there is a great deal of infiltration the searing should be rather superficial. If this infiltration is thick a portion of it may be wisely destroyed.

When the epiglottis is involved without ulceration, a series of punctures should be made into the swelling about  $\frac{1}{8}$  to  $\frac{1}{4}$  inch apart. If ulceration is present the exposed tissues should be seared with a flat, broad knife. It is only in the exceptional case that one has to resort to a complete removal of the epiglottis, for although freedom from pain may not be quite so quickly obtained by the simple cauterization, the very great risk of a dissemination of the tubercle bacilli is avoided and we are able to preserve as much of the epiglottis as has not been absolutely destroyed by the tuberculous disease.

In the pyriform swellings of the arytenoid bodies ignipunctures are remarkably efficient, and the rapid reduction in the swelling is sometimes astonishing. As with the epiglottis the punctures should be made in a row from  $\frac{1}{8}$  to  $\frac{1}{4}$  inch apart. For true intrinsic lesions ignipuncture should not be used except perhaps in cases of distinct infiltrative and ulcerative disease of the ventricular bands, or of the wall of the larynx just above them. Infiltrative growths below this level, interfering with the functioning of the cords, are usually so intimately connected with these important structures that great care must be exercised in their reduction. The amount of destruction can be more accurately gauged if the tissue is attacked from the surface rather than from a puncture of indeterminate depth. Lesions of the vocal cords, of the vocal processes, and of the interarytenoid space or posterior commissure should be attacked with the small wire loop. The object desired is to remove the exuberant tissue down to the level of the normal structures and not deeper unless the diseased process has already destroyed their contour. When the ventricle of Morgagni is involved a broad knife should be bent sidewise so that it can be introduced just below the edge of the false cord.

If the cauterization has been done under careful visual control violent reaction following its use is extremely rare, though it is wise in the beginning of the treatment of a case to do comparatively little at the first sitting, in order to observe whether there is any idiosyncrasy on the part of the patient to this method of treatment. Usually the resulting soreness is only enough to cause a slight discomfort for the first twenty-four or forty-eight hours and frequently we are astonished by the diminution in pain which follows immediately after the burn. When the vocal cord is attacked

complete aphonia may follow for a day or so, but unless the cauterization has been unwarrantedly deep no permanent loss of voice is at all likely. Severe hemorrhage practically never occurs though sometimes a patient may spit a little bloody mucus for a day or so. The repetition of the cauterization in the same area should not be undertaken until the previous burn has healed. As is usually the case, however, other areas of the larynx need to be treated and then a second cauterization may be done within three or four days, or as soon as the danger of reaction has passed. After the cauterization, if there is any pain, the patient should be instructed to suck cracked ice and perhaps have an ice-collar placed around the neck. Rest of the voice should be insisted upon for several days, especially when the operation has involved structures near the vocal cords or the posterior commissure.

The benefit from the cauterization cannot be fully determined until probably three or four weeks have elapsed, during which time the slow process of cicatrization is going on, although it is not uncommon to see tuberculous ulcers covered with normal epithelium within a week or ten days following the treatment.

GEORGE B. WOOD.

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### MEDICAL TREATMENT OF LARYNGEAL TUBERCULOSIS

A normal larynx in a patient who has active pulmonary tuberculosis is rare.

Tuberculosis of the larynx, practically, if not always, is secondary to tuberculosis of the lung; consequently, it should be treated as a complication and not as a disease entity. It must be remembered that practically every disease to which the larynx is heir may occur in patients suffering with pulmonary tuberculosis. Not only is a correct diagnosis of tuberculosis of the larynx necessary to insure satisfactory results, but also the type, extent, and location of the lesions determine whether surgical or medical methods should be used. It is not a question of superiority of surgical over medical methods, but rather which method is better suited to each individual case and this must be determined by the laryngologist, aided by the internist.

Like pulmonary tuberculosis, the duration, extent, and severity of involvement of the organ, exert a powerful influence on the prognosis and treatment of the disease; consequently, an early examination of the larynx in all tuberculous subjects is imperative for the best results.

In considering the treatment of laryngitis in tuberculosis several things have to be considered. First, the particular lesion of the larynx with which we are dealing. Second, the particular line of treatment we should carry out. Third, the result for which we should strive in the particular case under consideration. Fourth, the effect of local treatment upon the general condition.

The most common lesion of the larynx in the tuberculous patient is not tuberculosis as might be supposed, but simple inflammation due to excessive cough and the irritation of bombarding secretions. Very often this irritation is most pronounced in the posterior wall of the larynx and

the arytenoids. It is bilateral and is frequently accompanied by thickening of the epithelium in long-standing cases and in the more acute cases by swelling of the tissues.

Syphilis may invade the larynx in tuberculous subjects and may be mistakenly diagnosticated as tuberculosis and it can be readily understood that any treatment but antisyphilitic would be useless.

In tuberculous patients of advanced years, malignant growths may be present in the larynx. These lesions must be ruled out before active local antituberculosis treatment is instituted.

Given a larynx which is definitely diagnosticated tuberculous, the results of local medical treatment depend upon the character of the lesion, the extent of the lesion, the condition of the lung, and the resistance of the patient. If the laryngeal lesion is seen early, the invasion is limited, the patient's general condition is good, and the lung lesion is under control, a cure may be expected under appropriate local treatment. If the laryngeal lesion is advanced through neglect or improper treatment, the lung lesion is controlled, and the patient's general condition is good, the prognosis as regards the larynx is good, if proper local treatment is carefully carried out. If the lung lesion is uncontrollable, or the patient has little or no resistance, the prognosis is hopeless, regardless of the extent of the laryngeal lesion. In these hopeless cases careful treatment may and often does check the laryngeal lesion or even improve it to a certain extent, but almost invariably the disease eventually progresses to a disastrous finish. In advanced hopeless cases, while cure or arrest of the disease is out of the question, enough good may be done by appropriate treatment to save the patient from a great deal of pain and discomfort and lessen the agonies of dysphagia and odynphagia.

The medical treatment of tuberculosis is both general and local. The general treatment is not in the province of the laryngologist and should be conducted by the internist, the laryngologist confining his attention to the local treatment of the upper air-passages, and be guided in the intensity of his treatment of the larynx by the opinion of the internist. There are times when the general condition of the patient forbids any active treatment of the larynx, and at such times it is jeopardizing the best interests of the patient to manipulate the already hypersensitive larynx.

The local treatment of tuberculosis of the larynx (excepting cautery methods) is necessarily applied to the surface of the organ, thus naturally topical applications can affect only surface lesions.

Clinically, 25 to 30 per cent. of pulmonary tuberculosis cases have laryngeal involvement. Many cases of tuberculosis of the larynx are unrecognized clinically, due to lack of routine laryngeal examinations and receive little or no treatment. Undoubtedly some of these undiagnosed cases are arrested spontaneously.

**Vocal Rest.**—The most important local treatment is vocal rest. Vocal rest means prohibiting the use of the voice; it does not mean merely changing from one form of vocal activity to another. Vocal rest should be complete in some cases, in other words, the patient should be compelled to use pad and pencil. In other cases, whispering or modified vocal rest may be allowed. "Stage whispers" are not permissible. A great deal more laryngeal strain is caused by stage whispering than by actual use of the voice in ordinary conversation. Partial vocal rest may be allowed in some

cases of mild involvement for two reasons: first, to keep up the tone of the muscles of the larynx, and second, to encourage a patient who may be despondent because of the fear of not being able to regain his voice. Partial vocal rest consists of absolute vocal rest excepting at stated periods of the day, and for stated lengths of time. I have found it a good working rule to allow patients under partial vocal rest to talk for a few minutes during meals. In this way the meal acts as a check, inasmuch as the patient knows that outside of meal hours he is not to use his voice.

The use of watery solutions either locally applied or by instillation, practically always produces cough or at least temporary irritation of the larynx. Consequently, any drug used in the larynx is better tolerated if mixed with oil or combined with oil solutions.

**Technic of Instillation.**—The instrument consists of a 2 or 5 cm. Luer or Record syringe, with a 4-inch laryngeal cannula (Fig. 457). The long old-fashioned so-called “laryngeal cannula” for intralaryngeal instillation is clumsy and does not answer the purpose satisfactorily.

The object of using the small cannula is to avoid touching any portion of the throat or larynx, thus removing cause for cough. It is not necessary to put the cannula between the cords for intratracheal medication. The oily preparation can be seen by the mirror until it passes the cords.



Fig. 457.—Laryngeal syringe. This is a 2-cm. Record syringe armed with a 4-inch cannula with a  $\frac{1}{4}$ -inch beak. It is specially designed so as not to touch any portion of the tongue, pharynx, or larynx, thus avoiding contact irritation.

The patient holds the tongue between the thumb and the index-finger of the right hand. The throat mirror is introduced with the operator's left hand, and the cannula of the syringe, filled with the required amount of solution, is placed back of the throat and a few drops expelled. The position of the syringe is regulated according to the direction of the dropping fluid. The oil should be dropped between the cords during gentle respiration and then the patient be directed to say “eh,” causing the fluid to drop upon the cords themselves through instilling oil first into the trachea and then upon the laryngeal surface. In cases where the larynx can be exposed only with difficulty, the oil may be instilled by dropping upon the posterior surface of the epiglottis, whence it will run down into the larynx.

**Direct Application by Swab Method.**—A properly shaped resilient laryngeal swab is saturated with the desired medication and gently rubbed into the ulceration for a few seconds. Vigorous scrubbing motions should be avoided. Applications in this manner should be made two or three times weekly.

In this chapter the writer is eliminating all drugs excepting those which are in common use at the present time and which have proved to be of value by the majority of men. Almost every known antiseptic and analgesic has been used in the treatment of laryngeal tuberculosis.

**Chaulmoogra Oil.**—Ordinary approved medical treatments for tuberculosis of the larynx proved very unsatisfactory to the writer until 1921

when he began to experiment with local application of chaulmoogra oil to the tuberculous larynx. Until this time the writer and his colleagues were very skeptical in regard to arresting tuberculosis of the larynx as they could not recall one definitely arrested case in six years' experience with tuberculous laryngitis. Blocking of the superior laryngeal nerve for dysphagia had been the almost daily occurrence. At the present time, however (since the results of the local application of chaulmoogra oil, after considerable experimentation, have been so gratifying) the use of alcohol injections in the Jefferson Hospital and Phipp's Institute have been reduced to an occasional one. Cases of dysphagia are now the exception rather



Fig. 458.—History.—A man aged twenty-two years had general symptoms of advanced pulmonary tuberculosis, hoarseness for two years before treatment, and slight dysphagia one year later. Became much worse during past two years. Liquids caused more pain than solids when swallowing. The larynx had been treated while in the sanatorium, without improvement. Chaulmoogra oil treatment was instituted after the patient had aphonia and severe dysphagia. Physical examination of chest showed a far advanced ulcerative tuberculosis with cavities in both upper lobes. Sputum contained tubercle bacilli.

A, Large vegetative outgrowth on right side of laryngeal surface of epiglottis involving the aryepiglottic fold. Smooth infiltration with superficial ulceration on left side of laryngeal surface of epiglottis. Both arytenoids were flask shaped. Vocal cords and ventricular bands were extensively infiltrated and ulcerated. Vegetative mass seen on right ventricular band near the arytenoid.

B, The same larynx twenty days later under chaulmoogra oil treatment. Most of the ulcerations have disappeared, and the vegetative masses are greatly reduced in size. Both arytenoids are also reduced in size. Subjectively throat is considerably more comfortable. Dysphagia greatly reduced. General condition of patient continued to become worse, although the laryngeal condition showed steady improvement.

than the rule. In my opinion chaulmoogra oil is the best medical treatment for tuberculosis of the larynx that I have used up to the present time.

Chaulmoogra oil is obtained from two varieties of trees and is known as Indian chaulmoogra oil and Burmese chaulmoogra oil. The Indian oil is a fixed oil, expressed from the seeds of *Gynocardia odorata*, a native tree of the East Indies. It contains only a small percentage of chaulmoogric acid and its properties are due to gynocardic acid. This is the false chaulmoogra oil and is of apparently little value in treating tuberculous laryngitis.

The true chaulmoogra oil is obtained the same way from the *Taraktogenus kurzii* (King), a native tree of Burma. This oil is rich in chaulmoogric acid and contains only a small part of gynocardic acid, and is efficient in treating tuberculous laryngitis.

Chaulmoogra oil may be used either in the diluted form (10 to 20 per cent. in mineral oil) or full strength. There is no ill-effect following its use if properly applied. If the drop instillation method is carried out care should be taken that the oil is dropped into the larynx and not outside of the larynx. While chaulmoogra oil is not irritating to the larynx, it is highly irritating to the mucosa of the stomach, and if repeated quantities are dropped into the stomach a violent gastritis may be produced.



Fig. 459.—*History.*—A man aged fifty-eight years complained of severe cough, marked hoarseness, and pain on swallowing, especially for liquids. Physical examination revealed advanced pulmonary tuberculosis. The sputum contained tubercle bacilli; the Wassermann test was negative. Prognosis was unfavorable.

A, February 8, 1921. The epiglottis was negative; the vocal cords were ulcerated in the posterior two-thirds; there were marked infiltration and extensive shallow ulceration of the ventricular bands; the interarytenoid fold was thickened and covered with large papillary excrescences; the arytenoids were infiltrated and about three times their natural size. The patient was bedfast.

Treatment from September 20, 1920 to January 31, 1921 consisted of applications of formaldehyde solution and lactic acid in ascending strengths, iodoform, iodine in glycerine, and argyrol. The tuberculous outgrowths on the interarytenoid fold were removed twice with a laryngeal punch, but recurred both times. Improvement was very slight.

B, The same larynx, February 2, 1922. When the chaulmoogra oil treatment was started, February 4, 1921, subjective symptoms were relieved almost at once. The distressing cough, due to the difficulty in raising tenacious sputum, was relieved after the first treatment and the sputum rendered more fluid. In less than a week the pain was gone and has never recurred. In three months' time the ulceration disappeared. The vegetations on the interarytenoid fold slowly diminished in size and finally disappeared. At the present time the infiltrated tissues are greatly reduced in size and the mucosa is smooth and clean. Hoarseness is still present, but greatly improved. The patient left the hospital three months ago and is being treated twice a week in the dispensary, practically an arrested case. The larynx shows no evidence of tuberculosis. The cords are chronically inflamed but smooth. They do not approximate because of ankylosis of the crico-arytenoid joints during the process of healing.

At the present time the patient is a street vendor and although he returns to the dispensary at irregular intervals for examination, he has received no local or general treatment for the past eight months. There has been no recurrence since the treatment has been stopped. The sputum is scanty and free from tubercle bacilli.

In the early stages of tuberculous laryngitis with slight infiltration or small ulcerations, the oil acts fairly quickly. In more advanced cases a rather long time is required for results as regards healing. Where dysphagia is present, relief often occurs in a short time, and while the dysphagia may persist for some time, still there is almost immediate relief to a more or less extent from the first treatment.

Chaulmoogra oil may also be applied directly to ulcerations by the swab

method. This method is especially valuable when the ulcerations are on the epiglottis or the arytenoids. Daily applications are unnecessary and

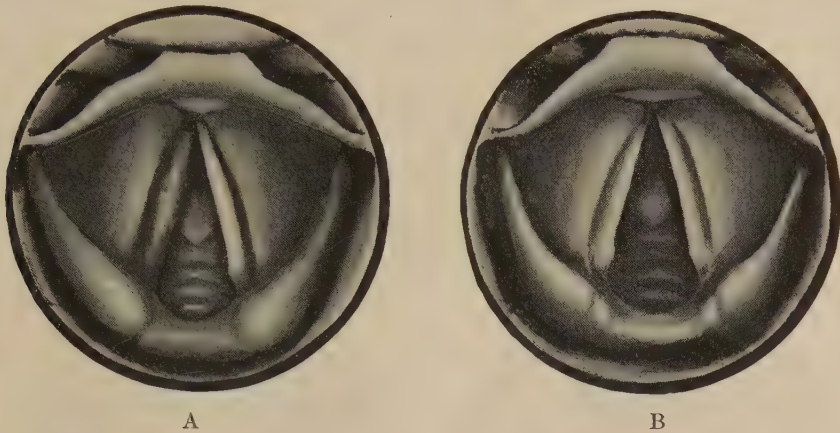


Fig. 460.—*History*.—Girl aged twenty-four with moderately advanced pulmonary tuberculosis. Repeated positive sputa for tubercle bacilli. She complained of hoarseness for a short period of time, no dysphagia. Routine examination of the larynx showed right cord to be red, spindle shaped, and granular. The rest of the larynx was comparatively normal. Chaulmoogra oil and vocal rest were begun February 1, 1926 and the right cord became normal by October 13, 1926.

A, Tuberculosis of the right vocal cord. Note the spindle shape and the granular surface.

B, Sketch of same larynx made seven months after A. Hoarseness entirely gone and patient allowed to use the voice.

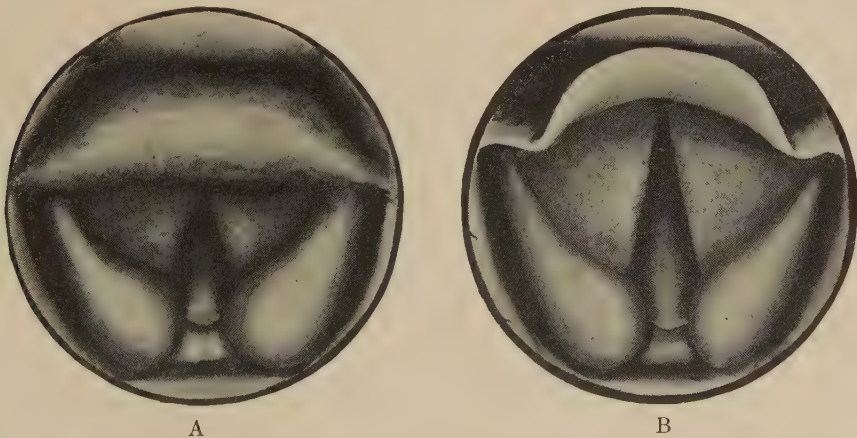


Fig. 461.—*History*.—A young man, with symptoms and signs of advanced pulmonary tuberculosis. Tubercle bacilli in sputum; blood Wassermann positive. Patient bedfast.

A, Drawing shows epiglottis to be greatly thickened and granular. The color is dark red. The rest of the larynx is much paler in color. Diffuse ulceration of ventricular bands which hide the true cords. Arytenoids are of the pyriform type.

B, Same larynx three months later showing epiglottis which had returned to normal without scarring or deformity under antiluetic treatment. Chaulmoogra oil discontinued during the antiluetic treatment. Later the ventricular bands and arytenoids improved somewhat under the chaulmoogra oil treatment, but to no marked degree.

might cause undesirable reactions. Both methods of treatment require practice to be effective and many failures of chaulmoogra oil treatment are due to faulty technic.

Cocaine anesthesia to the palate, uvula, oropharynx, and larynx usually is required in the swab method until the patient learns to tolerate manipulations in the region of the larynx. Patients quickly become accustomed to the procedure and cocaine can be dispensed with after a few treatments.

Chaulmoogra oil treatment is applicable to ulcerative, tuberculous laryngitis and superficial infiltrations. Edema of the various elements of the larynx is better treated by galvanocautery methods.

The tenacious nature of chaulmoogra oil increases its effectiveness by prolonging its action upon the underlying tissues.

**Formalin.**—The value of formalin is said to be due to its ability to penetrate the tissues. Formalin to be effective should be made up fresh

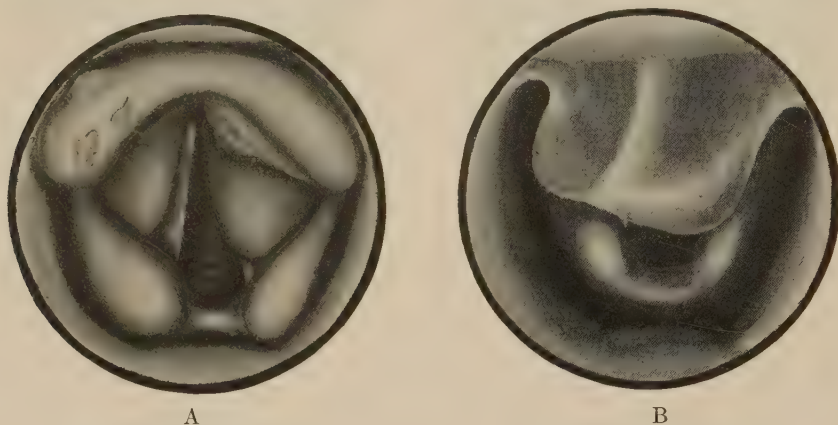


Fig. 462.—*History.*—A man aged fifty-five years. Hoarseness for four months before treatment. One month later, dysphagia. Chest examination revealed a moderately advanced pulmonary tuberculosis. Laryngological examination showed nodular swelling of the epiglottis and changes in the arytenoids and ventricular bands. There was a question as to malignancy in this case because of the laryngeal picture and age of the patient. Biopsy done by Dr. Chevalier Jackson showed that the lesion was tuberculous. Chaulmoogra oil treatment was instituted.

A, Shows infiltration of the epiglottis which approaches turban formation. On the posterior surface of the left side of the epiglottis there is a projecting infiltration. Both ventricular bands are infiltrated. There is also infiltration of the arytenoids and the intra-arytenoid fold. The right arytenoid is of the pyriform type. The two ulcerations on the epiglottis show where specimens were taken for biopsy by Dr. Chevalier Jackson.

B, This picture of the larynx is about one year after chaulmoogra oil treatment. In addition to chaulmoogra oil treatment one deep stab electrocautery puncture was done on the right side of the epiglottis in the early part of the treatment. The larynx has remained healed for the past two years. There is no ulceration, but there is slight swelling of both arytenoids. The interior of the larynx is hidden from mirror view by the epiglottis, which is quite distorted, being twisted over to the right and pulled down. However, there is no dyspnea or any inconvenience due to this distortion of the epiglottis.

daily from the full strength stock solution and diluted with water to the required strength. The larynx should be anesthetized with cocaine before the use of formalin until the patient becomes accustomed to its use. It must be used in very weak solution in the beginning and gradually increased, beginning with a 3 per cent. solution and increasing gradually up to a 50 per cent. solution. It is used by direct application with a laryngeal swab and is applied by a gentle rubbing motion to the infiltrated or ulcerated part of the larynx and not to the healthy tissue. Haphazard application without the use of the laryngeal mirror is to be condemned as slovenly and harmful. Two or three applications a week are usually sufficient.

**Lactic acid** is used in the same way as formalin, but is much more painful, and local anesthesia is required before each application. It is a caustic and produces a white eschar on the part treated. No further application should be made until the eschar has disappeared. It has no superiority over formalin.

**Dryness and Tickling in Throat.**—Mouth-breathing very often causes sensations of dryness and tickling in the throat, which are frequently due to inflammation of the arytenoids. These sensations are productive of irritating cough and are very annoying to the patient, and can be relieved to a great extent by appropriate treatment of the nose. The use of cocaine sprays tends to increase the dryness of the throat.

**Pain.**—The pain in tuberculous laryngitis varies in extent from dryness of the throat, through actual pain and odynphagia, to excruciating throat pain transmitted to the ear. Routine attention to the nose and nasopharynx is essential in treating this condition. The nose should be cleansed of mucoid or mucopurulent secretions, preferably by suction, and some mild oily shrinking solution applied to the mucosa. An excellent formula (H. C. solution, Jefferson Hospital) consisting of:

R.	Cocaine alkaloid.....	gr. ij.....	13
	Menthol.....	gr. j.....	06
	Oil of rose geranium.....	gtt. iiij.....	2
	Liquid albolene.....	f 3j.....	30
M.			

is the one I usually employ and also prescribe as a nasal spray to be used by the patient three times a day.

Five drops of compound tincture of benzoin upon a small lump of sugar and swallowed as it dissolves, frequently relieve the dryness temporarily. This is used after meals to prevent "upsetting" the stomach. Pain in varying degrees is usually due to swelling of the arytenoids or epiglottis or to actual ulceration and can be relieved only by local treatment to the part of the larynx affected.

Cocaine sprays afford but short relief and very often do not reach the spot at all. Cocaine also increases dryness of the throat and anorexia, both of which are extremely troublesome in the tuberculous.

**Dysphagia and Odynphagia.**—Swallowing may be difficult mechanically or so painful that the patient dreads eating. In either case the condition calls for immediate treatment. The painful swallowing not only causes suffering but also interferes with nutrition, resulting in partial starvation and lowered resistance.

The conditions causing dysphagia are edema and ulceration, especially of the epiglottis and arytenoids. In some instances the dysphagia is due to an extralaryngeal process. These cases are most rebellious to treatment.

The prophylactic treatment of dysphagia is of the greatest importance. Every case of pulmonary tuberculosis should be regarded as a future laryngeal case with its attendant dysphagia. Alcohol and tobacco especially should be absolutely forbidden. One patient admitted to the Jefferson Hospital, knowing that tobacco would be prohibited, went on a cigarette debauch the day preceding and acquired a marked acute laryngitis with considerable pain, which had been absent up to that time. In

spite of treatment, his larynx became rapidly worse and with great dysphagia, which was uncontrollable. Undoubtedly this would not have occurred had he refrained from irritating his larynx by constantly inhaling cigarette smoke.

The food should be of the blandest type. Upon the advent of hoarseness, the need of absolute rest of the voice should be impressed upon the patient. Cough, which is always excessive, should be allayed by sedatives. A great deal of the cough is unproductive, and consequently, unnecessary, and only aggravates the laryngeal lesion. Cocaine, perhaps, is the oldest and most widely used of the local applications for dysphagia. It very often relieves the pain temporarily, but has several disadvantages. In the first place it must be used frequently and only by the physician, directly to the larynx, and unless he is skilled in laryngeal work it is very likely to be ineffectual, and it must be increased in strength as its use is continued. Both morphine and cocaine tend to disturb the alimentary tract, which is already taxed to its utmost. Appetite, which is of much importance in tuberculosis, is greatly lessened by these two drugs.

A solution of camphor and menthol in liquid albolene as a spray to the throat gives some relief and is convenient, inasmuch as it can be used by the patient whenever necessary. It has a somewhat anesthetic effect and is cooling to the throat.

Insufflation of orthoform, analgesin, and anesthesin powders give relief for as long as twenty-four hours in the hands of some men, and can be used by auto-insufflation. Cracked ice, either sucked or applied externally, has its value. The various applications, formalin, lactic acid, argyrol, and more recently used chaulmoogra oil, tend to heal the ulcers and promote healthy granulations, thereby preventing or relieving dysphagia.

In dysphagia due to rigidity of the larynx causing aspiration of food and not due to pain, the Wolfenden or "horse position" very often allows the taking of fluids or semisolids with decreased pain.

With the patient in the upright position, gravity is added to muscular effort in the act of swallowing and in the event of an open, rigid larynx, some of the food will go the "wrong way" producing distressing cough. In the Wolfenden or "horse position" the patient's head is lower than the shoulders, consequently, the act of swallowing causes the food to pass uphill and is accomplished by muscular action of the throat and esophagus only. Gravity is eliminated and food does not drop into the larynx.

**Wolfenden Position.**—The patient lies prone on the bed or couch with the head, neck, and shoulders extending over the edge. Liquid food sufficiently thin to pass through a glass tube is placed in a vessel upon the floor. The patient lies with the head and shoulders below the level of the couch and sucks the food up through a glass tube.

**Insufflation and Auto-insufflation.**—The object of insufflation is to deposit a thin layer of an antiseptic or anesthetic powder over the surface of the larynx, which may be accomplished in two ways: First, by direct insufflation, the physician puffs a certain amount of powder through the proper instrument (Fig. 463). Second, by auto-insufflation, the patient himself operates the instrument.

**Technic of Insufflation.**—Load the insufflator by filling the reservoir in the same manner as filling a gelatine capsule. Replace the capsule

into the sleeve and force the powder into the barrel by pushing on the plunger. The curved end is placed at the back of the throat with the patient holding the tongue (it is not necessary to use a mirror). The patient is then directed to exhale to full limit and as soon as he inhales, sudden pressure on the bulb drives the powder the full length of the tube and into the larynx.

Auto-insufflation is carried out by loading the tube in the same manner, without the rubber bulb. The patient, holding the tube with his index-finger firmly pressed over the bulb end of the tube, places the curved end in the back of the throat after exhaling. He then closes his lips tightly around the tube and takes a deep inspiration and suddenly removes his finger from the tube. Cough occurs only occasionally after inspiration of powder and is then only of short duration. The powder most commonly used is orthoform. With the exception of the addition of small quantities of cocaine, orthoform is all that is necessary to produce partial or complete relief from dysphagia. I have never found any benefit in the use of powders in the tuberculous larynx excepting as anesthetics or analgesics.

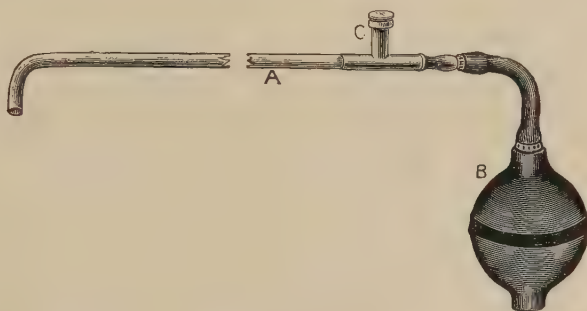


Fig. 463.—Laryngeal powder insufflator. This instrument is used for powder insufflations in the palliative treatment of laryngeal tuberculosis. It consists of a powder reservoir (C), a glass barrel (A), and a rubber bulb (B). It is loaded by means of the reservoir (C), and the powder is projected into the larynx during deep inspiration.

**Nerve-blocking.**—Blocking of the internal branch of the superior laryngeal nerve is perhaps one of the most efficient methods of controlling painful dysphagia. In some cases it fails of its purpose. These failures are due chiefly to faulty technic, or to the fact that the tuberculous process causing the dysphagia is extralaryngeal. This method was originated by Hoffman in 1908.

**Technic of Nerve-blocking.**—Thorough asepsis should be used throughout as regards the operator's hands, instruments, and skin of the patient's neck. The instruments and solution required are a Luer or Record syringe, tincture of iodine, and the fluid for injection, which is essentially a 65 to 85 per cent. alcohol. A glass hypodermic syringe may be used, with a  $1\frac{1}{2}$ -inch, twenty-four gauge needle with a sharp point. I use the following formula for nerve-blocking:

R.	Procaine.....	gr. ij.....	13
	Chloroform.....	℥x.....	66
	Alcohol.....	f3vj.....	24
	Aquæ dest.....	f3ij.....	8
M.			

The patient may either lie on the back with a sand pillow under the shoulders, or he may sit upright. The upright position is the most convenient both to the operator and to the patient. Male patients should have any beard removed from the site of the injection. The side of the neck should be painted with iodine. Palpate with the index-finger for the thyrohyoid space on the side to be injected. Locate the superior cornu of the thyroid cartilage and the greater cornu of the hyoid bone.

Select a point 0.5 cm. anterior and midway between these two horns with the index-finger of the hand on the opposite side to that of the patient. If it is to be on the left side, the right hand of the operator is used, and if the right side, the left index-finger. With this same index-finger just anterior to the artery, there will be no danger of injuring the carotid artery, the jugular vein, nor the vagus nerve because the vein and nerve lie behind the artery. If the left side is to be injected, keeping the index-finger of the right hand in position, the syringe point is plunged through the skin

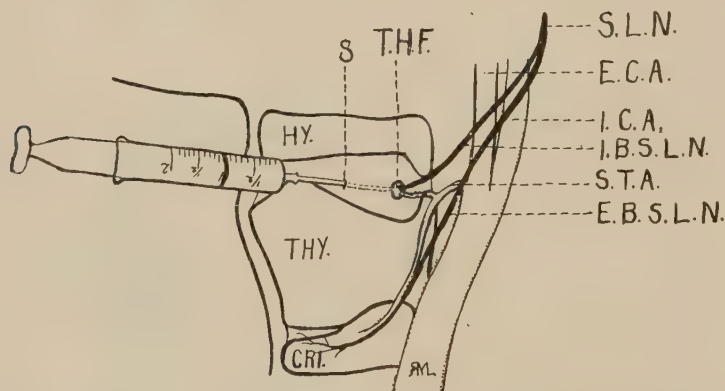


Fig. 464.—Schematic sketch of the relations of the internal branch of the superior laryngeal nerve: *HY.*, hyoid bone; *THY.*, thyroid cartilage; *CRI.*, cricoid cartilage; *S*, point of entrance of needle through the skin; *T.H.F.*, thyrohyoid foramen; *S.L.N.*, superior laryngeal nerve; *E.C.A.*, external carotid artery; *I.C.A.*, internal carotid artery; *I.B.S.L.N.*, internal branch superior laryngeal nerve; *S.T.A.*, superior thyroid artery; *E.B.S.L.N.*, external branch superior laryngeal nerve. The dotted outline between *S* and *T.H.F.* indicates the portion of the hypodermic needle beneath the skin extending from the skin puncture to the thyrohyoid foramen.

into the thyrohyoid space to a distance of about  $\frac{1}{2}$  to 1 cm. Revolve the point until a sharp pain is felt in the ear. This indicates that the internal branch of the superior laryngeal nerve has been touched. A few drops of the solution is injected. If there is no cough, then inject the remainder of the solution. If the patient coughs, it indicates that the needle point has penetrated into the larynx. This causes no harm, but the needle point should be withdrawn a very short distance and the injection continued, using  $\frac{1}{2}$  to 1 c.c. of solution. The right nerve is blocked in a similar manner excepting that the hands of the operator are reversed, the right hand using the syringe and the left index-finger guarding the great vessels. A good plan before nerve-blocking is to have the patient try to drink a glass of water and then immediately after the operation drink another glassful, so that the difference in swallowing may be noted. If it is impossible to obtain the pain reflex which sometimes happens after repeated nerve-blockings, in-

ject at the location of the nerve. These injections may be repeated any number of times. The relief lasts from twenty-four hours to several weeks. There are no known contraindications to nerve-blocking of the superior laryngeal nerve except in those cases in which the patient is comatose or moribund.

**Pain in the Throat Referred to the Ear.**—Intense swelling or extensive deep ulcerations of the arytenoids frequently cause excruciating pain in the throat which is referred to one or both ears. This type of pain is extremely difficult to abolish or even to relieve and is an ominous sign.

**Treatment for Ear Pain of Laryngeal Origin.**—Earache produced by tuberculous ulceration of the larynx can be controlled by cocainization of the nasal ganglion. Dr. Greenfield Sluder obtained such remarkable results in this condition following tonsillectomy by cocainizing the sphenopalatine ganglion, that Dr. Louis H. Clerf, at the suggestion of Dr. Chevalier Jackson, employed the Sluder method to control earache in laryngeal cancer with the same excellent results. The writer has used this method in 3 cases due to tuberculous ulceration. In all cases the earache was abolished for varying periods of time.

**Technic for Cocainizing the Sphenopalatine Ganglion.**—The nasal mucosa is first cleansed. A cotton-tipped applicator saturated with 5 per cent. cocaine solution is inserted into the nose back of the posterior end of the middle turbinate along a line extending from the floor of the vestibule of the nose to the posterior end of the middle turbinate. It is left in position for five or ten minutes. The otalgia is abolished for several hours to three or four days.

**Summary.**—Treating the larynx without due consideration of the pulmonary lesion is like bailing out a leaky boat and neglecting to plug up the leak. However, if we consider tuberculosis of the larynx as a complication and not as a disease entity, and fit the treatment to the individual case (no one method of treatment is specific) with due regard to the patient's general condition, the results of treatment will be vastly improved. Vocal rest is the best single treatment and should be combined with all treatments.

In conclusion, the writer would say that in his opinion the best medical treatment for tuberculosis of the larynx is vocal rest combined with local chaulmoogra oil treatment, either by instillation or direct application.

ROBERT M. LUKENS.

## AGRANULOCYTOSIS OF THE LARYNX

The larynx is involved in a small proportion of the cases of agranulocytosis; the laryngeal region is overshadowed by the necrotic ulceration elsewhere, especially on the tonsils, velum, and pharynx; by the serious general condition of the patient, the blood picture, and the usually fatal termination. Examination of the blood shows a progressively increasing loss of leukocytes and an even greater loss of granulocytes. No treatment has any influence on the local lesion or on the blood-making dyscrasia.

CHEVALIER JACKSON.

## BLASTOMYCOSIS OF THE LARYNX\*

**Definition.**—An inflammatory disease due to infection with blastomycotic organisms.

**Incidence.**—It seems certain that many cases are overlooked; therefore no accurate estimate of incidence is possible. It is much more common in Peru than elsewhere (Ernesto Raffo<sup>5</sup>), though this may be due to the more frequent recognition of the disease in Peru.

**Etiology.**—The specific cause is the mycotic organism, *Cryptococcus Gilchristi*. The infrequency of the disease taken in connection with the almost constant exposure of persons in certain occupations seems to point to the existence of very important predisposing factors in the few who acquire the disease. Exposure is fairly clear in the histories of some cases. One gave a clear history of having cleaned out old wood from the bottom of a grain elevator a few days previous to the onset of the initial symptoms. Another patient was probably exposed almost constantly to fermenting starchy foods in his occupation as poultry huckster. Occupations such as that of the old-time miller must have involved frequent exposure and possibly "miller's consumption" may have been a pulmonary blastomycosis. All patients so far reported in the United States have been adult males.

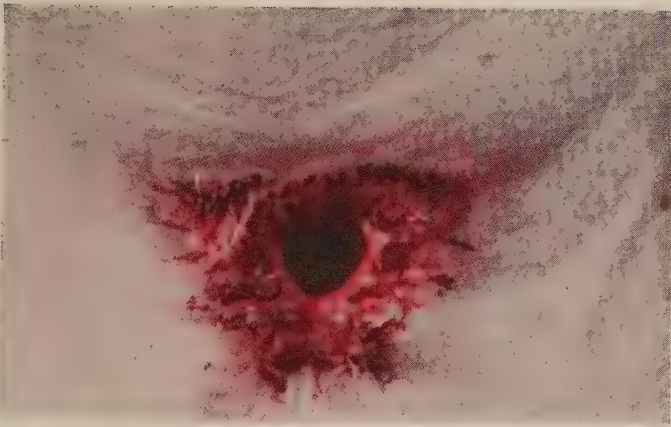
**Pathology.**—Blastomycosis limited to the larynx is exceedingly rare, though possibly failure to make the diagnosis may make it seem rarer than it really is. The cases reported, however, with one exception show associated involvement of the pharynx, tonsils, trachea, and lungs. The skin was involved only in one patient, and this was a secondary extension after tracheotomy. Leukocytosis was evident in one patient, the count being 19,000. This was a patient in whom the pulmonary involvement was fatal. Minute abscesses are characteristic of the blastomycotic lesions. In the larynx, those of the abscesses that are superficial rupture on the surface, leaving an ulcerated spot among other as yet unruptured nodules.

**Histology.**—The histological picture is suggestive of tuberculosis. Inflammatory infiltration, proliferation of the epithelium, giant-cells, and minute abscesses are present. The characteristic feature is the presence of spherical, encapsulated, parasitic cells. They can be demonstrated in the tissues on examination of unstained sections. Mycelia have not been found in the tissues.

**Bacteriology.**—The sputum is scanty unless the involvement of the lung is extensive, but the blastomycetes have been demonstrated in smears of sputum or secretions in all cases. Mycelia have been demonstrated in the sputum. Sartory, Petges, and Claoué<sup>4</sup> believed the organism found in the laryngeal cases to be a new one, to which they gave the name *Cryptococcus laryngitidis*. They describe the organisms as spherical or slightly ovoid, from 4 to 8 microns in diameter. In some instances the contents were homogeneous, in others a vacuole with a brilliant granule was noted. These investigators cultured the organism on carrots, potatoes, bouillon, gelatine, agar, and Roulin's fluid. Carrots gave a luxuriant growth, white, glossy, and moist, soon extending over the entire surface. The best temperature was found to be from 28° to 32° C. Subdermal and

\* The facts in this article were taken from a paper by Chevalier Jackson, read before the American Laryngological Association, May, 1925. The paper was published in the Proceedings and also in the Archives of Oto-Laryngology.<sup>1</sup>

PLATE XI



The upper illustration shows a mirror view of the stenotic laryngeal lesion in a case of primary blastomycosis of the larynx. (From an oil color drawing by Chevalier Jackson.)

The lower illustration is a tinted photograph of the front of the neck, showing the butterfly-shaped dermal lesion, the result of extension by continuity from the larynx after tracheotomy performed for blastomycotic laryngeal stenosis.



peritoneal inoculations in the guinea-pig produced a local lesion, and the two pigs peritoneally injected died in thirty-eight and forty-two days, respectively, with pulmonary lesions in which the parasite was found. Other observers found guinea-pigs refractory to inoculation. Dr. Crowell reported that growth on Sabouraud's medium, on glycerine gelatine and in maltose broth gave an appearance similar in all respects to *Cryptococcus dermatitidis* (Gilchrist and Stokes, 1898). As observed by C. J. Bucher in the author's case the growth was slow in starting, but afterward developed rapidly (Fig. 465).

**Symptomatology.**—The initial symptoms are hoarseness and cough; the latter may soon become paroxysmal and severe. The expectoration is slight unless there is lung involvement. It may be mucoid, mucopurulent, or purulent, and may contain membranous shreds. The history of onset is the usual one of a "cold that settled in the throat." A local soreness,



Fig. 465.—Luxuriant growth of blastomycotic organisms from laryngeal secretions obtained directly from the laryngeal lesion in a case of blastomycosis of the larynx. The growth was a long time in starting, but later grew rapidly.

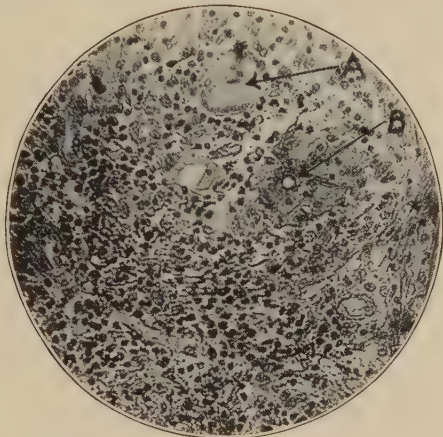


Fig. 466.—Photomicrograph showing the histology of the lesion in the case of blastomycosis primary in the larynx. The giant-cells (A, B) are suggestive of tuberculosis. In giant-cell B is shown the blastomyces, probably dead before the specimen was taken. No mycelia were found in the tissues. (Histologic report by B. C. Crowell, author's case.)

pricking or jagging may be noted. Dyspnea supervenes as the stenosis increases. Dysphagia may develop. Progressive loss of weight and strength, and slight intermittent rises of temperature have been noted in all cases, but are probably due, in part at least, to the accompanying pulmonary lesion. Dermal blastomycosis may be present (Plate XI).

**Laryngoscopic Appearances.**—The larynx as seen in the mirror is intensely inflammatory, with grayish, minutely nodular surface in some portions of the lesion, and there may be a few minute, isolated yellowish nodules. In some cases an appearance like that seen after an application of nitrate of silver was noted, and some of this membrane came away on coughing, leaving a red, inflammatory, eroded and, in small spots, ulcerated surface. The epiglottis may be involved, and there may be ulceration on the free margin; the lesion may extend into the tongue. The cords were nodular in one patient. Glottic stenosis as seen in the later stages may require tracheotomy. In considering laryngoscopic appearances, it may

be well to remember that the stage of the lesion, or part of the lesion, is important. In the early stages it may be flat; the nodules with yellow or grayish points are minute, superficial abscesses. When the abscesses rupture, the lesion flattens at that point. Exudate may cover considerable areas of the lesion. I have noted an opaline "frog-spawn" appearance of the tiny nodules which is quite unlike anything else I have ever seen in the larynx (Plate XI). The mucosa that was not covered with grayish nodules was deep crimson, the intense redness being in marked contrast to the pallor so often seen in the pale type of laryngeal tuberculosis.

**Diagnosis.**—The diagnosis can be made with certainty only by biopsy, and bacteriological examinations. Dr. Crowell points out the diagnostically important fact that the blastomycotic organisms are not readily seen in stained sections, hence are likely to be overlooked unless unstained specimens are searched. With pulmonary blastomycosis in addition to the laryngeal blastomycosis the whole clinical picture is that of tuberculosis. Biopsy showing the very obvious giant-cells (Fig. 466), may support this erroneous diagnosis. The intensely red and inflammatory appearance of the larynx as seen in the mirror is not typical of laryngeal tuberculosis and is not particularly suggestive of cancer. But appearances in the mirror should not lead anyone to neglect biopsy, which is the final arbiter in ulcerative laryngeal lesions.

**Prognosis.**—If untreated, probably all patients die from extension of the disease to the lungs or other tissues. If the diagnosis be made early there is good reason to expect favorable results from treatment.

**Treatment.**—The unfavorable results from surgical treatment of blastomycosis of the skin lead one to expect little from surgical treatment of laryngeal blastomycosis. The extension along the tracheotomic fistula to the skin seems to furnish support for this view (Plate XI). The good results obtained by the dermatologists with potassium iodide in blastomycotic dermal lesions point to treatment by this drug as the method of choice. The selective effect of potassium iodide on the laryngeal mucosa strengthens the indication for its use.

CHEVALIER JACKSON and CHEVALIER L. JACKSON.

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#### BENIGN TUMORS OF THE LARYNX

**Definition.**—Clinically, a benign growth is one that does not infiltrate normal tissues. It may displace normal tissues in a very aggressive way and it may cause the death of the patient; but its cells lack the infiltrative quality, whatever it may be, that characterizes malignant growths. A

benign growth may be accompanied by similar growths at more or less remote locations; but these are not metastases in the sense in which the word is used in malignant disease. A benign growth may be a true neoplasm, or it may be a cystic retention of normal secretions, or a retention of extravasated blood as in hematoma, or a group of dilated blood-vessels or dilated lymph spaces, or it may be of inflammatory origin as in the case of granulomata, edematous polypi, and papillomata. Tuberculoma and syphiloma are not usually included as tumors, though they require exclusion before a diagnosis of benign growth can be established.

In this section, for the reader's convenience, the author will also consider briefly a few conditions which, though not neoplastic, require, in some cases, exclusion of new-growth for their diagnosis.

**Symptomatology.**—*Hoarseness, dysphonia, cough, croupiness, a desire to clear the throat, stridorous breathing, and dyspnea* are the chief symptoms. A benign growth may be present in certain locations in the larynx for a long time without marked symptoms. For clear phonation the cords must (a) approximate, (b) draw tense, and (c) vibrate. Any growth that interferes with these mechanical movements will cause impairment of the voice. The impairment may be an alteration of pitch, higher or lower, constant or intermittent. The voice may be reduced to a whisper; but if any air at all can be forced in or out through the larynx the whispering voice is never lost. When complete occlusion occurs and tracheotomy is necessitated to prevent asphyxia, the whispered voice is lost; not because of the tracheotomy but because no air goes through the larynx. If occlusion of the larynx is not complete the patient can whisper by occluding the cannula with the finger. Children with the larynx occluded with papillomata usually acquire, spontaneously, a buccal voice such as a laryngectomized patient does. In the period before this form of voice is acquired the child may be regarded by the parents and even physicians as a mute or mentally defective child. Dyspnea may be inspiratory or expiratory and of all degrees; it is first noticeable on exertion, then during sleep, and finally it is constant. It may be accompanied by stridor and wheezing; cyanosis is seen at a very late stage; an ashy-gray color is more often seen than blueness, until the final stage of asphyxia is reached, when the patient may be "black in the face." Early diagnosis and treatment will forestall these serious symptoms.<sup>7</sup>

**Diagnosis.**—The symptoms enumerated in the foregoing section are all to be taken as indications for laryngoscopy (direct in children, with the mirror in adults) rather than as diagnostic points. Inferential diagnosis of laryngeal disease is dangerously misleading and should be condemned. The larynx of any human being whose mouth can be opened can be examined. In adults and in children over six years of age there are very few patients whose larynx cannot be seen with the mirror; in some patients it is easy, in other individuals it requires great patience and many trials. In very young children and in a few adults the larynx can be seen only by direct laryngoscopy (*q. v.*). This simple method of diagnosis, giving all the certainty of direct inspection with the eye, can and should be used, without any anesthesia, general or local, in children, and with only local anesthesia in adults. It requires at most only a few minutes, often less than one minute, including the time necessary to remove a tissue or swab specimen, or even the entire growth itself, in many instances. This state-

ment, however, applies only to direct laryngoscopy in trained hands, with trained assistants, and carried out as hereinafter described. But attempts by the untaught, however otherwise skilful, to perform direct laryngoscopy, especially on a child in the sitting position, will inevitably result in failure and possibly condemnation of one of the greatest technical advances in laryngology since the discovery of the mirror method of laryngoscopy.

A frequently made erroneous diagnosis is *asthma*, because of the wheezing respiration and stridor, worse at night. Inspection of the larynx at once shows the tumor as the cause of the wheezing and stridor. A child with long continued laryngeal obstruction, as by a benign tumor, soon learns that he gets more air by quiet shallow breathing. This has led to an erroneous diagnosis of *paralysis of the diaphragm*, a diagnosis that is promptly negatived by noting the indrawing at the suprasternal notch and at the diaphragm, as was done in two instances by one of our secretaries at the Bronchoscopic Clinic. *Diphtheria* has been mistakenly diagnosed and antitoxin given in quite a number of cases of benign tumor in children sent to the Bronchoscopic Clinic. The symptoms had been of long duration, but an acute intercurrent laryngitis had elevated the temperature and exaggerated a croupy cough.<sup>7</sup>

*Biopsy.*—Taking a specimen of tissue for histologic examination is the final arbiter in the diagnosis of benign growths of the larynx. The essential difference between a benign and a malignant process is in the behavior of the deep cells. There is no way to determine positively what these deep cells are doing early in the disease, without taking a mass of them out along with some of the normal base to see if the latter is being invaded. Radical removal of normal basic tissues is, however, contraindicated in benign growths; very little of the normal is needed for histologic examination. In cases of small growths and those in which there is nothing in the appearance to support a diagnosis of cancer, it is better to remove the entire growth rather than a mere specimen. In the case of ulcerated large tumors the specimen should include the edge of the ulcer. Tuberculosis should be excluded by the usual methods, always including Roentgen-ray examination before deciding to remove a specimen; and syphilis should be excluded by a Bordet-Wassermann test. Presence of either syphilis or tuberculosis does not prove that the laryngeal lesion is a *syphiloma* or a *tuberculoma*, but it usually calls for postponement of the biopsy except in case of clear indications to the contrary. The technic and a further discussion of this subject will be found under Direct Laryngoscopy.

*Prognosis.*—If asphyxia be avoided by early diagnosis and treatment there is little or no danger to life from benign tumors of the larynx. Recurrences after complete removal are rare except in cases of papillomata. In adults benign growths, especially papillomata, may be succeeded by a malignant growth. We have seen at the Bronchoscopic Clinic a number of cases in which the epithelial cells after repullulating luxuriantly on the surface at last broke through the basal barrier and invaded the submucosa, becoming typically carcinomatous.<sup>1,2</sup> As shown by Semon there is no evidence to indicate that a change from benign to malignant qualities is induced by partial removal or local treatment of a benign laryngeal growth. There are certain kinds of limited overgrowth of epithelium, such as keratosis and leukoplakia, that while not strictly neoplasms require excision to

prevent the development of cancer. They are essentially *precancerous*, however faulty such a word may be from the strictly scientific and histologic point of view.<sup>1</sup> The prognosis grows less favorable the longer they are allowed to remain.

**Treatment.**—There may be an occasional case in which a small, benign laryngeal growth that is producing no symptoms may be allowed to remain, as for instance when a patient has serious organic disease elsewhere. In the absence of some such contraindication, benign growths, except lymphomata, should be completely removed by direct laryngoscopy; but radical removal of the base is strongly contraindicated, as mentioned in the consideration of papillomata. Radical removal, may ruin the quality of the voice, impair the expectorative and protective functions of the larynx, and cause stenosis; whereas the best results as to vocal and other functions of the larynx, with equal chances of freedom from recurrence, are had by superficial removal repeated if necessary. Great care is necessary to avoid cutting into the crico-arytenoid joint, which is quite superficial. Nipping off the tip of the vocal process of the arytenoid cartilage may result in necrosis, recurrent fungations, or contact ulcer (*q. v.*). Applications of silver nitrate to, or cauterization of, the basal tissues after removal of a benign laryngeal growth are quite unnecessary, and needlessly incur risk of damage with no effect as to recurrences. Pulling on the tough fibrous attachment of a pedunculated fibroma may damage the laryngeal structures. Stripping off a layer of vocal cord is to be avoided by reversing the direction of the pull and by properly sharpened cupped-forceps. The direct method of removal is preferred except by a few of the older group of laryngologists who have had unlimited practice in the use of the MacKenzie forceps guided by the reversed image in the mirror. With the direct laryngoscope in the left hand, the forceps in the right, a co-ordinate manipulation under direct unreversed control of the eye gives a precision that is invaluable. The further discussion of this subject and the operative technic will be found in the chapter on Direct Laryngoscopy. Radium is better than endolaryngeal surgery for *lymphoma* of the larynx. These growths fade away in a most satisfactory, almost magical way without ulceration under external applications of radium. The use of radium for this kind of growth in the larynx was first demonstrated by Delavan. Careful technic as to screening and dosage is essential. Perichondritis, necrosis, stenosis, and ruin of the larynx are sure to follow overdosage. These might not matter much in cancer, but they are avoidable disasters in benign growths.<sup>7</sup>

#### VARIETIES OF BENIGN GROWTHS OF THE LARYNX

**Angioma** is a relatively frequent form of tumor. I have seen 227 cases in forty years. The most frequent site is the vocal cord; the tumor is usually in the form of a small, smooth, globular, bright-red, shining nodule, 2 or 3 mm. in diameter. Sometimes it is pedunculated. Often there are dilated capillaries visible on both the cords, sometimes only on the cord bearing the tumor. This type of angioma is most often seen in those who abuse their voices occasionally but not continuously. I have seen these growths follow a hematoma due to shouting, as at football games and the like. They are benign and have very little tendency to recur after removal if vocal abuse be stopped. In a few instances a new angioma appeared on

the other cord or in a new location on the same cord. The small type of angioma occurring in adult life is often a *fibro-angioma*. There is another and larger type of angioma more nearly resembling the angiomas seen elsewhere, that is rare and may have a congenital basis. It may involve a whole cord or even fill almost the entire larynx. Both kinds of angiomas can be removed, using cupped forceps, and scalping the small growths off leaves the cord with a perfectly normal appearance. These small growths need cause no anxiety as to postoperative hemorrhage. The very large type might require tracheotomy and packing of the larynx, but I have not seen such a case.<sup>7</sup>

**Hematoma** may be simply a blood collection beneath the mucosa due to external trauma, as from a fall across a bicycle handle-bar or the edge of a step. There is often no sign of trauma on the skin surface. Small hemorrhages under the epithelium may occur on the cord from ruptured capillaries in shouting, or even less violent use of the voice. Usually complete disappearance follows absorption of the clot. Occasionally a small elevation remains and sometimes an angiod growth follows.

**Fibroma** is usually of the soft type, but occasionally a tough pedicle is encountered at removal. The most frequent point of attachment for these tumors is one of the vocal cords, but they may spring from anywhere. They are usually small, but may be large enough to occlude the larynx. They should be removed by direct laryngoscopy. If they are simple fibromata, that will usually end the matter. If, however, after removal the histologic structure should show evidence of malignant change, such as *fibrosarcoma* or *neurofibroma*, laryngofissure may be necessary for adequate removal of a sufficient area of normal basal tissue.<sup>7</sup>

**Lymphangioma** is not very common. It occurs on the ventricular bands as a result of angioneurotic edema. In this region it is rather diffuse and does not call for removal. The dilated lymph-spaces may be contracted by galvanocautery applications, but extreme nicety of puncture with a small point is needed to prevent disaster.

**Myoma** is uncommon. More frequently we see *fibromyxomata* and *myxosarcomata* (*q. v.*), or *myxochondromata*. They usually require direct laryngoscopic removal with forceps, except the malignant mixed tumor types which are dealt with as malignant disease of the larynx (*q. v.*). Myxomata are true neoplasms; edematous polypi are not.

**Lymphoma** in the larynx is usually benign, and does not recur after removal. They may, however, be associated with a general lymphomatosis, when the prognosis becomes more serious. They should not be operated upon because of the wonderful results obtainable by the external application of radium as above mentioned. Great care must be taken to avoid injury to the parathyroids and to the laryngeal cartilages. Perichondritis, necrosis, stenosis, and death may result from overdosage. Pernicious anemia may follow radiation in general lymphomatosis.

**Lymphosarcoma** is rare, and if limited to the larynx should be dealt with as other malignant growths (*q. v.*). If associated with a general lymphosarcomatosis the laryngeal lesion may of course be regarded as a minor element in a fatal disease.

**Lipoma** is a fatty tumor, rather uncommon in the larynx. I have seen 7 cases in forty years. All the patients got well after removal of the growth, but in 1 case three removals were necessary and in another case a

second removal was required. They may be large or small; but are not narrowly pedunculated. They may occur at any age.

**Chondroma** usually occurs as *myxochondroma*, or as *myxochondro-osteoma*, sometimes as *chondrosarcomata* (q. v.). These forms are infiltrative, hence malignant lesions, but they show little or no tendency to metastasize. If small, they may be removed endoscopically; if large, external operation may be required. In 6 cases I have found it necessary to advise total laryngectomy for chondroma because of the massive involvement including both the cricoid and thyroid cartilages which obliterated the larynx (Fig. 467). Fixation of one arytenoid had been observed in this patient 4 years previously. It was mistaken for paralysis. In one case in which consent to operation was unobtainable the patient died of sarcomatous degeneration and metastases. Tracheotomy is usually necessary as a palliative measure, if radical operation is not done.

**Osteoma** is usually of mixed type and is semimalignant in its clinical features. If small they can be removed endoscopically, but malignant growths are better dealt with by external operation.

**Tophi** are small collections of uric acid crystals under the mucosa. I have seen only 1 case; whitish nodules on the epiglottis were removed for diagnosis in a patient supposed to have tuberculosis. They are probably more numerous in other countries than in the United States, but they probably do not require removal.

**Cysts** may be of the glandular or connective-tissue types (Plate XII). In both the sac should be entirely removed by direct laryngoscopy, whether the growth be large or small. If this is done they will not recur. I have seen 35 cases, of which 23 sprang from the epiglottis. If neglected, cysts usually increase in size and in many cases endanger life by laryngeal obstruction, and asphyxia may supervene.

**Amyloid Tumor.**—This is a very rare condition in our experience. We have seen only 1 case at the Bronchoscopic Clinic. The tumor was a fibroma that had undergone amyloid degeneration. Unlike amyloid tumors in general, it occurred in an apparently healthy individual. It did not recur after removal.

**Thyroid tumors** are islands of thyroid tissue. They are rare in the larynx; less rare in the trachea. I have seen 11 cases. In 8 the growth was pedunculated, in 1 it was connected through the tracheal wall with a goiter externally. In 3 of the cases the mucosa covering the tumors was normal in appearance, in 4 it was very dark in color. A nodular appearance of the surface was noted in 1 case. In 2 of the cases the thyroid tumor was malignant. One of these was externally removed by Dr. Hartley Anderson with perfect recovery; the other was arrested by Roentgen-ray treatment by Dr. I. Seth Hirsch.

**Edematous Polypi.**—These are not true neoplasms, but clinically they are tumors requiring removal. This should be done superficially with cupped forceps, the growth being scalped off level with the surface. There



Fig. 467.—Pencil sketch of a chondroma in a man aged forty-four years, as seen in the mirror. The entire larynx was obliterated by a huge mass of chondromatous tissue that included the cricoid and thyroid cartilages. The entire pharynx was occluded by the growth; tracheotomy had been done to prevent asphyxia. Laryngectomy was required.

is little tendency to recurrence if the always-associated chronic laryngitis and vocal abuse are stopped.<sup>7</sup>

**Neurofibroma.**—This is a very rare form of growth. The only case of this kind that we have seen was in a woman twenty-three years of age, who had had a carcinomatous goiter removed before by Dr. Stuart McGuire. It was not associated with visceral or dermal lesions of similar character. Because of the undoubtedly carcinomatous condition of the thyroid gland we all expected to find the laryngeal lesion carcinomatous also. Dr. Baxter L. Crawford, however, found that the histologic structure of a specimen removed by direct laryngoscopy showed a neurofibromatous growth with no sign of carcinomatous structure (Figs. 468–470). Direct laryngo-

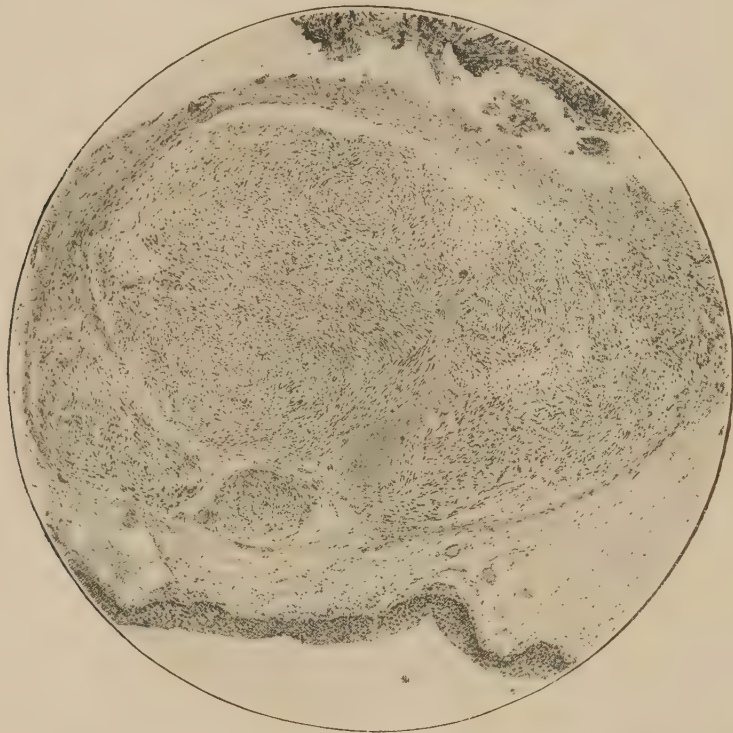


Fig. 468.—Photomicrograph of a histological section of a neurofibroma of the larynx in a woman aged twenty-four years. There was no histological evidence of cancer in the larynx (Chevalier Jackson's case, histologic diagnosis by B. L. Crawford).

scopic removal would have been easy, but was decided against as not indicated.

**Papilloma in Adults.**—Multiple papillomata differ from all other laryngeal benign growths in their strong tendency to repullulate not only at the site of removal, but at relatively remote locations in the larynx, fauces, pharynx, anterior nares, or elsewhere. They grow out from the surface by a great piling up of epithelium, but the epithelial cells do not infiltrate downward through the basal cells into the submucosa, and they show no tendency to metastasize in the accepted sense of that term. In a very small percentage of patients, after few or many years, the papillomata develop that malignant quality, whatever it may be, that enables the

epithelial cells to break through and invade the subepithelial tissues, and a cancer develops. This is very rare in the larynx compared to the bladder, where papillomata are regarded as almost always potentially malignant. Laryngeal papillomata should be treated as benign growths unless and until the removed specimens show malignant qualities. Superficial scalplings with the cupped forceps, as described under Direct Laryngoscopy, should be persisted in until recurrences and new growths in new locations cease, as they nearly always do. Desperation on the part of patient and laryngologist at the constant recurrences may lead to the trial of desperate measures, such as galvanocautery, fulguration, diathermy, laryngofissure, or radical applications of radium and the Roentgen ray. These afford

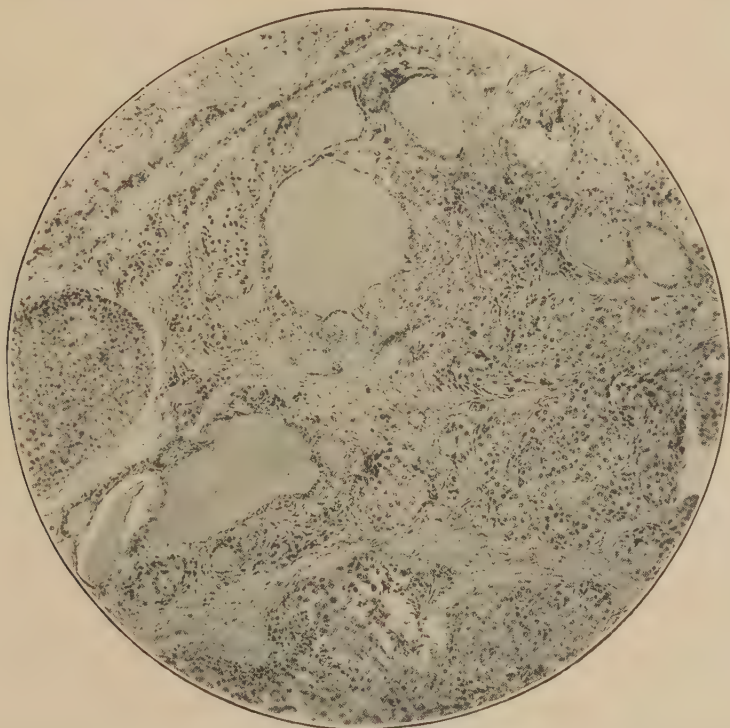


Fig. 469.—Photomicrograph of a slide showing cancer of the thyroid gland in the same patient referred to in Fig. 468 (concurrent neurofibroma and cancer).

no better results in prevention of recurrence than scalping, and are usually followed by total atresia of the larynx. In using radium and the Roentgen ray the slightest overdosage will end in disastrous perichondritis, necrosis, stenosis, or sloughing, with consequent ruin of the voice. On the other hand, scalping, when done with great care to avoid injury to the normal tissues, will end ultimately in freedom from recurrence, a perfect voice, and a larynx indistinguishable from normal. We have had over 100 such cures. It is impossible to say how much the scalplings had to do with the ultimate cessation of recurrence, but it is beyond question that our careful avoidance of injury to the normal tissues resulted in a normal larynx and an unimpaired voice.

When a laryngeal papilloma has become the site of a malignant growth

the whole situation changes at once. Radical measures are indicated, and laryngofissure is the method of choice if the lesion is intrinsic and of limited extent (see Malignant Disease of the Larynx).<sup>7</sup>

**Papilloma of the Larynx in Children.**—(See 4, Plate XII). The following résumé presented with an exhibition of patients a few years ago formulates the conclusions reached at the Bronchoscopic Clinic. Experience since has served to emphasize the correctness of the conclusions.<sup>7</sup>

1. Multiple recurrent laryngeal papillomata in children constitute a benign self-limited disease.

2. Those occasional cases that recover after a single operation are examples of a short limit; or the operation has been done near the end of the

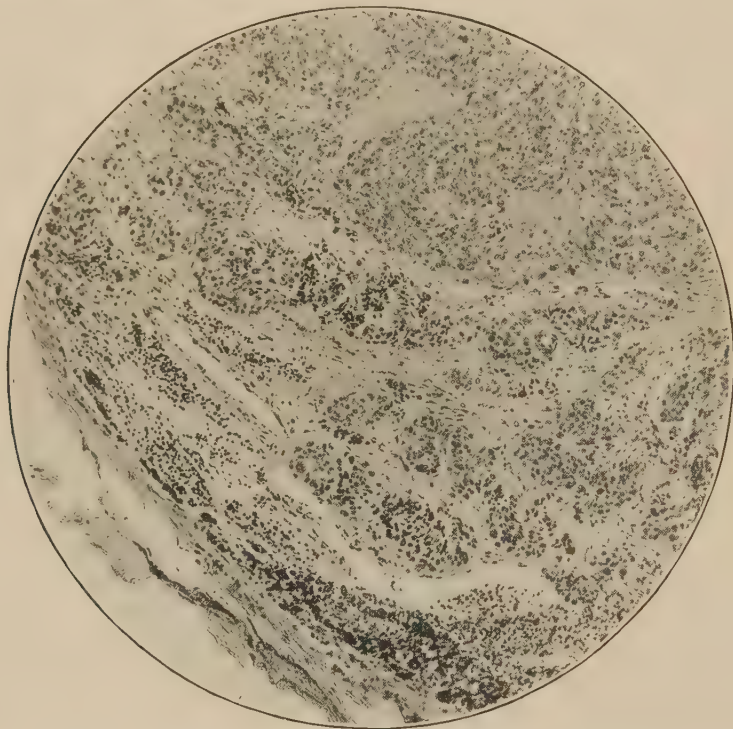


Fig. 470.—Photomicrograph of a histological section of a lymph-node on the same patient as shown in Figs. 468, 469. The concurrence of cancer of the thyroid with metastasis in the lymph-node along with a neurofibroma in the larynx is noteworthy.

time limit. The operator is likely to be misled into attributing a remarkable recovery to the particular method he happened to have used.

3. Papillomata repullulate on the surface; they do not infiltrate.

4. After removal of papillomata, no matter how radically, repullulations occur at the site of removal and new papillomata appear at new locations in the larynx, or even the pharynx, fauces, lips, and anterior nares.

5. Even if the larynx be eviscerated, the patient will get well no quicker than if the growths were superficially removed, and evisceration will have irremediably ruined the voice.

6. The methods employed in cancer are utterly out of place in cases of papilloma in children. Cancer is an infiltrating disease with no limits

PLATE XII



Direct and indirect laryngeal views from Chevalier Jackson's oil-color drawings from life. 1, Epiglottis of child as seen by direct laryngoscopy in the recumbent position. 2, Normal larynx spasmodically closed, as is usual on first exposure without anesthesia. 3, Same on inspiration. 4, Supraglottic papillomata as seen on direct laryngoscopy in a child of two years. 5, Cyst of the larynx in a child of four years, seen on direct laryngoscopy without anesthesia.



# PLATE XIII



Papillomata on the right side of the fauces of a child aged two years. These faucial growths appeared after the removal of recurrent laryngeal papillomata. Repullulation occurred at the site of removal and new papillomata appeared in the trachea. After thirty superficial removals from all locations all repullulations ceased and development of new growths ceased. The patient recovered an excellent voice with a larynx indistinguishable from normal.



except the life of the patient. Unless stopped it has 100 per cent. mortality. Multiple recurrent papillomata of children constitute a benign self-limited disease, usually ending in recovery if asphyxia is prevented by a timely tracheotomy. Scars, tissue destruction, vocal ruin, or radium burns are of no consequence in cancer; they are disasters in papilloma.

7. The best method of cure of multiple laryngeal papillomata is to scalp off the projecting parts of the growths, without any attempt at removal of the base, repeating the procedure as often as repullulating buds appear. The removal requires but a minute or two with the direct laryngoscope and proper forceps. No anesthetic, general or local, is necessary, and a sedative is superfluous. Even children two years of age will usually open their mouths and permit the procedure without protest, when they know by previous seances exactly what to expect.<sup>7</sup>

**Mycosis Fungoides.**—The multiple tumor formation on the skin in this disease is in a very small proportion of cases accompanied by small tumors on the mucosa of the larynx as well as on the mucosa of the air-passages above and below the larynx. In the reported cases the tumors were small, oval in shape, and ulcerated. As the laryngeal tumor formation is only a small part of the general disease the laryngologist's only duty is to see that tracheotomy is done if obstructive laryngeal dyspnea develops.

**Pachydermia laryngis** is not strictly speaking neoplastic, yet its inclusion here is justified by the fact that differentiation from neoplasms is in some cases a clinical necessity. So far as is at present known, it is the result of chronic hypertrophic laryngitis, and yet it is essentially different from the ordinary cases of that disease; and there are grounds for believing that it will eventually be established as a distinct disease. The posterior third or half of the interior of the larynx is covered with a whitish membrane-like covering that can be removed in strips. Histologically the stripped-off material does not resemble diphtheritic membrane; it is composed of piled-up masses of epithelium, some of which is becoming epidermoid. The whole interior of the larynx is chronically inflammatory, and it is hypertrophied, especially posteriorly. The symptoms are hoarseness and in some cases cough; rarely dyspnea on exertion. The diagnosis can be made with certainty only by the histologic examination of a specimen. The latter should be taken by direct laryngoscopy. The prognosis is good as to life in most cases and the patient will probably always have phonation though it will be rough and husky. Rarely the patient's life may be endangered by stenosis. Treatment in most cases is that of the accompanying laryngitis. Direct laryngoscopic removal of membrane or tissue may be indicated, and Fielding O. Lewis found it necessary to remove a whole larynx for this condition. In any case signs of obstructive laryngeal dyspnea should be watched for, so that tracheotomy may be done if necessary.

**Keratosis of the larynx** is not, strictly speaking, a tumor of the larynx, yet its inclusion here is justified by the fact that, clinically, it must be differentiated from new growths. It consists in a whitish patch or patches of hyperplastic epithelium, resembling *leukoplakia*, on the upper surface or edge of one or both vocal cords. Diagnosis can be made only by the histologic examination of a specimen removed by direct laryngoscopy. If it does not promptly disappear in a month or two under a rigid régime of vocal rest and general eliminative treatment it should be externally or endolaryngeally excised with a wide area of normal tissue, because keratoses

and similar overgrowths of epithelium are to be regarded clinically as potentially precancerous, however faulty such a term may be from a strictly scientific or histologic point of view.<sup>1</sup> Purists may argue that a growth is either cancer or not a cancer; but surgeons have found that, as a clinical fact, if certain kinds of growths are allowed to remain, both patient and surgeon will in a large percentage of cases have cause to regret it. As James Ewing, the pathologist, said of one such case, "This is the stage in which to cure cancer, not after it has become established."<sup>1</sup>

**Acromegaly of the Larynx.**—Overgrowth of the laryngeal tissues, if asymmetrical, may simulate a tumor of the larynx in certain cases of



Fig. 471.—Acromegaly of the larynx. The enlargement and thickening of the various prominences of the larynx is bilateral. The asymmetry seems rather a distortion, though at 4 is shown a larynx with elongation of one aryepiglottic fold associated with thickening of its fellow. The larynx shown at 3 was so suggestive of the typical acromegalic bands as to merit the appellation of "gingerbread larynx." The whole larynx by external palpation seemed symmetrically enlarged in all cases except the one shown at 2, in which the overgrowth was limited to the epiglottis.

acromegaly. In the first case encountered at the Bronchoscopic Clinic a diagnosis of laryngeal cancer had been made.<sup>4</sup> The chief symptoms are hoarseness, deepening of the pitch of the voice, and dyspnea on exertion. The diagnosis is usually made by the accompanying general manifestations and the associated overgrowth in the nose, fauces, uvula, or tonsils. If any doubt remains as to a particular region in the larynx a specimen can be taken by direct laryngoscopy. Prognosis is that of the general disease; but laryngeal stenosis may introduce special risks. Treatment is that of

PLATE XIV



The upper illustration shows a keratotic disease of the left vocal cord that seemed clinically so suspicious of a cancerous or precancerous condition as to warrant removal. The histological findings after the removal of the entire growth were not inconsistent with this clinical opinion.

The lower illustration shows that at laryngofissure the extension downward usually seen in malignancy was lacking, and the growth was rather less granular in appearance. Only a moderate removal was decided upon. At the end of five years the patient was still free from recurrence and had a fairly good voice. (Photoprocess reproduction of an oil-color drawing by Chevalier Jackson.)



the general malady, but laryngeal stenosis (Fig. 471) should be watched for, and the tracheotomy should be done early rather than late. As in so many other cases the final asphyxia comes on very suddenly.

CHEVALIER JACKSON and CHEVALIER L. JACKSON.

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## PROLAPSE OF THE LARYNGEAL VENTRICLE, AND EVERSION OF THE SACCULUS\*

Much confusion and ambiguity exist as to the nomenclature adopted in connection with the above subject. The term "prolapse of the ventricle" was introduced into laryngological terminology by Lefferts in 1876, and that of "eversion of the sacculus" by Moxon in 1868. Morell Mackenzie in 1880 (using the terms "ventricle" and "sacculus" synonymously) referred to eversion of one or both ventricles as an extremely rare form of intralaryngeal dislocation, and considered that it was the only doubtful source of error in the diagnosis of benign growths of the larynx. He stated that he knew of only 3 such cases in medical literature, 2 of these being recognized on postmortem (Moxon's and his own case), and a third (Lefferts') in which it was recognized during life.

The term "prolapse of the ventricle" is at present used universally for all manner of conditions. Mygind (Copenhagen), in 1901, expressed the opinion that "prolapse" was merely a localized hypertrophy of the mucous membrane which lies round the orifice of the ventricle, and that it is called "prolapse of the ventricle of Morgagni" because it was formerly supposed to be prolapse of the mucous membrane.

Garel (Lyons), in 1901, reviewed the literature and found that the laryngeal condition described as "eversion of the ventricle" was employed for very dissimilar lesions, owing, he says, to the fact that authors had based their conclusions only on cases observed by themselves.

\*Revised and brought up to date from an original paper read before the Section of Laryngology, Royal Society of Medicine, in 1921, and published in extenso in the *Journal of Laryngology and Otology* in 1922.

A Study of 85 cases—the total number recorded in the literature since the first case of the latter condition—was described by Moxon in 1868. (By kind permission of the Editors.)



Fig. 472.—Horizontal section of the normal larynx above the glottis, showing the relations of the sacculus. A black bristle has been passed through the right ventricle into the corresponding sacculus. (Dissection by Professor S. G. Shattock, F. R. S.)



Fig. 473.—Larynx with right ala of the thyroid cartilage removed, showing the external or thyroideal aspect of the right sacculus laryngis covered by its glands, and the relationship of the thyro-arytenoid muscle. (From a dissection by John Hilton.)

Moller (Copenhagen), in 1905, remarked that most authors consider the term does not cover the nature of the disease and that the majority of the cases described in the literature deal with something entirely different

from a prolapse. He referred to the term as having lost its meaning, and quotes Lussan (Paris) who says: "By prolapse of the ventricle of Morgagni, one understands a more or less big tumor of the larynx, red in color, and with a smooth surface, which seems to emanate from the ventricle."

Delsaux (Brussels), in 1905, remarked that since the publication of Fränkel's studies in 1894 unanimity has not been reached as to what is to be understood by "eversion and prolapse of the ventricles of Morgagni." In drawing attention to the rarity of the occurrence he showed a pathological specimen removed from a cured patient, and suggested that by "prolapse"



Fig. 474.—Section through the right side of the thyroid and cricoid cartilages showing the left ventricle, the compressor sacculi laryngis (aryteno-epiglottideus inferior) muscle, and a portion of the inner wall of the sacculus exposed by removal of the mucous membrane. Note the weak triangular area below the lower fibers of the aryteno-epiglottideus muscle, through which a hernia of the sacculus might protrude. (From a dissection by John Hilton.)

was understood a hernia of the mucous membrane through the natural orifice of the ventricle.

**Definition of Terms.**—The *laryngeal ventricle* is a horizontal space, elliptical in shape, which is situated between the true and false vocal cords, from the roof of which, toward the anterior part, there arises a vertical pouch known by anatomists as the sacculus.

The *sacculus laryngis* or, more correctly speaking, the sacculus ventriculi laryngis, lies between the ventricular band and the inner surface of the thyroid cartilage (Figs. 472-475). It extends directly upward from the anterior part of the ventricle, and represents the cervical pouches or air-sacs which in anthropoids are connected with the ventricles, and in some cases extend over the front of the neck and chest, even as far as the axillæ.

By "prolapse of the ventricle," the term in general use, is to be understood (as a critical examination of the different cases shows) the protrusion of a portion of the ventricular mucosa as a result of inflammatory edema or hyperplasia, so that parts of the ventricle normally out of sight are carried inwards along with the swelling, and come to be brought into view of the laryngoscope.

By the term "eversion of the ventricle" should be understood an inward displacement or invagination of the mucosa of the ventricle, so that

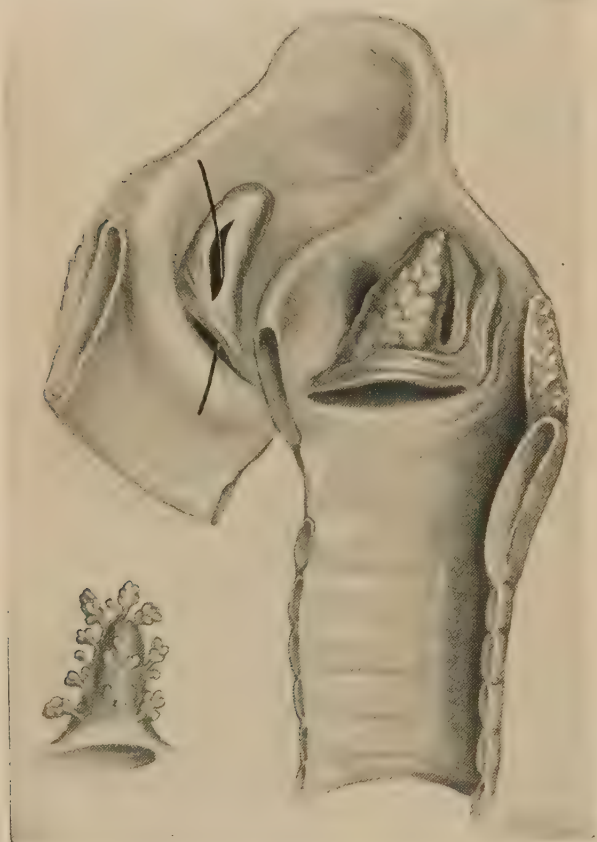


Fig. 475.—View of the interior of the larynx showing the sacculus laryngis on the right side exposed by removal of the mucosa with its glands resting upon it, and on the left side cut open and a bristle passed through the natural opening into the ventricle.

The illustration to the left represents the sacculus laryngis with the glands and their ducts raised from the pouch. A sketch showing their general character rather than actual size and relative position. (From a dissection by John Hilton.)

it protrudes between the ventricular band and the vocal cord into the air-way. This has not been observed apart from an inflammatory edema or thickening, or apart from the concurrent growth of a cyst or tumor.

In the case of the sacculus, the term "eversion" signifies a turning inside out of the sac, and its subsequent displacement between the ventricular band and vocal cord into the air-way. Although the condition of eversion of the ventricle has not yet been observed, it is conceivable that after com-

plete eversion of the sacculus the process of displacement might extend and subsequently involve the mucous membrane of the ventricle itself.

**The Normal Histology of the Ventricle and Sacculus.**—Lennox Browne in 1899 pointed out that the mucous membrane in the ventricle presents well-marked corrugations, especially in the upper and outer walls “irregularities of surface often so accentuated as to constitute a long tongue-like fold projecting across the ventricle almost to the glottic aperture” (Figs. 476–478). He remarked that “this is of sufficiently frequent occurrence as to afford a reasonable explanation of the condition described as eversion



Fig. 476.—A coronal section of one-half of an adult male larynx, carried through the middle of the sacculus (after hardening in formol solution), showing the cross-sections of three or more horizontal rugae projecting from the inner wall of the sacculus, such as may undergo hyperplastic lengthening in tuberculosis and come to project through the ventricle of Morgagni so as to simulate eversion. The uppermost of the three projections is the section of the cushion of the epiglottis. (From dissections by Professor S. G. Shattock, F. R. S.)

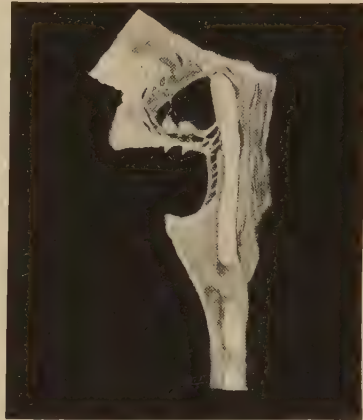


Fig. 477.—The same coronal section from which the fat has been removed by ether, and the inner wall of the sacculus forcibly drawn toward the middle line, showing the way in which (in this particular specimen) the outer wall of the sacculus is attached by fine processes of strong fibrous tissue to the perichondrium over the thyroid ala. The small spherical structures projecting from the roof into the artificial space, previously occupied by fat, are glandular. The fibers attaching the sacculus to the thyroid cartilage are not referred to by Hilton, who describes no direct attachment of the sacculus on its outer aspect. They are, however, only occasionally present, there being, as a rule, no attachment whatever in this position. (From dissections by Professor S. G. Shattock, F. R. S.)

or prolapse of the ventricle, for the ‘tongue,’ or corrugation might under certain morbid conditions become sufficiently hypertrophied and prominent to constitute the characteristic laryngoscopic appearance often described under the above name.”

Koschier (Vienna) in 1897 first suggested that edema and hyperplasia of the ventricle generally started from these irregularities and later projected into the interior of the larynx.

The normal human larynx (Fig. 478) shows one such horizontal fold in the ventricle (*c*). Compare also with Albrecht’s findings in the ventricle of the dog.

**Morbid Anatomy of the So-called Prolapse of the Ventricle.**—Stoerck (Berlin) in 1880 is said by Koschier to have been the first to ascribe the primitive origin of these “tumors” to a catarrh of the peripheral portion of the vocal cord, as well as of the mucous membrane of the ventricle itself, leading to the production of “a flat pad-shaped” protuberance of these parts.

Chiari (Vienna) in 1895 refers to this theory as having been advanced by Schroetter (Vienna) in 1887, and confirmed by Fränkel (Berlin) in

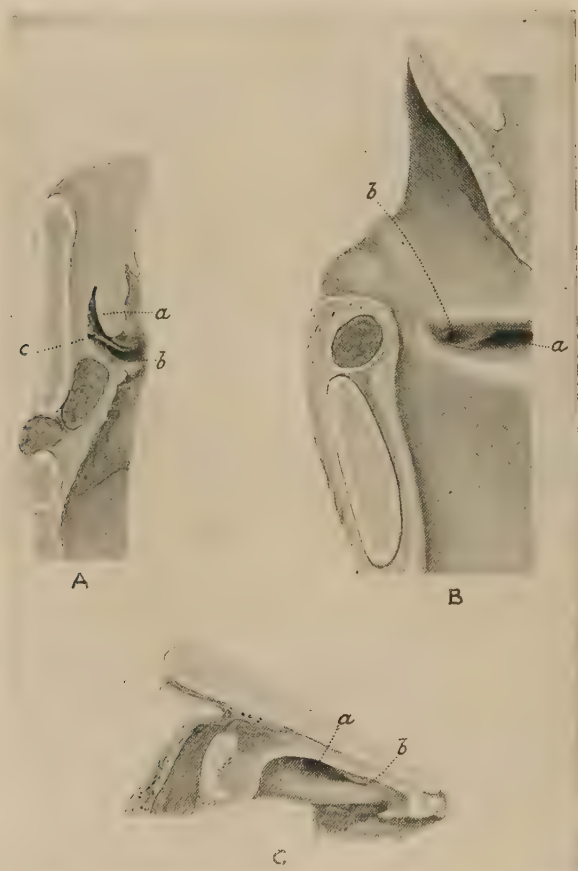


Fig. 478.—A, Coronal section of larynx through the middle of (a) saccus and (b) ventricle, showing opening of former into latter and (c) a horizontal fold of mucosa. B, Posterior half of the same larynx viewed from the inner aspect, showing (a) opening of saccus, (b) posterior end of ventricle. Between these the posterior frenum of Hilton is seen. C, Horizontal section of right half of same larynx, carried between ventricular band and vocal cord—viewed from below—showing (a) mouth of saccus, (b) cut edge of mucosa which marks the limit of the ventricle. (Dissection by Professor S. G. Shattock, F. R. S.)

1894, and describes these tumors as hypertrophied folds of mucous membrane proceeding from the wall of the ventricle, and penetrating into the larynx; the mucous membrane being displaced into the interior of the larynx by its own weight (assumed by Chiari).

Koschier agrees with these views, and in 1897 described histologically “tumors” or portions of tumors removed endolaryngeally in a series of 19

cases of so-called "prolapse of the ventricle" observed in his clinic during six years. Four were bilateral, 3 were situated on the right, and 11 on the left side. In 16 of these cases a chronic catarrhal inflammation was present along with edematous hyperplasia of the subepithelial connective tissue, which confirmed the opinion previously expressed by Fränkel in 1894, as to the histology of these "tumors." Mucous glands were either absent or present in small numbers only.

Koschier has experimentally produced an acute hyperplasia (prolapse) of the mucous membrane of the ventricle in the dog, by penetrating the larynx with a needle. He found that the base of the prolapse was situated exactly along the muscular band in the ventricle (described by Albrecht) and concludes that the prolapse may be ascribed to edema of the tissues accompanying an inflammation of the mucous membrane.

#### DISPLACEMENT OF THE MUCOUS MEMBRANE OF THE LARYNGEAL VENTRICLE—THE SO-CALLED PROLAPSE

**Classification.**—Group I.—Prolapse-like "tumors," or protruding folds of mucous membrane arising from relaxation of the mucosa.

The eversion may be due to:

- (a) Mechanical or non-inflammatory causes, *e. g.*, traumatism, voice strain.
- (b) Acute inflammatory edema or hyperplasia, *e. g.*, acute catarrh.
- (c) Chronic hyperplasia, *e. g.*, chronic catarrh, tuberculosis, syphilis.

Group II.—Eversion due to the traction of cysts and tumors.

- (a) Retention cysts, *e. g.*, Koschier's cases.
- (b) Neoplasms, *e. g.*, Koschier's case of myxofibroma; Moure's case of angiomyxoma described by Noack; Garell's case of fibrolipoma; Chappell's case of carcinoma.

**Conclusions from the Recorded Cases of So-called "Prolapse" of the Ventricle Without Involvement of the Sacculus.**—*Statistics.*—Chiari in 1895, in a review of the literature, stated that up to that date 40 cases had been diagnosed as "prolapse of the ventricle." The present writer has investigated these cases and has in addition traced a further 42 cases—making a total of 82 recorded up to the present time.

*Etiology.*—One of the predominant co-existing features of so-called "prolapse" is chronic catarrh and ulceration of the respiratory tract, the inflammatory condition eventually involving the ventricle itself. Coughing may or may not be present. In one case the condition was said to have arisen *during* a violent attack of coughing (Solis-Cohen).

One author, believing that these cases were true eversions of the ventricular wall, and not due to swelling of the mucosa, has assigned, as the only direct cause, the violence to which the tissues are subjected during the act of coughing, although how this operates, he admits it is not easy to understand, unless, as he says, one presupposes a relaxed condition of the mucous membrane (Bosworth).

The condition of true eversion, if it should occur under such conditions of relaxation of the mucosa and coughing, would be best explained, perhaps, by Shattock's theory of negative pressure produced by the blast of air liberated above the cords during the act of coughing.

It is said that voice strain, in some cases, may produce local relaxation of the mucous membrane in persons suffering from chronic catarrh, just as

cough accompanying chronic catarrh may produce the same thing (Scheimmann).

That a concussion or a severe blow on the exterior of the larynx might be sufficiently powerful to throw all the mucous membrane out of the cavity on to the vocal cord is hardly conceivable, although this has been suggested (Jellenfy, Coakley). In one case traumatism was supposed to have been the cause of an inflammatory edema leading to "prolapse" of the ventricular wall (Abraham).

In view of the fact, however, that most, if not all, of the cases of so-called "prolapse" are due to inflammatory swelling of the ventricular mu-

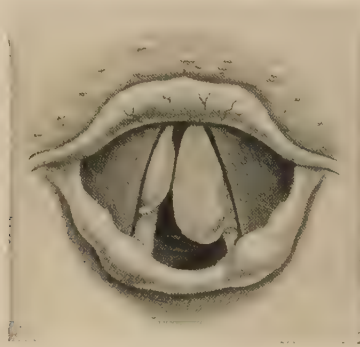
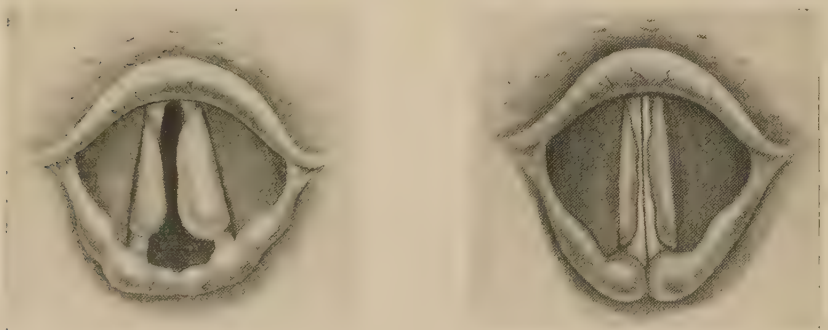


Fig. 479.—Prolapse of both ventricles of the larynx. (Lefferts' case.)



During respiration.

During attempted high chest notes.

Fig. 480.—Eversion and prolapse of both sacculi laryngis. (Louis Elsberg's case.)

cosa, the condition of eversion, except in connection with cysts and neoplasms, need not be further discussed.

A simple, true eversion is well-nigh impossible, owing to the adhesion of the ventricular mucosa to the surrounding parts and its connection above with the sacculus, the eversion of which would be entailed by that of the ventricle.

Mucosal or submucosal retention cysts and neoplasms may produce "eversion" by the displacement which the growth involves, or by the traction due to their weight. The writer refers later to the possibility of true eversion of the ventricle occurring slowly and by degrees—secondarily to,

and as a final stage in, eversion of the sacculus. This is probably an explanation of Fletcher Ingal's case.

Tuberculosis and syphilis are supposed to be predisposing causes, and it is said, in a general way, that a considerable number of the patients were suffering from one or other of those diseases.

Gouguenheim, in 1889, expressed the opinion—from the five cases he recorded of ventricular tumors presenting all the signs of prolapse (three occurring in patients suffering from tuberculosis, and one doubtful)—that "prolapse" of the ventricle is *mainly attributable* to tuberculosis.

Reference, however, by the present writer, to Gouguenheim's original paper, shows that there is nothing to lead the reader to infer that the tu-



Fig. 481.—Prolapse of the laryngeal sac. (Solis-Cohen's case.)

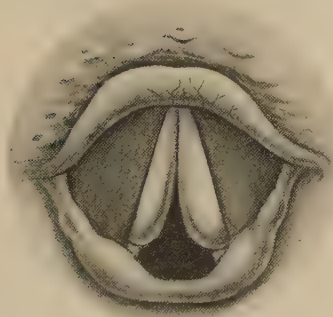


Fig. 482.—Edematous folds of the ventricular mucosa simulating prolapse of the ventricle. (Chiari's case.)



Fig. 483.—Prolapse of mucous membrane of the ventricles of Morgagni. (Schnitzler's case.)

berculous lesions were situated in the larynx; on the contrary, allusion is made only to the presence of the usual pulmonary signs.

Hence it is plain that there is no laryngoscopic evidence to show that laryngeal tuberculosis was present in any of the 5 cases recorded by Gouguenheim, and its coexistence as a causative factor has only been surmised.

Tuberculosis may undoubtedly lead to spurious forms of eversion of the ventricle or the sacculus and may simulate true eversion of them, as in a specimen preserved in the Museum of Golden Square Hospital (Figs. 491, 492).

Ruault has referred in discussion to the rarity of tuberculous hyperplasia of the ventricle simulating so-called prolapse, and has found from a series

of 167 cases of tuberculosis of the larynx that only 3 cases of "prolapse" occurred.

A study by the present writer of 82 cases of so-called prolapse, without inversion of the sacculus, recorded in the literature reveals the fact that laryngoscopic signs of laryngeal tuberculosis were present in only 3 cases; while in 4 cases the local condition was only associated with tuberculosis in the sense that it co-existed with pulmonary phthisis.

Moure has suggested that the formation of these ventricular folds may possibly in some cases be due to syphilis, but of the correctness of this no pathological evidence is forthcoming.

The breaking down of a gumma in the region of the ventricle might conceivably lead to loosening of the ventricular wall and eversion,

Syphilitic lesions of the larynx have only been observed on autopsy in 2 cases, and laryngoscopic evidence of gumma only in one; while in the remaining 5 cases, although syphilis co-existed with the laryngeal condition, its action as the causative agent of the latter was only surmised. Tumors



Fig. 484.—Prolapse of the ventricles of Morgagni. (Moller's case.)

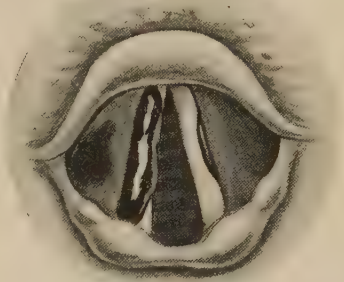


Fig. 485.—Traumatic laryngitis with prolapse of the right ventricle. (Abraham's case.)

accounted for 5 cases (4 benign and 1 malignant); cysts, 5. Of the remaining 57 cases chronic catarrh was present in the majority.

Hence it is clear that beyond the inflammation and ulceration to which tuberculosis and syphilis give rise, with their accompanying coughing, there is no ground whatever for believing that they play any special part in the production of so-called prolapse.

There is no clear example among the whole series of cases recorded of a simple eversion of the ventricular wall unaccompanied by an inflammatory swelling.

*Age.*—The condition in every case was observed in full adult life varying from the age of twenty to sixty-five.

*Sex.*—In only 30 of the cases recorded was the sex definitely stated, and of these, 25 were males and 5 females.

*Side.*—In only 39 cases was this recorded: 12 were bilateral, and 27 unilateral (15 on the right, and 12 on the left, side).

*Symptoms.*—These are a chronic cough or a single severe attack of coughing in a patient suffering from chronic catarrh of the pharyngolaryngeal tract and hoarseness ranging from a coarse whisper to a gradual or sudden complete aphonia, which may be intermittent or persistent, and

may have existed for a period of time varying from twenty-four hours to many years. The aphonia is caused either by the mucous membrane protruding between the vocal cords and mechanically preventing adduction, or by its lying on the vocal cords and interfering with their vibration. In one case a harsh voice had existed since infancy. Dyspnea, inspiratory stridor, and even cyanosis have been observed in many cases, as well as dysphagia and pain over the larynx.

**Laryngoscopic Appearance and Diagnosis.**—It can be clinically diagnosed by its well-defined appearance; viz., a longish or pear-shaped, smooth or edematous-looking tumor, representing all the characters of mucous membrane, or differing only little in appearance from the adjoining mucosa, of a reddish or pale-red color, distinct from the true and false vocal cords protruding from the ventricle and covering the true cords partly or entirely. The tumors have been described as tapering anteriorly, the posterior extremity being more rounded, and the upper surface flattened. The anterior and posterior borders of the tumor are said always to fall sharply to the level of the vocal cord (Jellenfy). The swelling may vary in size from a mere fold to that of a small cherry (Solis-Cohen) or a pigeon's egg (Reardon). It is soft, easily indented with a probe, and can be readily replaced or tucked into the ventricle by means of a bent probe—to protrude again immediately on coughing. If it is in conjunction with cyst formation, the tumor may look pale and tense, with a grayish, glassy appearance. In one case the edges of the ventricular band were quite indistinct during respiration but were *sharply defined* during phonatory effort (Elsberg). In another, the diagnosis was made partly on the fact that there was a *lack of a line* of demarcation between the ventricle and the ventricular band (Solis-Cohen). It is also said that prolapse can be diagnosed by the partial rolling of the mass out of sight into the ventricle on phonation and, in rare instances, its complete disappearance, leaving a flabby, wrinkled fold; while on respiration it protrudes more distinctly.

Again, in 2 cases recorded by Nolan Mackenzie, forced respiration had no effect upon the tumor, and no change was observed on phonation, except that it became more tense. It has been shown that shrinkage follows the application of astringents to the swelling, which confirms its acute inflammatory nature in some cases.

**Differential Diagnosis.**—New growths, benign or malignant, may be mistaken for it, and it has been found difficult to diagnose it not only from a new growth but also from the ventricular band (Semon). New growths, however, are usually irregular in outline and firmer in consistence. Benign growths are generally pedunculated and readily movable. It has been mistaken for a polypus (Waldenburg's case). It may be diagnosed from a fibroid growth by its size, absence of a pedicle, its color and smooth surface, and its softness on palpation (Lefferts). A fibroma, a fibromyxoma, a fibrolipoma, and an angiomyxoma, have each been mistaken for prolapse of the ventricle. From a cyst it may be diagnosed by puncture, or microscopical section after removal. It may be distinguished, again, from simple eversion of the sacculus since the protrusion in the latter case is confined to the anterior portion of the elongated ventricular opening, and conclusive proof of the nature of the protrusion (if removed) may be found on microscopical examination (Uckermann's case). Malignant growths are attached by a broad base, and may be irregularly nodular and firm on palpation, and ac-

accompanied by glandular involvement. Carcinoma has been confused with it (Chappell's and Schutter's cases). From tuberculosis it may be excluded by the absence of other symptoms, and such diagnosis rendered improbable by the rarity of tuberculous disease of the ventricle, as is shown by the statistics of Ruault, and by those gathered by the present writer from the records of all the published cases. One case has been diagnosed as tuberculous, and treated as such (Delsaux). It may be mistaken for a syphilitic gumma; the latter, however, is usually unilateral, firm in consistence, and not susceptible of being reinserted into the ventricle. The diagnosis from a gumma is difficult when the tumor fills the laryngeal cavity (Merrick's case).

**Treatment.**—If causing no inconvenience no treatment is necessary. If, however, marked dyspnea or stridor is present it may be necessary to remove the tumor. Among the 82 cases recorded, urgent symptoms requiring immediate operation only occurred in the 2 cases. In acute cases, rest and inhalations, cold compresses along with astringent sprays and injections of menthol have been successfully employed with disappearance of the swelling after the accompanying laryngitis has cleared up. Intubation has been employed in one case (Reardon). In more chronic cases daily replacement of the swelling with a bent probe, followed by painting with astringents, *e. g.*, persulphate of iron, sulphate of copper, iodide of zinc, chromic or lactic acids, have effected relief and even cure.

The tumor has been regarded by one author as a sort of incarcerated hernia, and in several cases he attempted to shut off the circulation by a series of superficial incisions over its contour, thus successfully reducing the "prolapse" (Jellenfy).

The galvano-cautery has been employed in one case (W. S. Jones), and it is said that a swift cure may be effected by this method (Delsaux). In chronic cases in which marked dyspnea or stridor was present, removal has been effected endolaryngeally by forceps, the cold wire snare, or the laryngeal guillotine. Thyro-fissure has been performed in one case, under the belief that endolaryngeal removal might result in stripping of the ventricle, *i. e.*, turning the ventricle inside out (Lefferts); and a preliminary tracheotomy, followed by thyro-fissure, has been carried out in a second case on account of stridor (Lang). In a case of eversion secondary to cyst formation the cyst was first punctured, and the everted ventricle was later removed by means of a wire snare (Fletcher Ingals).

#### DISEASES AND MORBID CONDITIONS OF THE SACCULUS VENTRICULI LARYNGIS

With the approval and kind co-operation of the late Professor S. G. Shattock I have tabulated these diseases or abnormalities as follows:

1. Hydrops (unobserved).
2. Mucocoele (unobserved).
3. Pyocoele, *e. g.*, the case of Kistner.
4. Pneumatocoele:
  - (a) Laryngocoeles, *e. g.*, the cases of Bennett, Parker, Gruber.
  - (b) Hernia, *e. g.*, the cases of Solis-Cohen, Garel, Frederick Spicer.
5. Eversion of the sacculus:
  - (a) Without eversion of the ventricle, *e. g.*, Moxon's, Morell Mackenzie's, and Uckermann's cases.
  - (b) With eversion of the ventricle, suggested by cases recorded by Fletcher Ingals, Koschier, and Joseph Cohen (Cologne).
6. Gland cysts of the wall of the sacculus (either with or without eversion). It is possible that the 3 cases last enumerated belong more properly to this group.

## EVERSION OF THE SACCULUS WITHOUT EVERSION OF THE VENTRICLE

Walter Moxon (Guy's Hospital), in 1868, was the first to study and record this condition. The "tumor" was unexpectedly observed at the autopsy of a male patient who died of cancer of the stomach. No laryngeal symptoms were present during life. The larynx is now in the Museum of Guy's Hospital. It shows a small pendulous "tumor," hemi-elliptical in shape, which protrudes from the anterior half of the ventricle, and hangs down over the vocal cord (Fig. 488). Moxon stated that it could easily be put back into



Before opening the cyst and evacuation of the fluid. After opening the cyst, showing the everted ventricular wall.

Fig. 486.—Eversion of the left ventricle of the larynx secondary to a cyst involving the larynx and the side of the neck. (Fletcher Ingals' case.)



Fig. 487.—A cyst of the left laryngeal ventricle. (Joseph Cohen's case.)

the usual position of the sacculus of the larynx, *i. e.*, it could be inverted and returned behind the false cord. When replaced it very easily fell out of its position and again reappeared. On these grounds he regarded it as the everted sacculus laryngis. Arthur Durham, in 1883, referred to this case as "extroversion" of the mucous membrane of the left "ventricle" of the larynx, simulating laryngeal polypus.

Permission was recently obtained by the writer to re-examine this specimen. On removing a portion of the left thyroid ala it was found the sacculus was absent from its normal position—which proves that Moxon was cor-

rect; and although one-half a century has elapsed since this specimen was mounted, the everted sacculus could easily be replaced into its normal position, and again everted into the position which it now occupies.

Morell Mackenzie in 1871 recorded the second specimen, now in the Museum of Golden Square Hospital (Figs. 489, 490). It was unexpectedly observed after death in a male patient, who had been admitted to the hospital one evening suffering from tuberculosis of the lungs and larynx, and who died the next morning. No notes could be taken, and there is no record as to the state of his voice. Morell Mackenzie stated that the left ventricle of Morgagni was entirely everted, and that the right sacculus protruded slightly from the ventricular orifice. It is evident that Morell Mackenzie

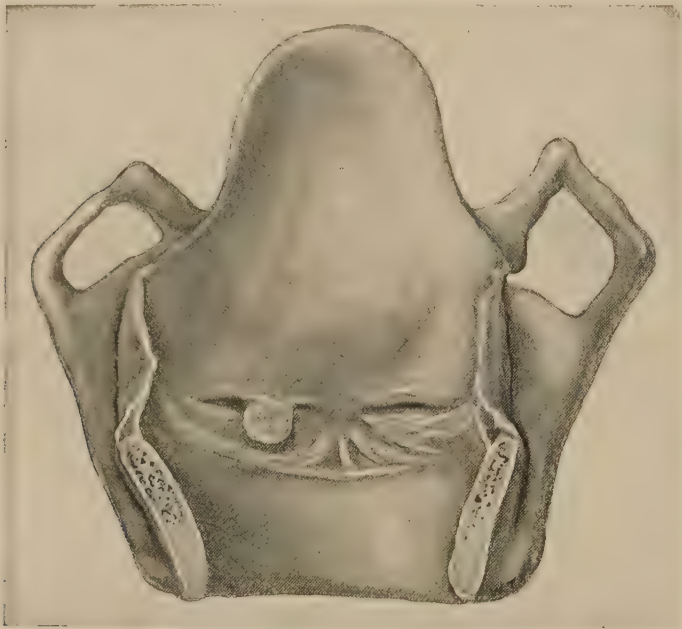


Fig. 488.—Eversion of the left sacculus laryngis. (Moxon's case.) Specimen from the Museum of Guy's Hospital.

employed the terms "ventricle" and "sacculus" as synonymous with each other, for he referred to having resected a portion of the left thyroid ala from without, and not only found that the sacculus laryngis was absent from that side, but that the protruded sac could be inverted and replaced in its normal position, presenting the well-known appearance of a Phrygian cap. A recent examination of this specimen shows that the tongue-like protrusion occupies the anterior third only of the ventricle of Morgagni (Fig. 489), that there is no eversion of the proper ventricle; and that the everted sacculus could readily be replaced into its normal position (Fig. 490), and again everted into the larynx. There is a similar but less pronounced eversion of the sacculus of the opposite side. The mucous membrane of the larynx in this specimen is the seat of tuberculous ulceration, most marked on the ventricular bands and cushion of the epiglottis. There were tuberculous cavities in the lungs.

Morell Mackenzie, in referring to Moxon's case, says: "There is every reason to believe that the 'prolapse' took place *in articulo mortis*," and he

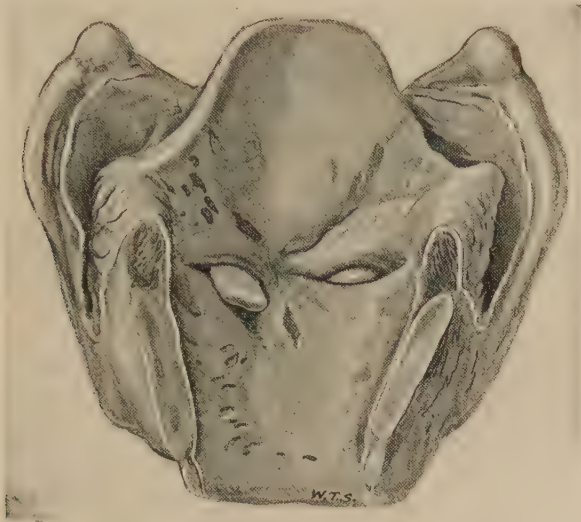


Fig. 489.—Eversion of the left sacculus laryngis; partial eversion of the right sacculus laryngis. (Morell Mackenzie's case.) Specimen from the Museum of the Hospital for Diseases of the Throat, Golden Square.



Fig. 490.—The same specimen with the everted sacculus laryngis replaced into its normal position and lightly distended with cotton wool. (Morell Mackenzie's case.) Specimen from the Museum of the Hospital for Diseases of the Throat, Golden Square. (Redissected by Professor S. G. Shattock, F. R. S.)

suggests the same possibility in his own case, but he gives no reason for justifying such a conclusion. Lefferts in 1876, referring to Moxon's and Morell Mackenzie's cases, takes the same view on the grounds that "there

were no symptoms during life indicating so serious a lesion," an opinion formed without an inspection of Moxon's specimen, in which the projection

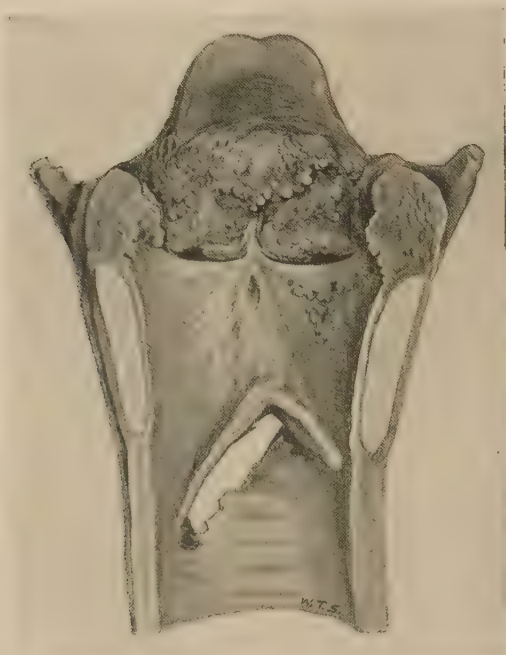


Fig. 491.—Larynx showing hyperplastic tuberculosis simulating prolapse of the sacculus ventriculi. Specimen from the Museum of the Hospital for Diseases of the Throat, Golden Square.

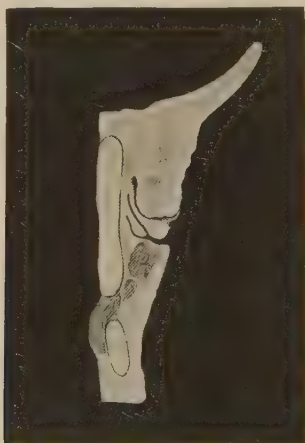


Fig. 492.—Coronal section of ventricle and sacculus (see specimen, Fig. 491), showing hyperplastic tuberculosis and formation of folds, simulating prolapse. Interior of larynx is site of tuberculous disease of similar hyperplastic character. Section by Professor Shattock, F. R. S., of a specimen in Museum of Golden Square Hospital.

is so small and thin that the absence of symptoms referred to in the notes of the case is quite explicable. In Moxon's specimen of true eversion of the sacculus, recently confirmed, the mucous membrane covering the upper

part of the tumor merges into that of the upper surface of the ventricular band. The explanation of this is that in addition to eversion of the sacculus there is, as Shattock pointed out, some dragging on the roof of the ventricle between the mouth of the sacculus and the free border of the ventricular band—an indication that the eversion occurred during life, and not *in articulo mortis*, as suggested by Morell Mackenzie.

All the authors cited, almost without exception, appear to be unaware of the pathology of Moxon's and Morell Mackenzie's specimens, and having seen no true case of eversion themselves, have either endeavored to explain away the existence of these, or to include them in the other group into which their own cases fall, viz., inflammatory hyperplasia.

Uckermann (Christiania), in 1914, showed a small irregular tumor the size of a pea, proceeding from the right ventricle of Morgagni, in a youth aged eighteen. It was removed by means of forceps, and on microscopical examination was found to consist of mucous glands embedded in loose connective and fatty tissues, as well as round cells, that is to say, hyperplasia of the mucous membrane coupled with inflammation, or, as it was formerly designated, "eversio ventriculi Morgagni." (Professor Shattock considered that there was definite microscopical evidence to show that this was a case of eversion of the sacculus.)

#### SPURIOUS EVERSION OF THE SACCULUS ARISING FROM HYPERPLASTIC TUBERCULOSIS OF THE MUCOSA

Tuberculosis undoubtedly, as has been shown, leads to spurious forms of eversion. An excellent demonstration of this condition is afforded by a specimen in the Museum of Golden Square Hospital labelled "Prolapse of the Ventricle" (Fig. 491).

The writer thoroughly investigated this specimen with the late Professor S. G. Shattock, F. R. S.

A close inspection shows that the mucosa is beset with nodular elevations, and the sacculus with flattened, leaf-like processes. In the other section the ventricle and the sacculus are easily recognizable, but from the upper part of the sacculus there project two lamelliform processes with slightly enlarged free ends, which occupy the cavity of the ventricle, from which the higher slightly projects.

"A microscopic section of one of the processes showed it to contain numerous giant-celled systems. The mucosa in general is the seat of a papillary hyperplastic tuberculosis" (Fig. 492).

#### EVERSION OF THE SACCULUS

**Etiology.**—It is highly probable that chronic inflammatory conditions of the sacculus may furnish one of the predisposing causes by producing relaxation of its supports, and subsequently leading to eversion as a result of the negative pressure induced by coughing.

Nolan Mackenzie, in recording his 2 cases of "prolapse of the ventricle," and employing the term "ventricle" as synonymous with sacculus, discusses the question of etiology of "eversion of the sacculus," and has suggested that long-continued inflammation may lead to infiltration and fatty degeneration, and finally induce a paralytic state of the muscular surroundings of the sac, leading eventually to abrogation of the suspensory function of the internal fibrous ligament of Hilton—in other words, a perisaccular in-

flammation accompanied by fatty degeneration and wasting of muscles. Again, he suggests that descent of the sac may be facilitated by hyperplasia of the areolar tissue surrounding it, which may push the walls of the sac downwards towards the cavity of the larynx. Later on, when eversion is complete, strangulation of the mass takes place and the sac presents itself in the laryngeal cavity as a congested tumor.

Cysts may possibly arise in the saccular wall and cause eversion; those cases recorded by Fletcher Ingals, Koschier, and Joseph Cohen probably originated primarily as cysts of the sacculus, and secondarily produced eversion of the ventricle. As demonstrated by the specimen of Koschier, eversion of the ventricle may be caused by the weight of a tumor growing in and dragging on its walls, and everting the sacculus.

The late Professor S. G. Shattock suggested, in 1921, that *negative pressure* caused by violent coughing was the main factor in producing eversion of the sacculus, and he demonstrated, by means of a model, the mode in which this might take place. He referred to the effective closure of the glottis which precedes coughing, and which takes place at the site of the true cords; and he pointed out that any approximation of the ventricular bands can be only a concomitant, seeing that the latter are unprovided with muscle. He says: "When the glottis is suddenly opened in coughing, the blast of liberated air rushes by the ventricle and orifice of the sacculus; and if often repeated the negative pressure so induced may lead first to some degree of loosening of the attachments of the sacculus, and then to its complete eversion. There may be another factor. The need of occasionally 'clearing the throat' while speaking, is possibly due to the descent of mucus from the sacculus and its engagement in the glottis, which impairs the proper vibration of the cords; a slight expiratory effort dislodging the secretion and restoring the voice. The act of forcible coughing might thus, should an unusual amount of stringy mucus project from the sacculus, be followed by a drag which would tend to empty the latter and loosen its connections."

**Symptoms.**—There may be no symptoms, as shown by the history of Moxon's case. Slight dyspnea may be present if the swelling is bilateral and causes narrowing of the glottis. Sudden loss of voice may occur. This, however, as previously stated, need not be construed as due to a sudden eversion but to constriction and congestion of the sacculus at the ventricular opening.

**Laryngoscopic Appearance and Diagnosis.**—1. The situation of the tumor in the anterior third of the larynx would suggest eversion of the sacculus, but this cannot be depended upon, for even if the swelling is confined to the anterior portion it might be a simple hyperplasia, or a spurious form of tuberculous hyperplasia (Figs. 491 and 492).

2. The true vocal cords stand clear of the mass, as may be seen by pressing the free end of the swelling upwards and outwards with a probe. This was observed in Morell Mackenzie's and Moxon's specimens.

3. The contour of the ventricular bands is not lost. Its upper border does not merge into the upper surface of the swelling as in eversion of the ventricle, but forms with the tumor a shallow step. This was observed in Morell Mackenzie's specimen. In Moxon's specimen, however, as previously stated, the protrusion of a portion of the mucous membrane of the ventricular band had, along with the eversion of the sacculus, obliterated the contour of the ventricular band.

4. The tumor may be replaced partially or fully into the ventricle by means of a bent probe, only to fall out again almost immediately.

**Differential Diagnosis.**—It is said that it can be diagnosed from chronic catarrhal hyperplasia, abscess, and tumors, by its sudden development, but eversion of the sacculus may exist for years, and attention may only be

drawn to it by symptoms which suggest a sudden development, but which really are caused by constriction and congestion of its neck at the ventricular orifice.

**Treatment.**—If causing inconvenience or dyspnea removal may be necessary. This can be carried out by forceps or snare, but the risk of tearing out or “stripping” the ventricle must be considered. Removal by thyro-fissure would appear to be the best surgical procedure. A window resection of the thyroid ala in conjunction with endoscopic replacement of the sac, followed by its fixation in its normal position by means of ligatures, or even removal by ligaturing off the sac at its neck, would appear also to offer a satisfactory method of dealing with the condition.

IRWIN MOORE.

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## CANCER OF THE LARYNX

**Introductory.**—The investigation of cancer, when it attacks the larynx, is particularly interesting because, in this organ, the disease may be manifested under two forms, of which one is more amenable to treatment than is cancer in any other internal region of the body, while, under the other form, it may present as the most hopeless and distressing manifestation of this disease.

**Classification.**—This striking difference is not based on any histological variation; nearly all cases are epitheliomata. It depends on the location of the point of origin.

It is forty-seven years since Krishaber made the far-reaching observation that cancer arising in the interior of the larynx ("*intrinsic cancer*") was extremely slow in evolution and, for a long time, remained well defined and limited, only invading the glands at an advanced stage, but that malignant disease of the margins or outer surface of the voice-box ("*extrinsic cancer*") was insidiously rapid and spread to the lymphatics at a very early part of its career.<sup>1</sup>

At its inception cancer is a localized disease. There is no internal region of the body where cancer gives such an early warning of its occurrence, where it is so slow in evolution, and where it so long remains localized—as on a vocal cord. In no other internal situation can surgery secure such enduring freedom from the disease.

On the other hand, when cancer originates on an aryepiglottic fold or the posterior surface of the cricoid, the invasion is insidious, the disease is not sharply circumscribed, and before it has made its presence felt by the patient it has frequently lost any frontier line and has invaded the glands. It is rare for it to be lastingly benefited by surgical operation.

INTRINSIC AND EXTRINSIC CANCER OF THE LARYNX<sup>2</sup>

A. **Intrinsic cancer of the larynx** generally attacks the area of the vocal cords or the subglottic region. I have never seen it originate on the ventricular bands or the interarytenoid region. It might as well be called "*chordal cancer*" were it not that, occasionally, it originates in the ventricle of Morgagni or in the more vascular and glandular tissue of the subglottic area. Chordal cancer always declares itself by an early and persistent hoarseness.

The subglottic variety is slower to make its presence felt. The voice may be a little muffled or "stuffy," but huskiness may hardly be noticeable until the disease has so far extended as to invade the cord and impair its action. The symptoms of intrinsic or chordal cancer and of its subgroup, subglottic cancer, are eminently vocal and respiratory. The subglottic group, as a variety of intrinsic cancer, was noted by Isambert fifty years ago.<sup>3</sup>

B. **Extrinsic cancer of the larynx** may originate on the epiglottis, on the aryepiglottic folds (upper margin or posterior surface), or on the posterior (pharyngeal) surface of the cricoid. In all these regions hoarseness is not an early symptom and vocal change is not noticeable until an advanced stage. The symptoms are those of other pharyngeal disorders, and are much more concerned with dysphagia than with dysphonia.

Clinically, as many cases do not present themselves until they have made considerable progress, we might add a third or

C. **Mixed group**, made up of intrinsic cancers which have spread beyond the glottic margin, or extrinsic cases which have extended into the cavity of the larynx.

**Etiology.**—Little is definitely known as to the cause of cancer. The latest conception (based on the work of Gye and Barnard) is that it is due to an ultramicroscopic virus common to different classes of animals. This virus, when it enters into the cell, stimulates it to characteristic, independent ("lawless") growth. But the mere presence of the virus does not produce

cancer, because by itself the virus is unable to attack a normal cell. To do this the presence of an accessory factor or factors is required.

What the factors may be in laryngeal cancer we do not certainly know. My own experience lends no justification to attributing the local predisposition to misuse or overuse of the voice, or of alcohol or tobacco.

The disease is met with in women who have never smoked and who have been life-long abstainers. Professional voice users are not more frequently attacked than others. Syphilis, though not unknown, only occurs in a small proportion of cases.

Nasal stenosis or suppuration cannot be invoked as contributory causes, and I have seen the disease, in early stages, in patients who have been edentulous for years.

Only two definite influences can be accepted as accessory factors, they are (a) age and (b) sex.

(a) As with cancer generally, the larynx is more often invaded as life advances. My youngest case was a man of twenty-three,<sup>4</sup> but Chiari saw it on the vocal cord of a girl of sixteen.<sup>5</sup> It is, however, rare before forty and increases with subsequent decades.

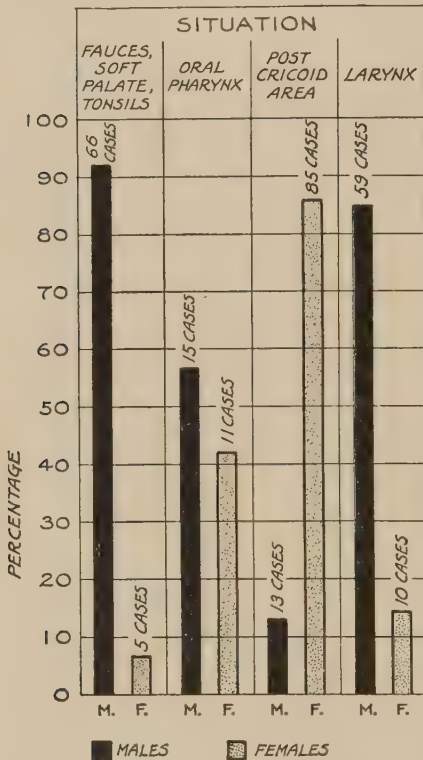


Fig. 493.—Graph of sex-incidence of carcinoma of the fauces, oropharynx, post-cricoid region, and larynx. (From statistics compiled and published by A. Logan Turner, *Jour. of Laryn.*, xxxv, 1920, No. 2, p. 34.)

and increases with subsequent decades. My oldest patient (operated on successfully and free from recurrence six years afterward) was eighty.

(b) The distribution of laryngeal cancer, according to sex, is definite and curious. Intrinsic cancer is rare in women. I have performed a laryngofissure for intrinsic or laryngeal cancer on 62 male patients and only on 8 females. This experience is universal (Fig. 493).

Extrinsic cancer appears to be less frequent with women (as occurs with malignant disease of the pharynx) except in one situation; viz., on the posterior (pharyngeal) surface of the cricoid. This posteriocricoid cancer is uncommon in male patients; relatively it is frequent in females. With them, also, it occurs at an earlier age (between thirty to forty years of age

it is not unusual) than do malignant growths in other parts of the pharyngolarynx (the sinus pyriformis) in males.

**Symptoms and Physical Signs.**—These are so different in the intrinsic and the extrinsic group that they require entirely separate consideration.

#### INTRINSIC CANCER OF THE LARYNX

**Symptoms.**—In intrinsic laryngeal cancer there is always one symptom and, for months, it may be the only one. It is hoarseness, slight at first, sometimes variable, but generally persistent and progressive. The hoarseness is often dated from a "sore throat," a "cold," or "an attack of influenza." It may be so insidious that neither the patient nor his friends can be positive about the date of commencement. Even when the first suggestion is a huskiness of "a month or two," it is generally possible to elicit a history of six or nine months.

The singing voice is the first to go. The hoarseness varies; it is worse in the morning and after an evening spent in bad air or very thick tobacco smoke. It abates with vocal rest, good air, and other favorable circum-



Fig. 494.—Intrinsic cancer of the larynx. Characteristic cupped, gray infiltration of the anterior half of cord. The cord moved freely. The patient remains well and free from recurrence five years after laryngofissure. He is a physician in active practice.



Fig. 495.—Intrinsic cancer of the larynx. Shows a cupped growth which extended deeply into the subglottic space. The cord was quite fixed.

stances. I have met with only one case where there was a genuine (though temporary) improvement. A patient was sent to me from India because, in Calcutta, a projecting growth had been seen and diagnosed as malignant disease. On the voyage he was violently sea-sick, spat up "a growth" in the Red Sea, and when he reached London his voice sounded nearly normal and there was only a small embedded tumor on the usual site. (This patient, No. 27 in my schedule of laryngofissures, is alive and well twelve years after operation, with a good, strong voice.)

The voice, after much use, may feel fatigued. There is no cough, no dysphagia and, generally, absolutely nothing else to complain of. The hoarseness for months at a time may remain stationary if there is no intercurrent attack of laryngitis, and hence, in too many cases, the condition is looked on as "chronic" and comes to be regarded by the patient, his friends, and even his family medical adviser as harmless and negligible. It is not at all uncommon for patients not to present themselves until they have been continuously husky for three, six, twelve, or more months and, even then, for the disease to be still limited to a cord.

When such cases are examined there will always be found a lesion of one cord, and the unilateral manifestation of a chronic condition should immediately warn the laryngologist that there must be a specific disease, and that ordinary catarrhal laryngitis, or even an infection from the pharynx, nose, or mouth, could not remain so long limited to one side.

Intrinsic cancer may attack any part of a vocal cord, but much more frequently it invades the middle and anterior thirds.<sup>6</sup>

It is a mistake to think—as was announced by Semon and others—that malignant disease is more frequently met with in the posterior half of the glottis.

The disease may appear on a cord as (a) a projecting or semipedunculated growth resembling a simple papilloma, (b) a superficial but limited infiltration which, later on, may ulcerate or fungate, and (c) as an embedded infiltration.

(a) *The projecting, papilloma-like growth* may occur in any part of the cord (Fig. 496). It is more common (thence more difficult to diagnose)

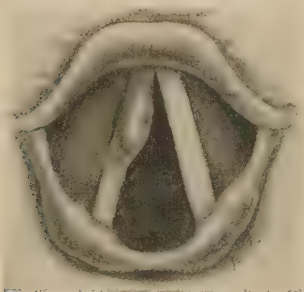


Fig. 496.—Epithelioma of right vocal cord as seen in mirror. The patient remains well and free from recurrence six years after laryngofissure. His voice enables him to carry on a large office practice as a lawyer.



Fig. 497.—Epithelioma of vocal cord, which also invaded the subglottic space. The cord was quite fixed. Laryngofissure thirteen years ago. He remains quite free from recurrence with a voice which enables him to address political meetings in the open air.

at the junction of the middle and anterior thirds. It is easily removed, *per vias naturales*, and this removal may be so complete that a lasting cure may be secured by this simple operation in cases where the cancer-cells have not penetrated the cord.<sup>7</sup> It is doubtless such cases which have prompted the suggestion that endolaryngeal methods might be relied on for extirpating a malignant growth well limited on a vocal cord. This is a dangerous doctrine for, although I have satisfied myself in my own practice that such a consummation may be achieved, it is running a dangerous and unnecessary risk, for there is no definite means of determining beforehand how deeply into the tissues a growth may penetrate, and most laryngeal growths are found (when viewed directly) to be more extensive than they appear when reflected in the laryngeal mirror.

These projecting and papilloma-like neoplasms may also be misleading for the following reason: The projecting mass, when removed, may be reported by the pathologist to be of an innocent character, while the more penetrating portion (which has escaped the grasp of the laryngeal forceps) may still be malignant.



Epithelioma of vocal cord removed by window resection by Seymour Jones. Hoarseness and dyspnea for three months. Cicatricial left cord and fixed arytenoid. Irregular, dull-pink, whitish pointed malignant growth of right cord from vocal process behind up to, and across, anterior commissure. Goes deeply into subglottic, causing stridor.



PLATE XVI



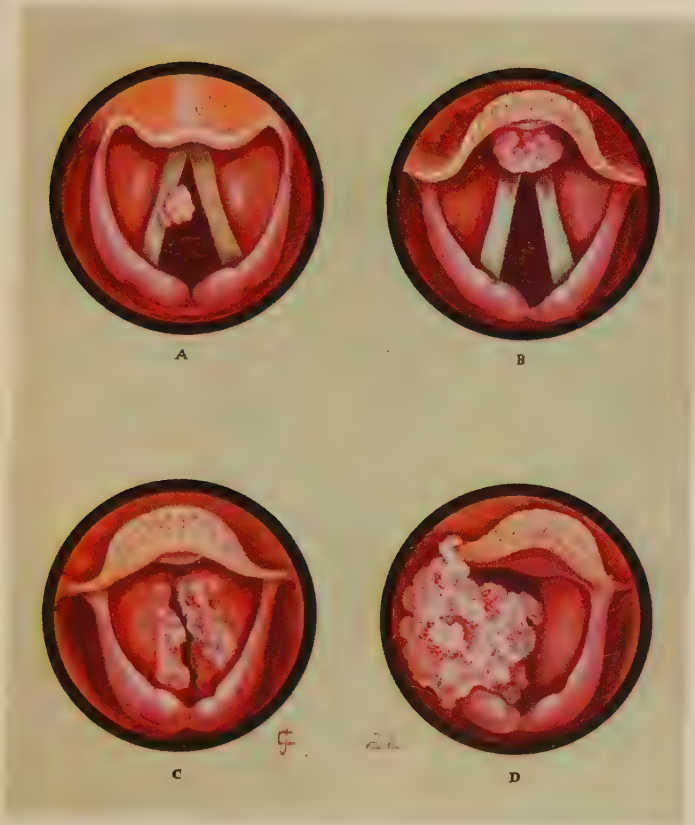
1. Epithelioma of a vocal cord. The growth did not penetrate deeply and simulated an innocent neoplasm.



2. Epithelioma of vocal cord. Laryngofissure on November 28, 1912. Drawing shows granuloma removed February 4, 1913.



# PLATE XVII



A, Intrinsic cancer of the larynx in a man aged thirty-six years. This was an ideal case for thyrochondrotomy. The patient is well now twenty-one years after operation. This can be classed as an anterior intrinsic growth; B, Anterior intrinsic cancer of the larynx involving the anterior ends of both cords, in a man aged forty-two years; C, Bilateral epithelioma that has extended too far posteriorly to permit of adequate removal of adjacent normal tissue. Laryngofissure would probably be inadequate in such a case. Laryngectomy was advised and was followed by relative cure. The patient is still well after five years; D, Extrinsic cancer of the larynx in a man aged sixty-two years. (Chevalier Jackson.)



(b) *Early malignant disease, as a limited infiltration*, may occur anywhere in a vocal cord or on the upper surface or the free margin (Fig. 496). But it is not common on the posterior third or the area of the vocal process. It occurs more commonly on the middle and anterior thirds, and as this is a common site for simple neoplasms, for singer's nodules or for leukoplakia, and as the limited thickening has few characteristics to differentiate it from other conditions, the diagnosis may be very difficult. When the thickening is more extensive and is seen to be spreading; when the surface is irregular

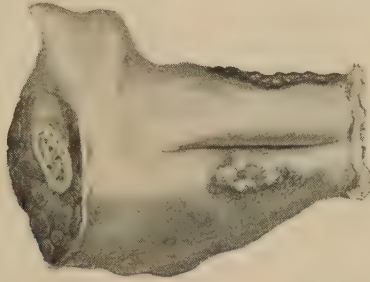


Fig. 498.—Intrinsic cancer of larynx. Shows extent of tissue removed by laryngofissure. It embraces the anterior commissure, the subglottic area, and a large portion of the arytenoid. Operation in December, 1924. No recurrence four years afterward; large air-way; strong, rough voice; full vigor.

and nodular; when it becomes dimpled; when it presents discrete, white spicules or even an appearance like a grass field which has been lightly dusted with snow; when it is fungating or even cauliflower-like, the condition is very suspicious and warrants further diagnostic observation.

(c) *A diffuse infiltration of one cord* is another form in which epithelioma may appear on a vocal cord (Figs. 494, 497). This infiltration occurs, by pref-



Fig. 499.—Limited posterio-cord epithelioma (extrinsic cancer of the larynx), from a woman aged thirty years, with six months' history of dysphagia. Larynx excised by E. B. Waggett. (E. D. D. Davis.)

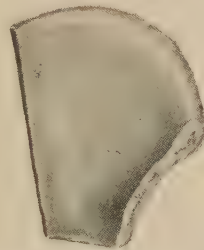


Fig. 500.—Shows the amount of thyroid ala removed in a laryngofissure operation.

erence, in the anterior two-thirds of the cord, but the whole extent may be invaded. Careful comparison with the opposite cord shows that the affected one has lost its outline, its flat, ribbon-like upper surface, its well-defined margin, and its white, glistening surface. The infiltrated portion has lost its polish and is dull, congested, or even beefy. The surface may be irregular and, as in the preceding type, it may develop a nodular, a dimpled, a spike-like, or a fungating surface.

**Mobility.**—Forty years ago Lublinski noted that in many of these in-

trinsic cancers the movement of the cord was impaired or even abolished.<sup>8</sup> This is correct in some cases. But his cases could not have been what we, now a days, call early cases, and it was unfortunate pronouncement of

Semon, in 1886, that a malignant tumor usually caused a considerable impairment of the mobility of the affected cord at a very early period.<sup>9</sup>

Now, the "impaired mobility" is not an early symptom, for hoarseness and physical signs in the cord are present for at least several months before it develops. It in-



Fig. 501.—Advanced postericoid epithelioma, involving the posterior surface of the larynx, the sinus pyriformis, and the wall of the pharynx. (E. D. D. Davis.)

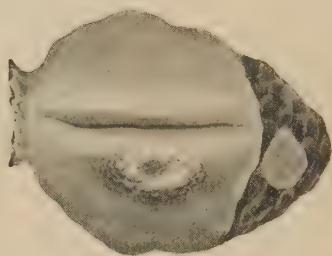


Fig. 502.—Intrinsic cancer, on the margin and inner surface of the right vocal cord. Shows tissue removed, embracing the anterior commissure and the vocal process. Patient is a physician in active practice and is without recurrence six years after operation.

dicates an advanced infiltration of the disease. In an analysis of 44 consecutive cases—where the site of origin was displayed at a laryngofissure and



Fig. 503.—Postericoid carcinoma. Shows (viewed from above) the complete ring of the lower pharynx, removed through a lateral pharyngotomy.



Fig. 504.—Postericoid carcinoma. The same specimen as Fig. 503, viewed from the side.

the nature of the growth verified by microscopic examination—the affected cord was quite mobile in 27 cases.<sup>10</sup>

Fixation of a cord may also occur with a syphilitic or a tuberculous de-

posit. Paresis is only of help, when it occurs, in distinguishing an innocent from a malignant neoplasm. It indicates an advanced and deeply infiltrating growth and, when present, is helpful in distinguishing an innocent from a malignant neoplasm. But its absence is no evidence that a growth is not malignant and if stress is laid on the absence of impaired mobility as a proof of innocence it may lead to disastrous delay. Finally, it is more frequent with subglottic growths.

*Subglottic cancer* is less often encountered than chordal cancer or than cancer of the laryngopharynx, but it is not rare. For instance, in studying 50 consecutive cases of laryngofissure for intrinsic cancer I find that in 13 of them the subglottic area was invaded; in some of these the disease had, undoubtedly, started in the subglottic region and had only manifested itself when the lower edge of the cord became involved (Figs. 495, 505).

In this form the mobility of the cord is more frequently affected: of these 13 observations the cord was quite fixed in 6, *i. e.*, in nearly one-half the cases; while in the other 37 chordal cases, the immobility of the cord was only noted in 11, *i. e.*, in about 30 per cent. of the cases. Of these 13 subglottic cases, 7 (a little more than 50 per cent.) died of local recurrence.<sup>11</sup>

**Progress.**—With unchecked progress the growth in time will become large enough to cause stenosis and, once the confines of the endolarynx have been passed, there will be added the symptoms met with in advanced extrinsic cases—aphonia, stenosis, pain, dysphagia, fetor, sepsis, and involvement of the glands.

**Diagnosis.**—Any one-sided affection of a vocal cord in a man of middle age, or over, should arouse suspicion of the possibility of a malignant neoplasm. This suspicion would be increased if a complete investigation excluded syphilis and tubercle. Still, it must always be remembered that malignant disease frequently develops in syphilitic subjects, that it may also attack patients with tubercle (of course, much more rarely, as they are generally young subjects), that evidence of old quiescent tubercle can be found in a majority of the population, that cancer may even attack young subjects, and that females are not exempt from the laryngeal form.

In all cases the diagnosis is assisted by putting the patient on absolute silence for two or three weeks, with complete abstention from tobacco and alcohol. Quite 30 per cent. of the symptoms in these cases are due to overuse and irritation of an affected cord. With rest and temperance these added symptoms will disappear: The local condition will then become well defined and clearer; the time can be employed making investigations of the temperature, chest, and sputum in regard to any active tubercle, or in administering antiluetic treatment. If the condition is a simple one, marked improvement will take place and justify further delay. If it remains stationary, or gets worse, or develops any of the possible appearances already suggested, operation or further watching or a biopsy may be indicated.

There is no risk in this delay if the case is so early as to render the diagnosis difficult. A limited epithelioma on a mobile vocal cord is so localized,

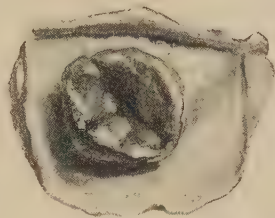


Fig. 505.—Epithelioma of left subglottic region. Operation August 3, 1923. Life size drawing by Mr. — of removed growth one week after it had been pinned out and laid in alcohol. Hence it has shrunk, particularly the ventricular band. No recurrence.

that it is quite justifiable to watch it, if necessary, for two or more months rather than risk operating unnecessarily on a simple or tuberculous larynx. There is much more danger in operating on a tuberculous larynx (mistaking it for a malignant neoplasm) than in delaying for a few months to settle the diagnosis of an early chordal epithelioma.

Tuberculosis of the larynx, in elderly men, not infrequently occurs without any indication of active pulmonary disease. In such cases the deposit of tubercle may be rough, knobby and whitish and it is only when this breaks down into a mouse-nibbled ulceration that the local appearances become more definite.

*Biopsy.*—Some authorities insist on a preliminary microscopic examination of a removed portion of growth before settling a diagnosis and many urge this procedure in all cases before advising operative measures. It should always be carried out when the neoplasm so presents itself that a good-sized portion embracing the deeper strata can be secured. This can be carried out very satisfactorily by using Jackson's punch-forceps and direct laryngoscopy (pp. 744, 996, 1003). If only a small superficial portion is obtainable, the microscopical evidence is likely to be unconfirmatory, as the real neoplasm may be seated some distance below a simple inflammatory or papillomatous surface. In early cases and when the growth is embedded in a cord (Fig. 496, p. 930) or situated in the subglottic area (Fig. 502, p. 932) the removal of an adequate portion may be impossible, or else it might require removal of so much of the cord that we might as well proceed straightway to a more radical removal. Besides, although a removed fragment might yield definite evidence of malignancy, a negative result would not necessarily remove all doubt. In that case the traumatism would obscure the picture and might lead to unfortunate delay in cases which, otherwise, might have been settled by the progress of a little time.

Still another objection is that the microscope is not infallible; for pathologists have pronounced a growth malignant when the subsequent history proved that this was wrong.<sup>12</sup>

**Prognosis.**—Untreated malignant disease of the larynx inevitably ends in death by asphyxia, dysphagia, hemorrhage, sepsis, or intercurrent complication. Intrinsic cancer is, relatively, a very slowly progressive affection. I have seen a suspected case take a whole year before the objective condition warranted a diagnosis. (Case 32, in Laryngofissure Schedule, suspected to have intrinsic laryngeal cancer in April, 1916. As the cord presented a boat-shaped ulcer, patient was sent to a tuberculosis sanatorium. Here no positive evidence of tubercle was found, but the Wassermann reaction was positive. In spite of active antiluetic treatment the laryngeal lesion persisted and at the end of a year, as the infiltration began to show a characteristic "cupping," a laryngofissure was done in April, 1917. Removed mass showed a superficial, limited epithelioma. No recurrence today, after eleven years.)

In a case recorded by Harmon Smith a definite diagnosis of epithelioma was demonstrated by pathological examination of a removed fragment no less than twelve years before the patient succumbed to the disease.<sup>13</sup> In a case of Chevalier Jackson's the microscopic specimen showed epithelioma; the patient then declined operation, and yet survived eight years.<sup>14</sup> Two or three years is not an unusual time for an intrinsic neoplasm to cause no other symptoms beyond hoarseness. This slow evolution appears to depend on the site of the growth. A vocal cord is, anatomically speaking,

the tendon of the thyro-arytenoid muscle; it is feebly vascular and the lymphatics within the larynx do not anastomose with neighboring lymphatics, but form a circuit and network of their own which empties into two small glands on each side.<sup>15</sup>

Once the intrinsic (glottic) cancer has penetrated or climbed over the walls of the voice-box the disease advances as rapidly and as ruthlessly as the extrinsic form.

The subclass of subglottic laryngeal cancer lies, as has been already described, midway between these two main groups as regards seriousness of prognosis. The area immediately below the glottis on each side is (particularly when compared with the tendinous cords) richly supplied with vessels, lymphatics, and glands. A subglottic cancer is, therefore, much more apt to be carried by the lymphatics, through the cricothyroid membrane into the extralaryngeal glands and tissues of the neck.

**Treatment.**—The only radical treatment for an intralaryngeal malignant growth is excision of the tumor with a surrounding area of healthy tissue—when this is possible. Such cases are those where an epithelioma is situated on the margin or upper surface of a vocal cord and has not extended backwards along the cord so as to encroach on the arytenoid body nor, in a forward direction, crossed the anterior commissure. The more defined and superficial the growth, the more it is limited to the central third of the cord and the more freely the cord moves—so much more promising is the prospect of permanent cure by operation.

If the growth has not only reached but extended along the anterior commissure to the other cord, the prognosis is not so good. Laryngofissure and a partial laryngectomy may be successful, but a complete laryngectomy may be required, particularly if impaired mobility, or even fixation of the affected side, shows that the disease has deeply invaded the tissues (Fig. 495). A complete laryngectomy is required if the intrinsic growth has reached the anterior surface of the arytenoid or extended to the ventricular band. If the arytenoid or aryepiglottic fold is invaded the disease is so quickly disseminated from this highly vascular and lymphatic area that even a complete laryngectomy holds out but poor promise of lasting cure.

When the growth is subglottic the prognosis of success by operation—whether that be laryngofissure, partial laryngectomy, or complete laryngectomy—is much less promising than in chordal cancer, although not so gloomy as in extrinsic laryngeal carcinoma. The reasons for this are as follows: The disease, in this situation, causes slight symptoms in its early career; it is considerably advanced before it attracts attention by hoarseness or impaired mobility of the vocal cord or other symptoms; and owing to its site there is a much earlier invasion of the lymphatics and extralaryngeal spread of the disease.

Although the records of recovery are not as satisfactory as in true intrinsic cancer, I have notes of subglottic cancer (and with a fixed cord) remaining free of disease (and with a useful voice) for ten and more years after a laryngofissure. This is the operation which should first be adopted whenever the disease is unilateral and not too extensive. If the mobility on the same side is quite abolished, if the disease extends backward or forward beyond the extremities of the cord, or if it descends lower than the cricoid cartilage—then a complete laryngectomy may have to be considered.

**Results of Operation.**—1. *Endolaryngeal removal*: Well authenticated records are available of lasting freedom from disease by removal *per vias naturales*. I have had one such case: A projecting growth was removed through the mouth and found under the microscope to be malignant. This was promptly followed by a laryngofissure, but I was surprised to find that the tissue removed at this operation showed no evidence of

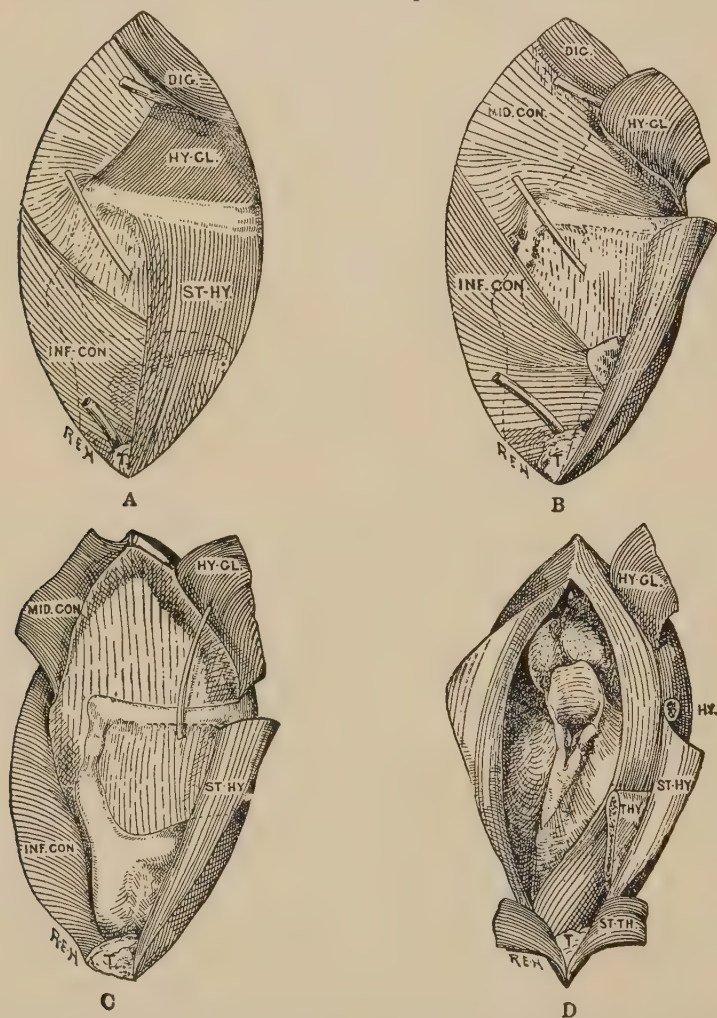


Fig. 506.—Four stages in the operation of transthyroid pharyngotomy. A, Lateral aspect of the pharynx at end of preliminary gland dissection. B, Hyoid muscles reflected. C, Pharyngeal constrictors reflected, aponeurosis of pharynx exposed; great cornu of hyoid and ala of thyroid prepared for removal. D, Lateral wall of pharynx incised, epilaryngeal region exposed. (Wilfred Trotter.)

epithelioma; it had all been removed by the laryngeal forceps. The patient is today well and vigorous, with a strong rough voice, fourteen years after the operation.

But the method is not to be recommended. It is exceptional to find in the larynx malignant neoplasms of this type. Cancer growths are more apt to infiltrate; they are generally, when exposed to the light of day, much more

extensive than had been anticipated; the operation does not carry out the surgical principles of free exposure and as free removal as may be necessary: it leaves a crushed area where healing is slow and recurrence difficult to watch; it tends to disseminate the disease; and it entails the loss of valuable time.

2. *Laryngofissure* secures lasting freedom from disease in at least 80 per cent. of suitable cases. If the disease is limited and on the surface or free edge of a mobile cord, this proportion of lasting cures will be greatly augmented as patients apply earlier on account of chronic hoarseness and the diagnosis is more promptly made. The operation leaves the patient with a free and abundant air-way, a natural and useful voice, and with no disability (except that of not being able to sing) in civil life. The operative death-rate should be nil.

3. *Partial laryngectomy* may be employed in certain cases where the growth has crossed the anterior commissure or extended beyond the sub-

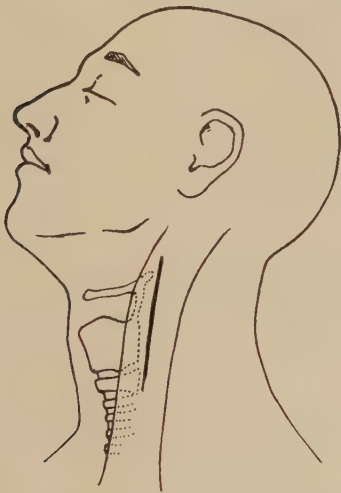


Fig. 507.—The straight, dark line indicates the situation and extent of skin incision for approaching growths around laryngeal orifice.

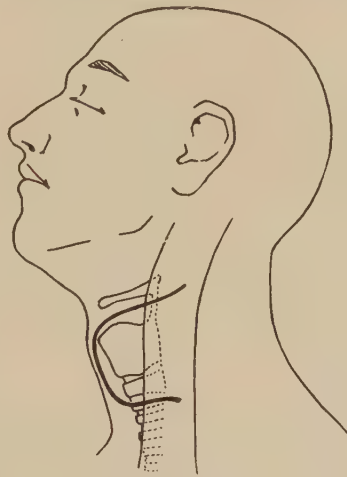


Fig. 508.—The dark, curved line indicates the situation of the incision required to make a flap of skin to be turned in.

Figs. 507, 508.—Partial laryngectomy by the pharyngeal route.

glottic area. It may also be indicated for a local recurrence after laryngofissure. The advantage of this operation is that it spares the posterior part (the signet) of the cricoid ring; hence some buccal air-way and a natural voice may be conserved, even though the reduced glottis may entail a permanent tracheotomy tube. It is sometimes difficult to decide whether this operation should be attempted or whether it is best to proceed at once to a complete laryngectomy.

4. *Complete Laryngectomy*.—INDICATIONS.—This operation is indicated in cases of intrinsic cancer which are unsuitable for either of the preceding operations and yet have not extended beyond the confines of the endolarynx. Such cases will be among those malignant growths which have invaded a whole cord and extended beyond its anterior commissure or its subglottic area, penetrating so deeply as to render immobile the same side of the larynx. If the growth has invaded the arytenoid or

aryepiglottic fold; if it has spread to the epiglottis, to the ventricular band, or up to the margin of the aditus; if the thyroid or cricoid cartilages are attacked (shown by perichondritis, granulations, pseudo-edema, or tender-

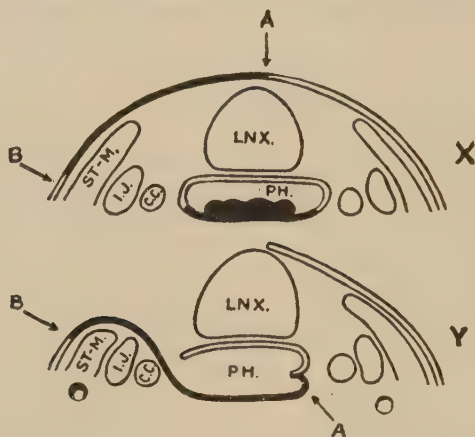


Fig. 509.—To show disposition of skin in operation for removal of hypopharyngeal growth—tumor on posterior wall. *X*, Before removal of tumor; skin-flap and parts to be removed shown in solid black. *Y*, After removal of tumor; skin-flap in place. *A* marks situation of apex of flap. *B* marks situation of base of flap. *LNX*, Larynx. *PH*, Pharynx. *C.C.*, Common carotid. *I.J.*, Internal jugular. *ST-M*, Sternomastoid. (Wilfred Trotter.)

ness and widening of the voice-box) the prospect is less hopeful, but the effort might be attempted. If the disease has extended beyond the confines of the larynx; if perichondritis is evident; if the glands are invaded; if the

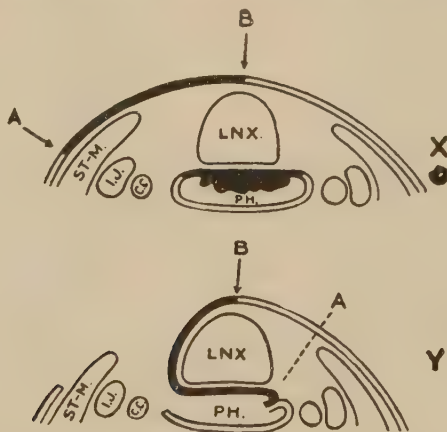


Fig. 510.—Lateral pharyngotomy for approaching tumors in the lower pharynx. Shows disposition of skin-flap in operation for removal of hyperpharyngeal growth—tumors on anterior wall. *X*, Before removal of tumor; skin-flap and parts to be removed shown in solid black. *Y*, After removal of tumor; skin-flap in place. *A* marks situation of apex of flap. *B* marks situation of base of flap. The fascial flap to protect the carotid vessels is not represented. (Wilfred Trotter.)

patient is showing any cachexia, or is exhausted with dysphagia, cough, pains, secretion, and sepsis—operation of any kind is useless and relief must be sought with radium or deep Roentgen-ray therapy.

**DANGERS.**—Ever since Solis-Cohen urged the method of severing the

trachea and detaching it from the larynx, pharynx, and esophagus, so as to secure its orifice to the skin in the middle of the neck, the technic of the operation has been steadily improving.<sup>16</sup> Formerly the death-rate was enormous. Of the first 25 cases operated on by Billroth before 1890, not one was alive at the end of a year.<sup>17</sup> As the cases submitted to it were chiefly of the extrinsic variety, recurrences in patients who survived the operation were frequent. In recent years the technic of the operation has been improved to such an extent that Mackenty has had a series of 31 laryngeotomies (all for intrinsic cancer) without a death following operation.<sup>18</sup>

**RESULTS.**—The results of the operation have also greatly improved, chiefly because it is now employed almost entirely for the intrinsic forms.

Tapia, whose record of total laryngeotomies has, so far, been unsurpassed, acknowledged that they were all for intrinsic cases which had advanced too far to justify a laryngofissure. In Spain the public have not sufficient appreciation of symptoms to present themselves for skilled advice for mere huskiness, nor will they submit to a laryngeal operation before they feel nearly choked. As the education of the public increases, and laryngologists become more expert in diagnosis and the safe conduct of laryngofissure, this operation will become more frequently performed while laryngeotomy must become relatively rare.

**Palliative Treatment.** *Cases Which Decline Operative Measures.*—The cases which present themselves in too advanced a state for any of the above measures and cases in which local recurrence has taken place, demand all the relief we can secure for what is otherwise a distressing and pitiful condition.

Deep Roentgen-ray therapy frequently gives ease to pain and discomfort and, in certain cases, retards the progress and acuteness of the disease.

Radium treatment has produced very promising results in the intrinsic form in cases which declined laryngeotomy and were too extensive for laryngofissure. Neither Roentgen-ray nor radium treatment should be used in cases where surgical measures hold out good prospect of lasting cure.

Tracheotomy, performed as low in the neck as possible, should be carried out under local anesthesia as soon as ever there are any symptoms of stridor.

The care of the mouth and teeth, the use of local antiseptics, and the assuaging of pain need only be referred to.

#### CANCER OF THE LARYNGOPHARYNX (OR HYPOPHARYNX), COMMONLY KNOWN AS EXTRINSIC CANCER OF THE LARYNX

**Definition.**—This group is made up of manifestations of cancer on the margins and posterior surface of the larynx. The neoplasms, particularly in early and operable cases, have few or no laryngeal symptoms. The symptoms vary considerably according to the exact site, but they are decidedly of a pharyngeal character. It would, indeed, be better if we agreed to limit the term "laryngeal cancer" to manifestations of this disease around the glottic space and to give the title "cancer of the hypopharynx" (or "the laryngopharynx") to the various manifestations which we have been accustomed to include in the vague category of "extrinsic cancer of the larynx."

**Situation.**—Extrinsic cancer of the larynx is conveniently studied if we follow the suggestion of Wilfred Trotter and further divide it into two groups, (a) the epilaryngeal, and (b) the hypopharyngeal.<sup>19</sup>

(a) The epilaryngeal type of cancer of the larynx originates in two principal sites: (1) the epiglottis, and (2) the aryepiglottic fold, especially towards its arytenoid extremity.

(b) The hypopharyngeal type—known also as postericoid cancer—originates in the narrow part where the pharynx is attached to the posterior surface of cricoid ring. It embraces two well recognized forms as it originates (1) in the narrow part of the lower pharynx where it is attached to the posterior surface of the cricoid ring (“postericoid cancer”) (Fig. 499), or (2) in the sinus pyriformis (Fig. 501).

**Frequency.**—It was formerly taught that malignant growths in this region were much less common than similar growths in the endolarynx, but this opinion has probably only been formed because it was based on the experience of laryngologists to whom, naturally, patients with laryngeal symptoms would first address themselves.<sup>20</sup> For patients with growths in the laryngopharynx are more likely to apply to the general surgeon and their opinion is that malignant disease in this region is not at all rare. Indeed it is not very much less common here than it is in such favorite situations as the mouth and the esophagus.

These growths in the epilaryngeal region occur chiefly in males who have reached or passed middle age.

In the hypopharyngeal region, those which start in the sinus pyriformis are also most frequent in males. But those which take origin from the pharyngeal wall attached to the cricoid plate are practically limited to females. Not only do they show this striking sex distribution, but among females they may occur at a much earlier age than do the other growths of the laryngopharynx among men. Hence postericoid cancer is not uncommon in the fourth decade of life and not rare between the years of twenty-five and thirty-five. Another very curious fact in its evolution is that many such women give a history of some dysphagia, or of “choking fits,” or of “food going the wrong way,” or of “attacks of relaxed throat” extending over several years.

**Symptoms.**—As already pointed out, these are primarily of a pharyngeal character. In early cases there is no change of the laryngeal voice—no hoarseness or huskiness—although speech may sound a little woolly. This is due to local interference with the distribution of mucus and saliva. There is hemming and hawking; a sensation of discomfort, as of a crumb in the throat; there may be no dysphagia or difficulty at meals and yet discomfort in swallowing saliva. Occasional attacks of catarrh or of subacute laryngitis may occur.

In the hypopharyngeal type the symptoms are chiefly those of dysphagia and much resemble the early cases of malignant disease of the esophagus.

Careful laryngoscopic examination, with the help of cocaine and good illumination, should be directed to the favorite areas referred to. It is particularly important to scrutinize the district during phonation as this brings into relief the base, outline, and movements of the epiglottis and the posterior surface of the aryepiglottic folds.

The hypopharyngeal growths are not visible, by indirect laryngoscopy,

in their very early evolution. But suspicion should be aroused if there is much mucus or frothing in this area behind the cricoid, if the patient has much difficulty in swallowing or hawking this up (when asked to "clear his throat"), and if there is a pool of saliva and mucus in the sinus pyriformis which quickly fills up after being cleared. When further advanced there will be noticed a purplish fulness of the mucosa, which is perhaps pleated or crinkled over the back of the cricoid plate (below the base of the arytenoids) or in the otherwise smooth and even depths of the sinus pyriformis. With a well anesthetized pharynx, a voluntary relaxed patient, and good traction on the tongue, these appearances are still better distinguished during prolonged phonation. Finally, in a more advanced stage, the irregular fungating edge of an epitheliomatous ulcer will be seen in the midline, low down on the posteriocricoid region, or fringing the hollow of the sinus pyriformis. If, in addition, the movement of the arytenoid is impaired or abolished, the diagnosis of malignant growth becomes almost a certainty. This impaired movement is not a good omen as it indicates deep penetration of the growth.

When the disease originates in the sinus pyriformis the glands are apt to be infected early.

With the above symptoms confirmation can be sought on the Roentgen-ray screen while the patient is swallowing barium and water, and by direct pharyngoscopy under a general anesthetic. To the educated finger these growths offer a firm, hard, and even cartilage-like resistance.

A fungating growth—even the margin of it—is enough as basis of a diagnosis of malignant disease, especially if syphilis can be eliminated, in a middle-aged or elderly individual.

More advanced cases need only briefly be referred to. They suffer from constant discomfort, salivation, pain, dysphagia, sloughing, fetor hemorrhage, stridor, enlarged glands, and cachexia. The growth becomes only too visible presenting the cauliflower appearance, deep and sloughy ulceration, hard sensation, and horrible fetor associated with cancerous growths communicating with the mouth. All these symptoms are intensified if there are diseased teeth or gums.

**Diagnosis** is not difficult. The situation and appearance of the growth; the age and sex of the patient; the steady and generally rapid progress of the disease, and the secondary infections hardly require the confirmation of the microscope. In the epipharyngeal group this latter help is readily available. In the hypopharyngeal group it is almost impossible before the disease is so advanced that operative treatment would be useless and the traumatism entailed by removing a portion could do nothing but add to the distress of the patient and the progress of the disease.

**Prognosis.**—Untreated, all these cases end in death. The progress of the disease—occurring in regions rich in vascular and lymphatic supply—is much more rapid than in (intrinsic) laryngeal cancer, and generally runs its course in twelve to eighteen months from the time when symptoms become definite and persistent.

Laryngectomy for extrinsic cancer of the larynx has earned a disastrous reputation. For this manifestation of cancer it has now-a-days been abandoned.

As regards other operative procedure, the most hopeless site is in the sinus pyriformis where the lateral wall of the pharynx, the side of the neck,

and the glands are generally invaded by the time that the patient presents himself or the diagnosis is settled.

At the base of the epiglottis surgical measures hold out little prospect of lasting relief. The disease has generally invaded the vallecula, the base of the tongue, or the side of the pharynx quite early in its evolution. But such cases may be greatly benefited by excising the growth with diathermy, the glands, if present, having been dissected out and the external carotid ligatured at a previous operation.<sup>21</sup>

When the disease is on the free margin of the epiglottis, or on the ary-epiglottic fold and well-defined, or diagnosed while in an early stage on the postericoid region—there is good prospect of prolonging life by removal through a transthyroid lateral pharyngotomy.

**Palliative Measures.**—(See page 939.) In addition many cases in this group will require an early gastrostomy. The section on Cancer of the Esophagus should be consulted.

### OPERATIONS

**Preparation.**—In all the operations to be described the usual preparation and precautions of modern surgery should be insisted on. The tendency to hemorrhage, particularly troublesome in this region, should be reduced by careful attention beforehand to the condition of heart, kidneys, lungs, blood-pressure, and so forth. Alcohol and tobacco should be abandoned for some days previous to operation, and it is of great advantage if the patient has a rest in bed in the hospital, with a reduced diet, for three days beforehand. Very important is attention to the teeth and gums as the chief causes of sepsis in this area. This is demonstrated by the slower progress of the disease and better convalescence after operation in edentulous subjects, and also by the observation that cases often improve remarkably and become operable after the mouth has been cleared.

SIR ST. CLAIR THOMSON.

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## LARYNGOFISSURE (THYROTOMY OR THYROCHONDROTOMY)

This operation would be better described now-a-days as a partial laryngectomy. It has evolved considerably since last century when it was described as simply "splitting the larynx, and removing a vocal cord." Unfortunately, this limited view, with its technic and application, is still too generally accepted, thus interfering with its usefulness and general adoption.

**Indications.**—So far as intrinsic cancer of the larynx is concerned, these are described on a previous page.

The operation, of course, has other fields of usefulness, such as in enchondroma of the larynx,\* or impacted foreign bodies.

**Anesthesia.**—The operation can be carried out under local anesthesia. I have tried it but find no advantage in it. The strain to both patient and surgeon is augmented, the feeling of hurry is inimical to delicate and thorough surgery, and the tendency to secretion and hemorrhage is increased. On the other hand, a general anesthetic, administered through a tracheotomy opening, is, as is well known, one of the safest methods of administration. There is no congestion, respiration is even and regular, tendency to hemorrhage is diminished, the parts are relaxed, and can be manipulated with less trauma and, if local (skin) anesthesia and the intratracheal injection of cocaine are combined with it (as described later on) the degree of narcosis is complete without being profound, recovery of consciousness is rapid and there is no shock. In an experience of 70 cases I have never seen any shock, although I have performed this operation for intrinsic cancer in 7 men over seventy years, in one of eighty years of age, and in a man of sixty-nine with a blood-pressure of 200. All my patients are out of bed the next day with a normal temperature, and many of them are sitting up and reading their newspapers on the evening of the operation.

The skin of the line of incision (*i. e.*, from the hyoid bone to the sternum) is injected with novocaine and adrenaline, or with "eudrenine," at least half an hour before operation. No alkaloid of any kind is injected beforehand and no morphia or other preparation of opium is allowed before or after operation. It is most important not to benumb the sensitiveness of the air-ways or weaken the respiratory center and so interfere with the act of coughing, which Chevalier Jackson so well calls "the watch-dog of the lungs."

As in other operations on the air-ways, chloroform (well diluted with air or oxygen) is to be preferred during induction of narcosis. Intratracheal

\* Thomson, St. Clair: Echondrosis of the Larynx: Two cases successfully treated by laryngo-fissure, Jour. of Laryn., vol. xl, 1925, No. 1, p. 1.

ether is not advisable, as the anesthetist's tube interferes with good surgery, and it is important to avoid the slightest traumatism of the endolarynx.

Before the trachea is opened its wall is stabbed with the needle of a hypodermic syringe and 10 to 20 drops of a  $2\frac{1}{2}$  per cent. solution of cocaine are injected into the lumen of the trachea.\* Five to ten minutes later, the trachea can be opened without any cough or disturbance. For the remainder of the operation the anesthetic is administered through the tracheotomy opening. It is well to commence with chloroform well diluted. Once the trachea has been opened ether is quite satisfactory, and it will be found that a light degree of anesthesia is sufficient. If the operator notifies the anesthetist about ten minutes before he removes the tracheal plug and finishes the operation, it will be found that the patient has recovered consciousness before leaving the operating table, and the effect of the intratracheal cocaine having then passed off he will be able to cough up any mucus or blood.

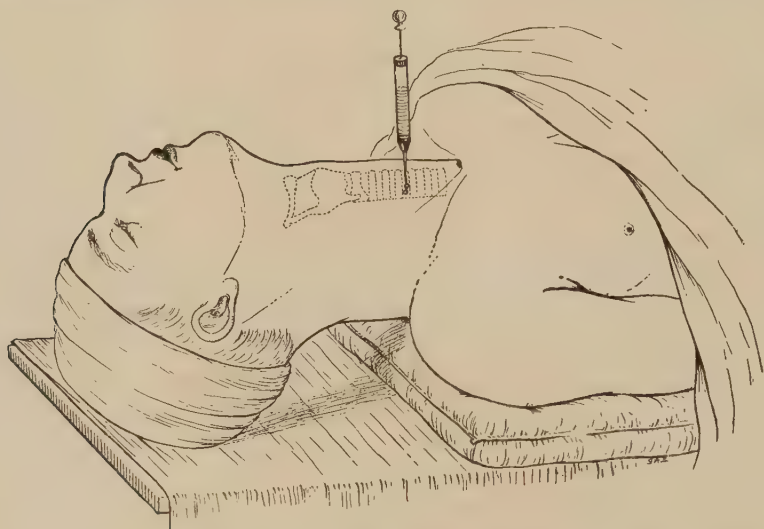


Fig. 511.—Laryngofissure. Semidiagrammatic illustration of correct position (*i. e.*, cushion under shoulders and not under neck) and of the method of anesthetizing the trachea by intratracheal injection of a  $2\frac{1}{2}$  per cent. solution of cocaine.

**Position.**—The patient should be in the tracheotomy position, with shoulders raised on flat pillows and the head well extended. Until the larynx has been opened it is a point of great importance that the midline of the patient's body (chin, midsternal notch, and umbilicus) be exactly maintained in one straight line and that the body is quite flat. If the neck is bent or rotated, or one shoulder higher than the other, the trachea will not be exposed at the center of its convexity and the anterior commissure will not be divided exactly in its center (Fig. 511).

**Incision.**—*Tracheotomy.*—An incision is made in the middle line from the upper border of the hyoid bone to the top of the manubrium sterni (Fig. 512). The usual dissection is made as in a tracheotomy, with the addition that each layer of fascia is divided in its whole extent from the upper border of the thyroid cartilage to the sternum. If the isthmus of the thy-

\* Green, Crosby: *Tr. Amer. Laryngol. Assoc.*, 35, 163, 1913. Thomson, St. Clair: *Tranquil Tracheotomy*, *Amer. Med. Assoc., Trans. Sect. of Laryn.*, 1919, p. 185.

PLATE XVIII



Birkett's case of laryngofissure. The upper illustration shows the condition before operation, the lower one shows it two and a half years later. (Proceedings of American Laryngological Association, 1922.)



roid gland lies over the second, third, or fourth rings of the trachea it is ligatured and divided (Fig. 521).

As soon as the trachea is exposed 10 to 20 drops of a 2½ per cent. solution of cocaine are injected into the lumen of the trachea, as already described, and also through the cricoid thyroid membrane into the cavity of the larynx (Fig. 511). The five to ten minutes allowed for this to act can be well employed in clearing the front of the thyroid cartilage and of the second, third, and fourth tracheal rings, and in arresting every trace of bleeding. Veins should be ligatured as well as arteries and they should be searched for in the retracted flaps or the capsule (if torn) of the thyroid gland. In the few cases I have had of postoperative hemorrhage the blood, in most instances, came from veins in the front of the neck and reached the air-way through the tracheotomy opening.

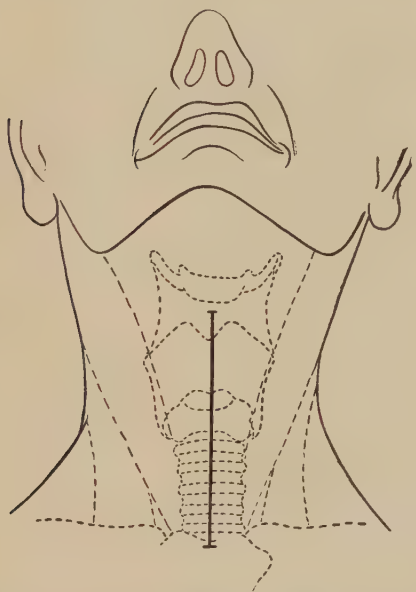


Fig. 512.—Laryngofissure. Site and extent of skin incision.



Fig. 513.—Operation of laryngofissure. Shows, on the left side, how the perichondrium on each side of the thyroid cartilage is reflected before a large portion of the thyroid ala is removed. The dotted lines indicate the extent of tissue removed in a typical operation.

The first ring of the trachea should be spared (in case the growth is found to have a marked subglottic extension and the cricoid may require division), and after the second, third, and fourth have been divided, a fragment is clipped off each divided side, so as to leave an elliptic (ovoid) opening through which a large-sized tracheotomy tube (Durham's lobster-tail pattern) is now inserted, and through this the anesthetic is administered during the rest of the operation. This removal, with a pair of scissors curved on the flat, of portions of the divided tracheal rings on each side, avoids the traumatism and doubling inward of bent or broken portions which is inevitable if a fair-sized tube is forced through a narrow slit. The tube fits the opening more snugly and can be accurately packed around with gauze. There is less danger if the tube is accidentally dislodged or deliberately changed. There is much less likelihood of necrosis of the rings, and the opening in the trachea heals more quickly and evenly.

**Opening the Larynx.**—The next step is opening the larynx by dividing the thyroid cartilage, exactly in the middle line of the body and by an incision which is absolutely perpendicular. If the cut is made on a slant, or obliquely, there is a chance of cutting into the growth on one side, or the healthy cord on the other.

**Dividing the Thyroid Cartilage.**—In women under fifty it is usually possible to divide the cartilage with a conchotome or cutting shears. In most men, particularly over fifty, it is well to avoid splintering the cartilage by commencing the division with a fine-toothed saw (Fig. 518). While the

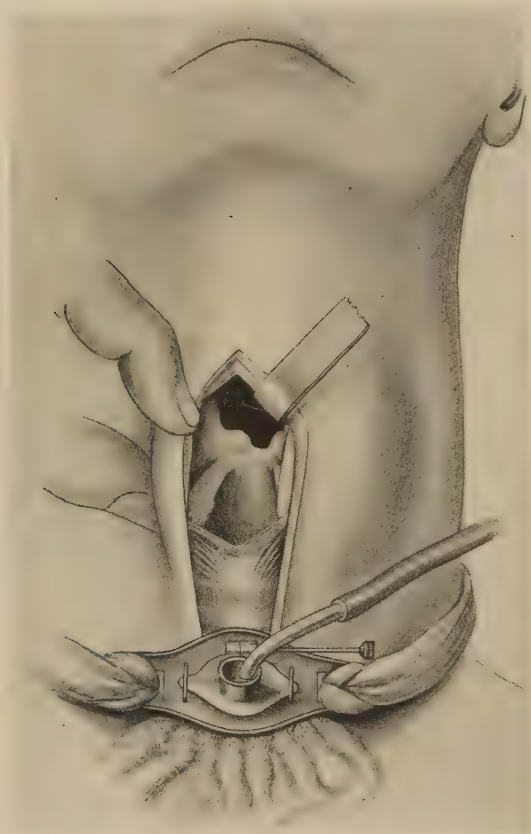


Fig. 514.—Laryngofissure. The tracheotomy tube has been inserted and the larynx split open. By tilting the posterior half of the right ala toward the middle line the affected cord on that side is brought more into view.

larynx is steadied, the saw is held vertically and strictly in the middle line, and short, firm, and gentle strokes are used until a decided gutter has been made, or until it is felt that the cartilage is nearly cut through. The tip of a knife is then inserted vertically, for about half an inch, through the cricothyroid membrane so as to allow of the easy introduction of the lower blade of the cutting pliers (Fig. 520). This is pushed into the lumen of the larynx—inwards and slightly upward—right up to the joint before the blades are closed with one, firm grasp.

**Exposing the Growth.**—The two alæ of the thyroid cartilage are now

held apart or separated with the very convenient thin blades of Killian's nasal speculum (for median rhinoscopy) (Fig. 522). There should be no coughing or bleeding if the technic has been carefully followed. If there is irritation or oozing a piece of ribbon gauze moistened with equal parts of cocaine (10 per cent.) and adrenaline is applied to the posterior wall of the exposed larynx.

Now is the time to confirm the diagnosis and to study the exact site and extension of the growth. It is generally found to be more extensive than it appeared by reflection in the laryngeal mirror. A tuberculous

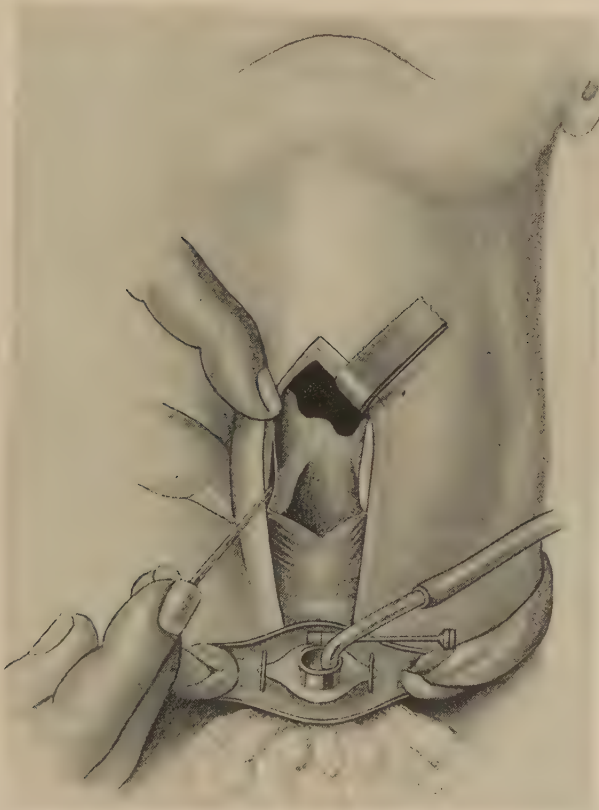


Fig. 515.—Laryngofissure. Shows the affected side displaced toward the middle line. With an elevator the perichondrium is being raised from the inner surface of the thyroid ala.

growth is very soft, while a malignant infiltration is always firm and almost cartilage-like to the touch.

The next step is to thoroughly pack off the lower air-way from any possible descent of blood or mucus (Fig. 517). This can be effected by taking a yard of 2-inch ribbon gauze. A knot, the size of a hazel-nut, is made at one end so that the extremity cannot make its way past the tracheotomy tube. With a short pair of nasal dressing forceps this knotted end is introduced into the lumen of the larynx and, without damaging the growth, is passed downwards until arrested by the convexity of the cannula. On the top of this the ribbon gauze is firmly and evenly packed down, so as to

completely fill the air-way upward to the level of the cricoid cartilage. If the procedure is successfully carried out it will be found at the conclusion of the operation that neither blood nor mucus has penetrated the packing, and that not a drop of blood has descended to the bronchi (Fig. 516).

**Excision of Growth.**—The removal of the growth, with a good free margin, must now be planned according to the conditions met with. The object is to remove the growth intact with a sufficiency of uninvaded tissue around it, to leave a free natural air-way and one free vocal cord, and to carry this out with the least amount of traumatism and without allowing any blood to descend into the bronchi either during or after the operation.

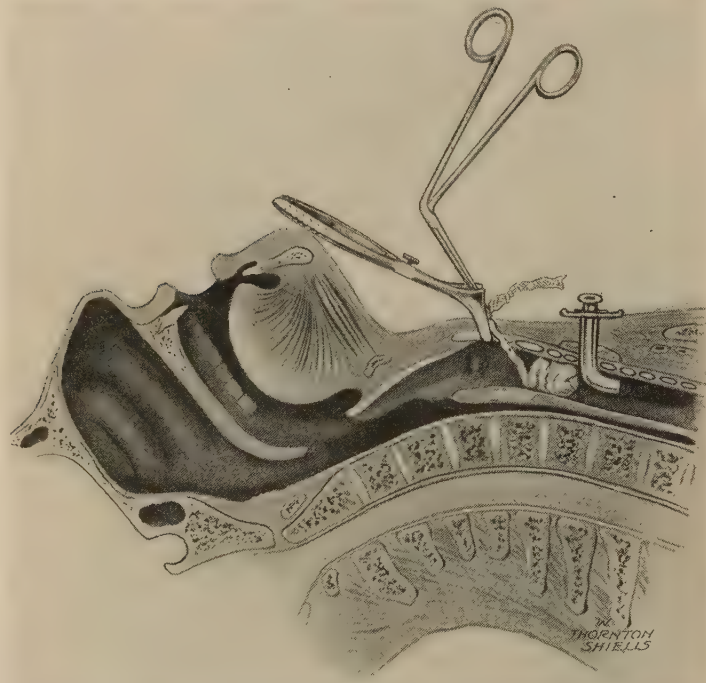


Fig. 516.—Laryngofissure. Shows how the divided thyroid alæ are held apart with a Killian nasal speculum, while knotted ribbon gauze is packed down on the top of the tracheotomy cannula.

In a typical case, *i. e.*, a growth limited to the cord and not extending beyond the anterior commissure in front or up to the arytenoid behind, the procedure is as follows:

Using the detacher we are all accustomed to use in resection of the septum, the perichondrium is raised from the thyroid cartilage on the affected side, starting from its anterior, divided edge, and carrying the separation upwards to the upper border, backwards until the arytenoid cartilage is reached and downwards to the subglottic area below the lower edge of the thyroid cartilage (Fig. 515). Here further progress is limited, as the perichondrium lining the inside of the thyroid welds with the perichondrium coating the outside of the same cartilage to form the cricothyroid membrane. Still it is possible, if required in subglottic growth, to split this membrane (without going through the air-tube into the tissues of the neck) though,

to do so it is sometimes necessary to divide the cricoid cartilage. This difficulty of raising the growth, with its surroundings in the subglottic area, is one of the reasons which renders a neoplasm in this region so much more dangerous than one limited to a cord.

When the perichondrium, bearing all the soft tissues of the endolarynx on the same side, has been raised, the same detacher is used for peeling back the perichondrium from the outer surface of the ala (Fig. 513). Attachments are clipped through above and below and, with a pair of shears at a lateral angle, the greater part of one wing of the thyroid cartilage is clipped off (Fig. 518).

This gives a much more open field for the careful excision of the diseased area with a good margin; it is easier

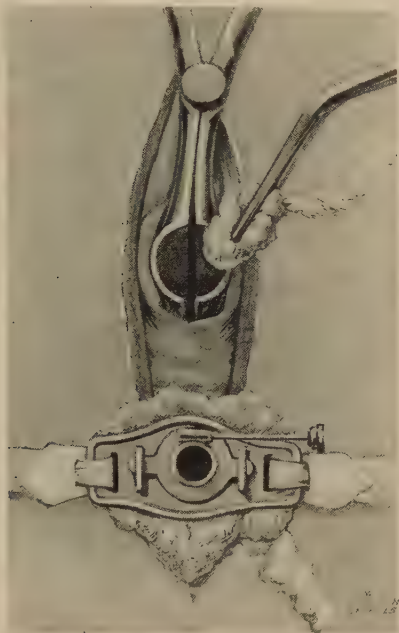


Fig. 517.—Laryngofissure. The tracheotomy tube has been inserted and packed around with gauze; the larynx has been split open and inspected; the thyroid alae are held open by Killian's nasal speculum so as to allow of knotted ribbon gauze being packed down on top of the tracheotomy cannula.

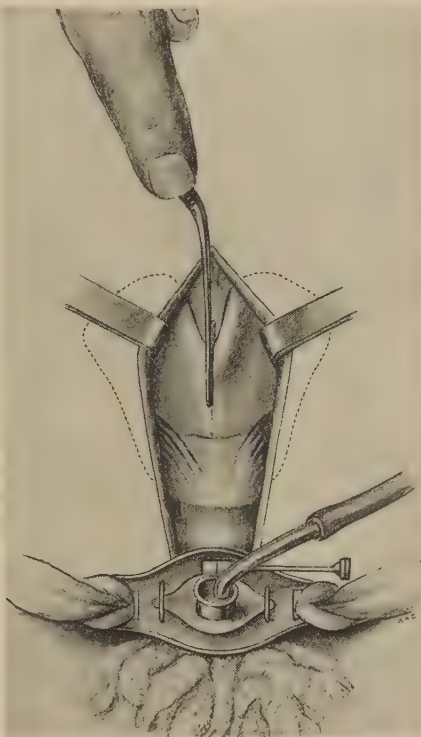


Fig. 518.—Laryngofissure. The tracheotomy tube has been inserted; ribbon gauze has been packed around underneath the shield; the anesthetic is being administered through the tracheotomy cannula. A saw with a bayonet handle is being used to divide the thyroid cartilage exactly in the middle line.

to arrest any hemorrhage; healing is more rapid than in my earlier cases when it took weeks and months for the denuded inner surface of thyroid cartilage to granulate over; the subsequent glottic space is more ample; granulation tissue gives less trouble; it has never been followed by any necrosis of cartilage and the voice is stronger. It has been carried out in the last 42 cases during the past ten years, and I remain well satisfied with it.

The undermined endolarynx on the affected side, now that there is no rigid cartilage behind it, is easily manipulated. Seizing it in front with a

pair of forceps, or a Luc's nasal forceps, it is divided above with a pair of straight or slightly curved scissors by a cut which passes backward, through the upper part of the ventricular band, to the aryepiglottic fold. Below, with the same pair of scissors, we make a cut directly backward, hugging the concavity of the cricoid, to just below the base of the arytenoid. Finally, with a sharp pair of scissors, curved on the flat to nearly a right angle—and while the mass of endolarynx with the neoplasm in its center is held forward and toward the opposite side—the last attachment is severed posteriorly by cutting upward in front of the arytenoid (Fig. 519).

The removed mass (Figs. 498, 502) should show the whole length of the cord from the anterior commissure backwards to the vocal process of the arytenoid cartilage, which should always be visible in a successful removal.



Fig. 519.—Laryngofissure. The diseased area, with a good free margin, has been raised on its perichondrium from the thyroid cartilage and divided above and below. With curved scissors it is now being detached posteriorly by cutting through the vocal process of the arytenoid with curved scissors.

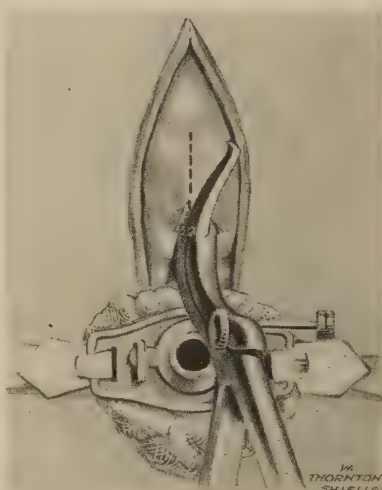


Fig. 520.—Laryngofissure. The tracheotomy tube has been introduced and packed around with gauze. A 2½ per cent. solution of cocaine has been injected into the lumen of the trachea and larynx. The picture shows the thyroid cartilage being divided through the anterior commissure with the cutting pliers.

It should show an intact ventricle of Morgagni and the greater portion of the ventricular band above, while below it should include the uninvaded subglottic area. If these conditions have not been satisfied, further portions of suspected tissue must be exposed and clipped away, but with experience it is possible, as it is always desirable, to remove everything necessary in one mass.

Hemorrhage is seldom troublesome beyond some oozing from the muscles around the arytenoid. With pressure, clamping, and occasionally ligaturing all oozing should cease before the gauze-ribbon plug is removed and the laryngeal opening closed by deep catgut sutures which unite the perichondrium over the remaining thyroid ala to the perichondrium which was peeled from the outer side of the opposite ala. Over this the fascia and muscles are drawn together and, finally, the whole skin wound—from the

hyoid level down to the cannula and from below this to the sternum—is closed with silkworm or horsehair.

While this is being done the anesthetic is discontinued and the end of the operating table gradually raised to accustom the patient to the sitting posture. The effect of the cocaine on the tracheal mucosa will have worn off and the patient should have recovered his cough-reflex before he leaves the table.

In most cases the tracheotomy cannula might be removed at once, but, after experimenting with this for several years, I have come to the conclusion that it is a wise precaution to leave it in place for some hours. There



Fig. 521.—Laryngofissure. The front of the larynx and trachea has been exposed, and the thyroid isthmus divided and held aside by catch forceps. The black line indicates the situation for incising the windpipe in median tracheotomy.

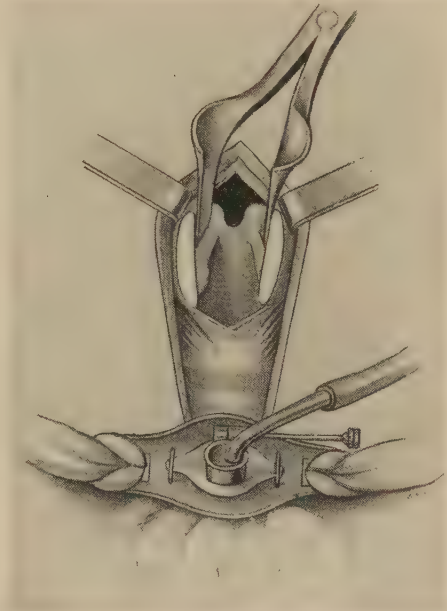


Fig. 522.—Laryngofissure. The tracheotomy tube is in position. The larynx has been split and the two thyroid alæ are held apart by a Killian median speculum so as to view the interior of the larynx and examine the extent of the growth. A small neoplasm is indicated on the anterior third of the right cord.

is no objection to this retention. It guarantees respiration should the larynx fill up with a clot; it makes the effort to expel clots of mucus more easy and effective; the patient is less anxious and, if there should be any intralaryngeal bleeding, it is easy—with the cannula already in place—to divide the stitches above it and plug the larynx.

The cannula is generally removed the same evening or, at latest, within twenty-four hours.

**After-treatment.**—The patient is put back to bed in a sitting posture and kept warm but with the windows freely open. No dressing is required over the skin wound, which heals by first intention if left alone, just like

a shaving cut. Over the cannula is placed some loose gauze with which the nurse seizes any mucus just as it is expelled.

The old-fashioned "steam-tent," "a uniform temperature" "rectal feeding," and such like fetishes, have long been abandoned. No opiate should be given. If restless, the patient can have, per rectum, some bromide and aspirin.

No swallowing of food or liquid should be attempted for eight hours. Some sterilized water can then be given in sips and, if successful, continued until next morning when the patient resumes ordinary diet and sits up out of bed.

If the first efforts with the sterilized water are not successful, owing to some paresthesia which is common after tracheotomy, successive efforts

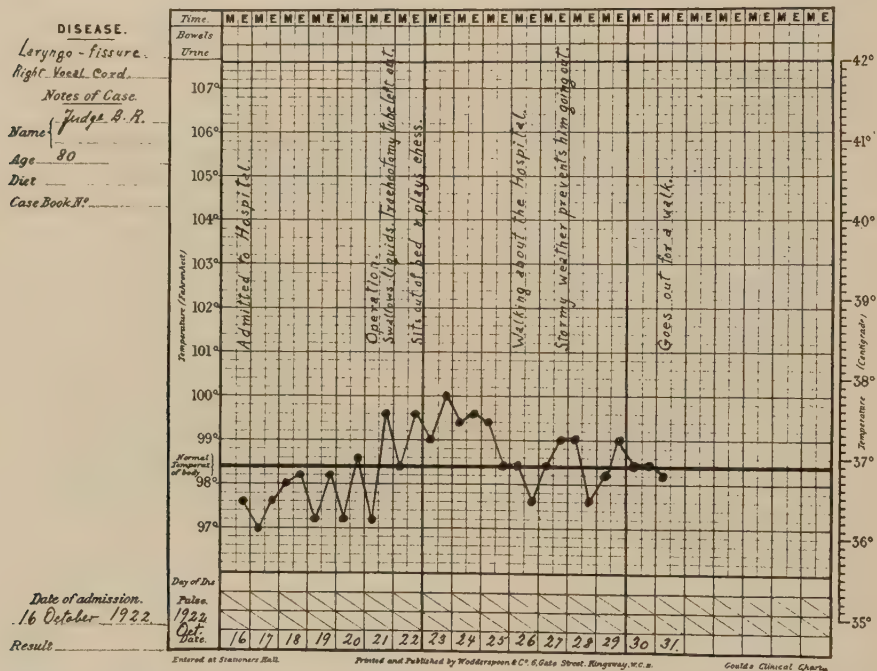


Fig. 523.—Operation of laryngofissure. Sample of an average temperature chart from a patient, aged eighty years, who is well and active, with a good voice and free airway, six years after operation.

are made every two hours. The patient is often nervous and swallows "the wrong way." He should be encouraged to persevere, in the sitting position, with the head forward and dropping the chin with each sip. It is many years since I had to use a feeding-tube in this operation.

The progress of an ordinary case is seen by referring to a typical temperature chart (Fig. 523). As a rule, the patient is walking out of doors within a week, and goes to the country within ten days or three weeks at the latest.

**The Voice.**—For these ten days the patient should remain silent, and then he should begin to use his voice, without effort, whispering for the first two or three months. A strong, rough, but serviceable voice is then developed, as the scar tissue on the operated side evolves a new cord which is almost indistinguishable from the healthy one—except that it is immobile.

During the convalescence—in four to eight weeks after the operation—a granuloma may form over the new (cicatricial) cord or in the anterior commissure. It is distinguishable from a recurrence by its more limited area, by being soft and gelatinous, and by its tendency to project instead of infiltrate. In most cases it will disappear in a few weeks, or it can easily be removed through the mouth, by the indirect method.

**Variations.**—*No Tracheotomy.*—There is nothing gained by trying to dispense with a tracheotomy. On the contrary, this avoidance only interferes with completeness of exposure and removal, while it seriously exposes the patient to the chief danger of this operation, viz., the descent of blood into the bronchi. Those who have tried to dispense with it report that “tracheotomy is not done unless it is necessary to pack the larynx for hemorrhage.” In such cases the mischief may have already been done: “packing the larynx” is a rough measure which is rarely required if, with the aid of a tracheotomy, bleeding is largely avoided and completely controlled by the technic I have described.

“Window resection” need not be discussed, chiefly for the same reasons.

**Postoperative Radium Treatment.**—Until this therapeutic measure is better established, it is well to avoid it. In a successful operation it is an unnecessary procedure. There is grave danger of causing necrosis of the laryngeal cartilages. It may, for all we know, devitalize healthy tissue and, even if it does not later conduce to the local development of fresh cancer deposit, it would certainly not help a second operation which (as I have shown) may be quite successful.

In fact some instances recorded of relatively early metastasis in a limited chordal cancer appear to me to be attributable to radiation.

**Recurrence.**—Experience fully confirms the observation first made by Semon that the first year after operation is the anxious one. During this time the larynx should be regularly inspected once a month, under the excuse that we are noting that the new voice is being exercised sufficiently and not too much. If this year of probation passes without a set-back, there is little anxiety for the future. A regrowth during this time should not be scheduled as a “recurrence,” it is really indicative of incomplete removal and shows that there was an error of technic or that the original disease was unsuitable for an ordinary laryngofissure and should have been, from the first, treated with a more extended operation or even a complete laryngectomy.

Still this monthly inspection, in the event of reappearance of the disease, will enable us to intervene a second time and more successfully. Moure has performed a second laryngofissure (within a year) with lasting good results.\* In one of my own cases, where a laryngofissure had to be followed within a year with a hemilaryngectomy, the patient is today well and free from recurrence eight and one-half years later.

**Local and Late Recurrence.**—“After many years patients may have a reappearance of cancer at or near the site from which a laryngeal cancer has been removed.”† My own independent observations entirely confirm this curious phase, which first struck me many years ago. My very first laryngofissure survived three years to die of a cancer of the lingual tonsil on the op-

\* Moure: Rev. Hebdomadaire de Laryngologie, xviii, ii, 1898, No. 43, p. 1265 and Soc. Française de Laryngologie, Mai, 1898.

† Jackson, Chevalier: Xe Congrès Intern. d'Otol., 1922, 19-22, Juillet.

posite side. Another case of cancer of the aryepiglottic fold was operated through a lateral pharyngotomy (*q. v.*) and survived seven and one-half years to die of cancer of the pharyngo-epiglottic fold on the opposite side. Not only may a fresh growth appear in the larynx on the side which has been operated and free for years, but a new-growth on the opposite cord (without any immediate anatomical connection) may appear after many years. One of my cases, a lady aged forty-six, had an epithelioma of one cord removed by laryngofissure and remained free for seven years. Then a malignant growth started on the opposite cord. A second laryngofissure was performed and she remains well five years afterward.

The longest interval appears to be the case of H. Tilley, where one side of a larynx remained free, after laryngofissure for epithelioma, for seventeen years, when a new growth appeared on the opposite cord.\*

Even a complete laryngectomy will not protect against this tendency to local recurrence. I performed a laryngofissure for epithelioma, and within

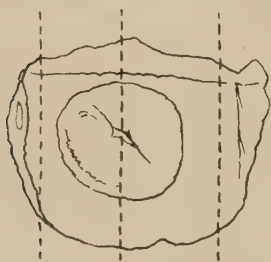


Fig. 524.—Laryngofissure for epithelioma of left subglottic area. Outline diagram indicates three principal sections made for microscopical investigation of the extent of growth. The patient, a female, is alive and well, with no recurrence, five and a half years after operation.

a few months the larynx filled up with what I took to be malignant disease. After consultation with Sir Henry Butlin I excised the entire larynx, but, to our surprise, no cancer could be discovered in the removed organ! Yet this patient, seven years later, died of malignant disease in the glands of the neck!

**Results.**—In all cases where a malignant growth is situated on a vocal cord and has not invaded the arytenoid posteriorly or crossed the anterior commissure, operation by laryngofissure should be free from any operative mortality and it should secure lasting freedom from disease, while it assures a free, natural air-way, a strong voice, and no social disability. Among my patients are physicians, clergymen, and lawyers who have been able,

for years, to gain their livelihood by their profession. Laboring men, being able to close their glottis and fix their chest muscles, continue heavy manual work.

Statistics show that this lasting cure is obtainable in 80 per cent. of all cases. This lower rate is because in some instances it was decided to make a first effort at cure by this operation rather than have recourse at once to the mutilating one of complete laryngectomy. Also, in some cases it was impossible to recognize the extensive subglottic growth, or even the subglottic origin, before exposing the endolarynx.

Besides, success has also attended this operation even when it had extended across the anterior commissure or originated in the subglottic area, although recurrence was much more frequent in the latter group than in the pure chordal cases. Figure 505 shows the excised tissue in a case where the laryngeal appearance was only that of a lightly invaded cord whereas the main growth was entirely subglottic and only the edge of it reached the cord. In this case the pathologist reported that there was a free margin of healthy tissue in front, behind, above, below, and in the depths—although the cancer cells passed down to and among the muscle-planes. Five and a half years have

\* Tilley, H.: Proc. Roy. Soc. Med. Laryngol. Sect., May, 1925.

passed and the patient (a physician's widow, No. 61 in schedule) is in good health, with a useful voice and free from any recurrence. In another case the vocal cord was fixed and the growth largely subglottic. It had penetrated so deeply that the inner surface of the thyroid ala—removed at the operation—was eroded. Yet thirteen years have passed; the patient is well and free from recurrence, and with a voice so strong that he is able to address public meetings in the open air.

**Microscopical Examination.**—Figure 524 shows how all excised growths are cut for examination under the microscope, so as to determine if excision is wide of the disease in all directions. In two of my earlier cases, where the pathologist reported that the incisions, at one point, must have passed through cancer growth I repeated the laryngofissure ten days later, with an enduring result.

### PARTIAL LARYNGECTOMY (BY A PHARYNGOTOMY ROUTE)

**Indications.**—The operation known as a hemilaryngectomy has fallen into disrepute, for the difficulty in guarding the air-passages from infection by the descent of blood and mucus is much greater than with laryngofissure or with a complete laryngectomy.

But, as described elsewhere, there are a certain number of cases of extrinsic cancer which can be satisfactorily dealt with, if approached from the side of the neck and through the pharynx. This is also a possible route for carrying out a partial laryngectomy in cases of local recurrence of disease after a laryngofissure.

**Anesthesia.**—This should be a combination of general narcosis with local anesthesia.

**Operation.**—*Tracheotomy* is the first and most necessary step. The skin incisions, and the subsequent steps, will be varied somewhat, according as the operation is employed for a growth in the epiglottis on the aryepiglottic fold, the laryngopharynx, the endolarynx, or for a recurrence. The following is, therefore, only a general outline of the technic.

**Skin Incisions.**—*Exposing the Pharynx.*—One incision is made along the inner border of the sternomastoid and another is carried forward from this at a right angle (Fig. 507). Part of the anterior triangle of the neck is exposed and the glands and fat are cleared from it. The internal jugular may require resecting if glands are adherent to it. The larynx and pharynx can then be displaced inward, while the carotid vessels are easily retracted outward to allow of the sternomastoid and deep fascia being pulled forward, over them, and then stitched to the prevertebral muscles, so as to act as a barrage and thus shut off and protect the carotid region from septic infection and secondary hemorrhage.

The *thyroid ala is exposed* and its perichondrial covering is reflected, with the attachments to it of the middle and inferior constrictor. It is now possible to pass the periosteum detacher inside the denuded thyroid ala, so as to separate the perichondrium lining from its inner surface and push this inwards with all the soft tissues of the endolarynx on the same side. The posterior two-thirds of the denuded ala are then clipped away.

The *pharynx is next opened* by a longitudinal incision; the cut edges are held apart; the glottis is plugged with ribbon gauze, and a

limited growth on the epiglottis or aryepiglottic fold can be completely exposed and removed (Fig. 506).

The *pharyngeal opening is closed* with mattress-sutures of catgut. The skin incision is closed except for some gauze packing.

A feeding-tube is required until the power of swallowing has been regained.

The tracheotomy tube should be retained for seven to ten days.

When a *postcricoid growth* is well defined and limited, it can be dealt with by an operation planned on the above lines. In such a case the defect in the pharyngeal wall must be restored by turning in a flap of skin, as indicated in Figs. 509, 510.

When the postcricoid growth has infected the opposite (posterior pharyngeal) wall, a complete segment of the pharyngo-esophageal tube may require excision and replacement by an annulus of skin.

The patient is fed for some weeks through the fistula in the side of the neck. This opening is closed by freeing and turning inwards the base of the skin flap, and the upper and lower free edges of this skin "tube" are finally sutured, above to the free edge of the pharyngeal wall and, below, to the area above the esophageal ostium.

The defect on the surface of the neck, left by the inversion of this piece of skin (and its conversion into part of the food tract) is closed by sutures or, if need be, by a plastic operation.

When **partial laryngectomy** is indicated, the above route by a lateral pharyngotomy, and removal of one thyroid ala, will give a satisfactory approach. The laryngeal lumen is preserved by turning in, from the neck, a flap of skin. This not only protects the trachea from sepsis during healing but also serves to form a new vocal cord.

SIR ST. CLAIR THOMSON.

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## TOTAL LARYNGECTOMY

A complete removal of the larynx may be performed by the one-stage operation or by the two-stage operation. Some surgeons, including the author, prefer the one-stage operation because if a previous tracheotomy has not been necessary for the relief of laryngeal stenosis, the field of operation can be rendered more aseptic, thereby insuring a better chance of primary union, permitting a better handling of the tracheal stump, also there is likely to be less shock to the nervous system. Those who advocate the two-stage operation feel that there is less danger of mediastinal infection.

**Preparation of the Patient.**—The patient should be admitted to the hospital at least three days before the operation. A complete physical

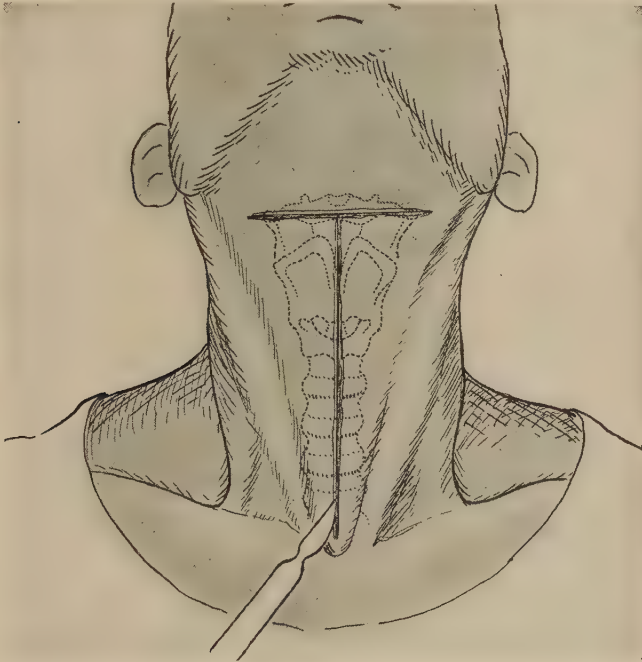


Fig. 525.—Skin incision.

examination should be made by an accomplished internist, this to include a complete chemical and serological blood examination and roentgenogram studies of the chest. Should any condition be detected which would contraindicate a prolonged surgical procedure and should its correction be not possible, laryngectomy would be ill-advised. Careful inspection of the nose, throat, and ears should be made to exclude any chronic infections in the paranasal sinuses, tonsils, and middle ear, the presence of which would prolong convalescence and might lead to serious complications. Infected teeth should be removed and dental hygiene instituted by an expert dentist. Diet is restricted. Free elimination by the bowels and kidneys is essential.

**Armamentarium.**—The instruments which the author has found useful are as follows: Head-mirror or a good head-light, sand-bag, two sharp

scalpels, a generous number of hemostatic forceps, thyroid clamps, Trousseau's dilator, probe, grooved director, blunt and sharp retractors, straight and curved Mayo's scissors, tenaculum forceps, large and small curved needles for suturing the skin and mucous membrane, needle-holders, Allis' dissector, laryngectomy tubes, iodoform and vaselized gauze, suction apparatus, rubber tubing 2 feet long and of sufficient size to fit snugly into the trachea, nasal feeding-tube, bronchoscope, forceps and battery, plain and rat-tooth tissue forceps, submucous elevator and Brunning's septal forceps, catgut, silkworm-gut, and silk sutures.

**Anesthetic.**—Both local and general anesthesia have their advocates. A combination of the two is preferred by some. To determine the kind of anesthetic to be used and the method of administering, the operator should consider the temperament and general condition of the patient,

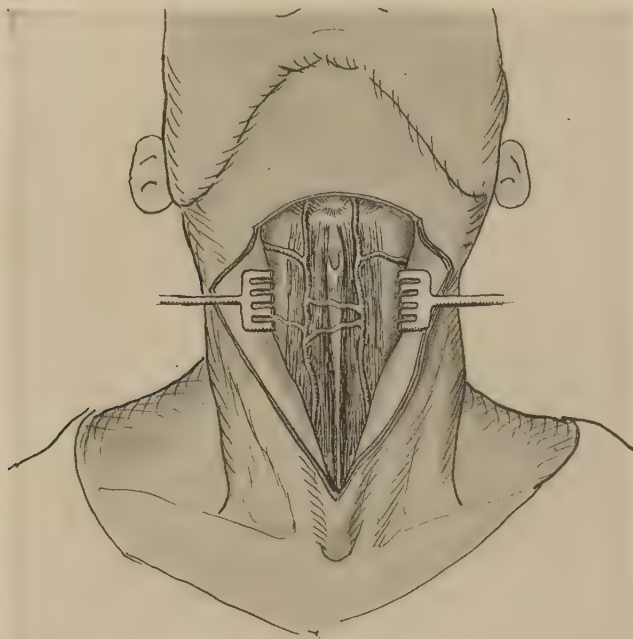


Fig. 526.—Skin, superficial fascia, and platysma are cut or retracted, showing sternohyoid muscles, the anterior jugular veins with their communicating branches.

and the experience of the anesthetist. As often quoted, "The best anesthetic is a good anesthetist." The author prefers rectal anesthesia by the ether and oil method, if administered by an experienced anesthetist. By this method less ether is required and the ill-effects, which often follow other forms of anesthesia, are noticeably absent. Ether anesthesia, by the drop method, is perhaps the safest and the one most frequently used. Opium and its derivatives should not be administered, as the cough reflex is a great aid in the expulsion of foreign secretions from the trachea and larynx.

**Operative Technic for the One-stage Operation.**—The following technic, being a modification of the Glück and MacKenty operations, has been employed by the author in seventy total laryngectomies to date with very satisfactory results.



Fig. 527.—Muscles and soft tissues retracted, exposing larynx and thyroid gland.

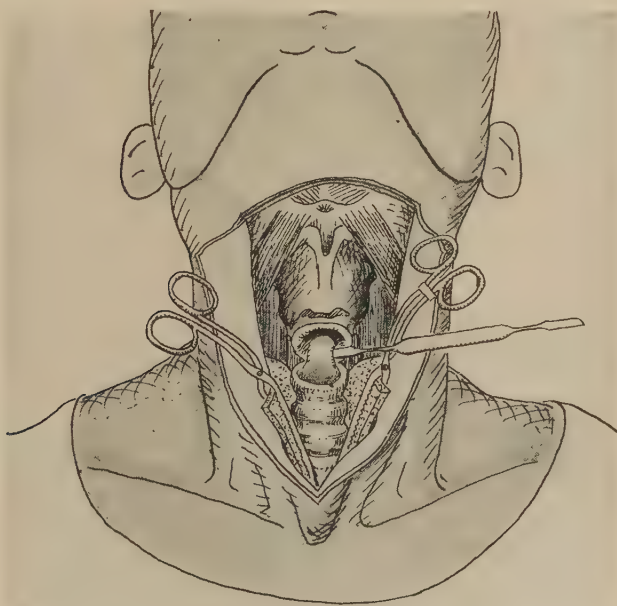


Fig. 528.—Thyroid isthmus clamped and cut, exposing trachea. Mucous membrane flap dissected from the surface of cricoid cartilage.

The patient is placed in a recumbent position on the operating table with the head slightly lower than the body. The shoulders are elevated by means of a sand-bag, so that the head may be fully extended, thereby

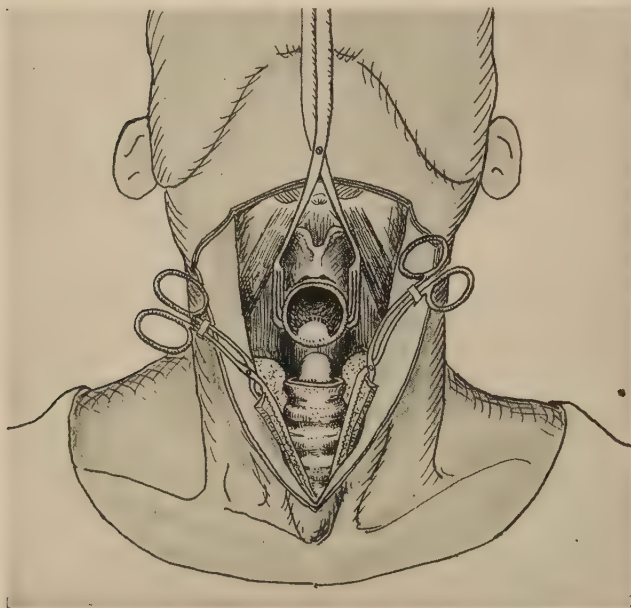


Fig. 529.—Larynx grasped with tenaculum forceps, lifted upward, and completely severed from the trachea.

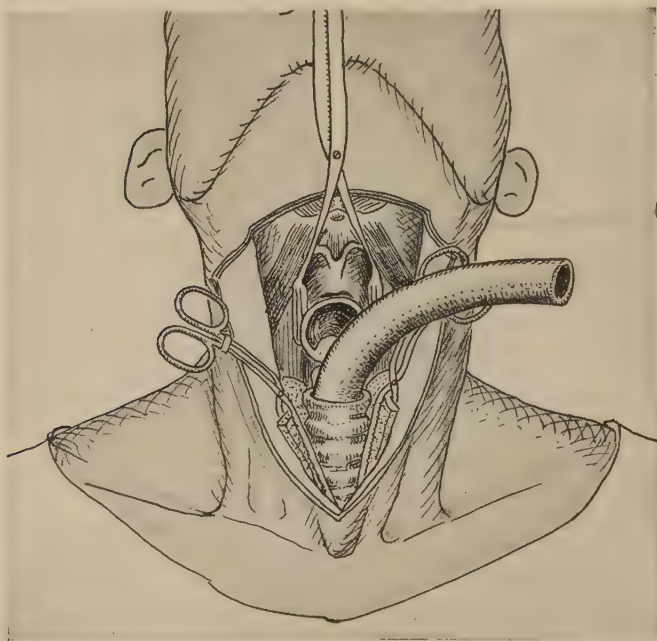


Fig. 530.—Rubber tube inserted into trachea.

bringing the front of the neck into prominence. The operator stands on the right side of the patient, the first assistant on the left side, and second assistant at the head.

A median incision is made beginning at the hyoid bone and extending to the suprasternal notch. A transverse incision is made at the upper end of the median incision forming the letter T, as shown in Fig. 525. The skin, superficial fascia, and platysma are dissected free, exposing the sternohyoid muscles and superficial structures of the larynx. The anterior jugular veins, with their communicating branches, are grasped and tied to prevent severe bleeding (Fig. 526). The sternohyoid muscles are severed at their hyoid attachments or retracted laterally. The sternothyroid muscles are divided at their thyroid attachments and retracted, exposing the thyroid gland. The thyroid isthmus is clamped and divided, exposing the trachea, as shown in Figs. 527, 528. The larynx is next freed from its remaining muscular attachments by careful dissection and all bleeding vessels grasped with hemostatic forceps.

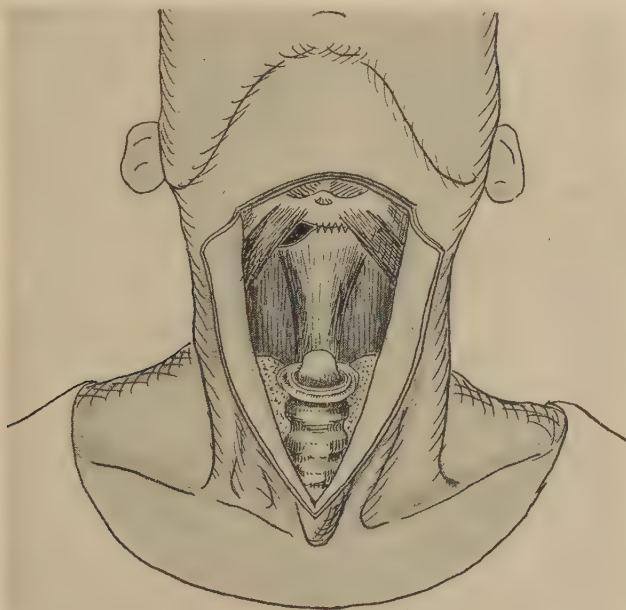


Fig. 531.—Larynx completely removed. Pharyngeal opening partially closed.

The trachea is now opened by an incision between the first ring of the trachea and the cricoid cartilage extending only half way through the trachea, so that a tongue-shaped flap of mucous membrane, about 1 inch long, may be dissected from the posterior and inner surface of the cricoid cartilage. The base of the flap is continuous with the mucous membrane of the trachea. The free end is subsequently stitched to the skin edges for making a better tracheal opening and acts as a barrier to secretions entering the trachea during the convalescent period. This, of course, is omitted if the disease extends as far down as the inner surface of the cricoid cartilage. The larynx is then grasped with a tenaculum forceps, pulled upward, and completely severed from the trachea (Fig. 529). A rubber tube is inserted into the trachea, as a protection against the insufflation of blood and as an aid to the anesthetist (Fig. 530). The larynx is dissected free from the esophagus from below upward to a point well behind the arytenoids.

It is then returned to its normal position, and an opening is made in the hypopharynx through the thyrohyoid membrane between the hyoid bone and the attachment of the epiglottis. Through this opening the entire buccal cavity is packed with iodoform gauze. If by careful inspection and palpation the growth is found to be entirely intrinsic, the larynx should be carefully removed with the view of conserving as much mucous membrane as possible. If there should be evidence of extrinsic involvement, the incision should extend well beyond the limits of its involvement, even to the extent of removing a large portion of the cervical esophagus, lateral wall of the pharynx, and base of the tongue. All bleeding vessels should be carefully tied. A Rehfuß feeding-tube should be introduced through the most patulous side of the nose to the stomach, and the pharyngeal opening closed by two rows of sutures, using No. 0 catgut (Fig. 531). The

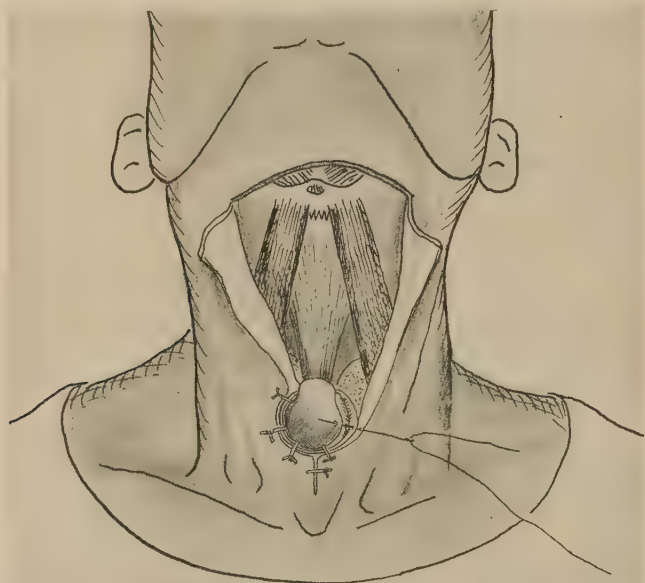


Fig. 532.—First ring of trachea removed submucously. Trachea sutured to skin.

tracheal stump is now attached to the skin surface by interrupted silk sutures. To make the union more secure and complete all fat is removed from under the skin edges on both sides, and the first ring of the trachea is removed submucously (Fig. 532).

The sternohyoid muscles are sutured together in front of the esophagus. Four open rubber-tube drains are introduced: two above; one on each side of the neck at the extreme end of the horizontal incision, two at the lower part of the incision at each side of the tracheal stump (Fig. 533). The horizontal incision above is completely closed. One mattress-suture is placed in the midline incision above and one just above the tracheal opening. The midportion is left open for drainage and inspection. A No. 8 laryngectomy tube is inserted into the tracheal opening and the wound dressed with moist bichloride dressings (Fig. 534).

**Postoperative Treatment.**—A competent nurse, especially trained in the care of laryngectomized patients, should be in constant attendance day and night for the first ten days. Drugs which inhibit the cough reflex should not be given. A substantial well-working suction apparatus should be easily accessible at all times. A short bronchoscope with extra lamps, small battery, and suitable grasping forceps are indispensable when plugs of dried blood and mucus are formed at the carina, corking up both bronchi. There should be placed near the bed a tray containing the following sterilized instruments: scissors, extra laryngectomy tube, dressing forceps, probe, metal applicators, curved Kelly hemostats, and a soft-rubber catheter. Sterile dressings, gloves, gown, and salt solution should be in constant readiness for the surgeon. The inner tube is removed and cleaned as often as necessary by the nurse. The entire tube and external dressings should be changed at least twice daily by the surgeon or a well-trained assistant.

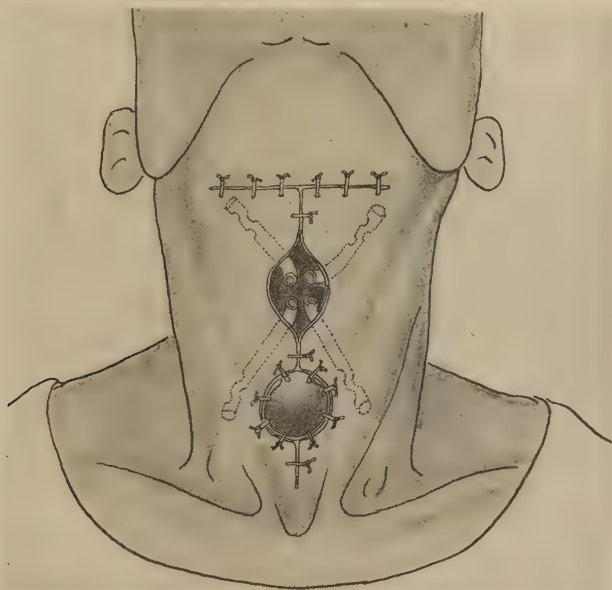


Fig. 533.—Drainage-tubes in position.

The wound must be kept clean. The patient should be propped up in bed the day after operation, and, if the condition permits, should be allowed to sit in a reclining chair on the third day. Care should be taken that the strength of a weak patient should not be overtaxed. The room should be well ventilated.

**Nourishment.**—The patient is cautioned against any attempt to swallow for the first few days. The saliva and secretions are expectorated into a basin, wiped from the mouth with gauze, or removed by suction. The mouth and teeth are cleansed several times daily by hot solutions of liquor antisepticus (1 : 3). For the first twelve hours following the operation water alone is given, 12 ounces every two hours. This is followed by concentrated liquid food mixtures every three hours, the amount and character depending upon the digestive powers of the patient, which have been quite variable in the author's experience. The mixtures which are

giving the best results in the author's experience are: oatmeal gruel, 4 ounces; cream, 2 ounces; whole milk, 4 ounces; one egg, and milk to equal 10 ounces; water, 4 ounces; beef broth, 8 ounces; butter, 2 squares; gelatine, 2 tablespoonfuls; water, 4 ounces; alternating with fruit and vegetable juices and whole milk. After each feeding a few ounces of water are passed through the tube to prevent it from becoming closed. The bowels and kidneys should be kept active.

**Complications.**—*Pneumonia*.—The trachea should be kept free from septic blood-clots and wound secretions by means of suction, the chief causes of this serious complication in laryngectomized patients.

*Hemorrhage*.—Postoperative hemorrhage should not occur if the operator is careful to see that all spurting vessels are securely tied. Secondary hemorrhage may occur from sloughing; should the vessel be large, death may result in a few minutes.

*Mediastinitis* as a complication has been quite rare since the advent of improved technic and better drainage.

*Hiccough* can be relieved by changing the position of the feeding tube.

Serious and alarming *dyspnea* may result from inspissated blood and mucus blocking the trachea or bronchi. A bronchoscope should always be in readiness for its detection and correction.

**Exceptional Conditions.**—In the author's opinion, many cases of laryngeal cancer with extrinsic involvement are operable. When operable a modification of the described technic is essential. It may be necessary to remove a portion of the base of the tongue, part of the pharyngeal wall, esophagus, and lateral structures of the neck. It is possible to remove in such an operation, part of the common carotid artery, internal jugular vein, and pneumogastric nerve of one side together with the adjacent structures. If a portion of the



Fig. 534.—Showing Rehffuss feeding-tube inserted through the nose to the stomach and method of applying dressings around the tracheotomy tube.

pharynx and esophagus is removed so that it cannot be united over the feeding tube, a permanent pharyngo-esophageal fistula in the neck is made by sewing the mucous membrane to the skin. This is subsequently closed by a plastic surgery.

**Laryngectomy in Two Stages.**—Surgeons who perform the operation in two stages, do so with the view of diminishing the danger of mediastinal infection. At the first operation, the larynx is isolated from the surrounding structures, but not severed from the trachea or pharynx. It is left in place and packed about with iodoform gauze; this results in the formation of granulations. Later the larynx is completely removed. Others perform a preliminary tracheotomy, dissect the trachea free from the surrounding structures, pack about with iodoform gauze at the first operation; at the second operation the larynx is completely removed.

FIELDING O. LEWIS.

## OBSTRUCTIVE LARYNGEAL DYSPNEA

Dyspnea is a symptom, not a disease, but it is a symptom of so many different conditions, and the prompt recognition of the laryngeal form is so often of life-saving importance, that its separate consideration has seemed advisable.

**Definition.**—Dyspnea means difficult breathing. Obstructive laryngeal dyspnea is the kind due to obstruction in the airway of the larynx or cervical portion of the trachea. The separate consideration of this form of dyspnea is not a mere academic recognition of an anatomical borderline. The facts are that if this condition is not recognized the patient will die, whereas life-saving relief could have been afforded in a few minutes by tracheotomy; and that there is no common condition more easily recognized nor so often unrecognized.

**Etiology.**—Obstruction to the lumen of the airway of the larynx and cervical trachea may be caused by many conditions, a few of which are given in the following list: Edema, hypertrophy, crusts, thick secretions, hyperplasia, wounds, instrumentation, cauterization, influenza, typhoid fever, diphtheria, the exanthemata, tuberculosis, syphilis, leprosy, scleroma, neoplasms, caustic alkalies or acids, attempted suicide by external or internal means, inspired foreign bodies or insects, gunshot and stab wounds, automobile accidents, war gases, abductor paralysis, crico-arytenoid fixation, and retropharyngeal abscess.

**Symptomatology.**—The chief symptoms are restlessness, increasing respiratory rate, stridor, indrawing at the suprasternal notch, and ashy-gray pallor. Inspiration may be more obstructed than expiration and vice versa. The appetite usually is poor; the child declines food because he is too busy getting in enough air to sustain life to tolerate the interruption to his breathing involved in the taking of food through the mouth. While the progress of dyspnea is usually gradual, the final onset of asphyxia is usually very rapid, often sudden. In angioneurotic edema, the time from the onset of dyspnea to death by asphyxia may be a matter of only a few minutes.

**Diagnosis.**—Every practitioner of medicine should be able instantly to recognize obstructive laryngeal dyspnea by the indrawing at the suprasternal notch. If this fundamental fact alone were drilled into every medical student, fatal diagnostic errors in these cases would be few. It is necessary, however, in a text-book to supplement this simple statement of fact.

The diagnosis as to the *cause* of the dyspnea requires mirror-laryngoscopy, direct laryngoscopy, and bronchoscopy. The larynx and trachea of any human being can be inspected by peroral endoscopy. The diagnosis of the conditions causing obstructive dyspnea has been dealt with elsewhere in this book. The important thing to emphasize here is the diagnosis as to the *location* of the obstruction. That is, as to whether the obstruction is above or below the clavicle; in other words, to decide in any case of dyspnea whether or not it is a case of obstructive laryngeal dyspnea. When the obstruction is in the larynx or the cervical trachea the unsatisfied negative pressure caused by the inspiratory expansion of the thoracic bellows causes *indrawing* of the soft tissues where they are unsupported by bone or cartilage (Figs. 535, 536). The locations of this indrawing are:

(1) The suprasternal notch; (2) the supraclavicular fossæ; (3) the intercostal spaces; (4) the epigastrium.

The most important of these is the indrawing at the suprasternal fossa; next is the epigastric indrawing, especially in children. But the deep depression going down back of the manubrium at each inspiration always means obstruction above that point; and preparations for tracheotomy should be made at once. The indrawing at the suprasternal notch is diagnostic of obstructive laryngeal dyspnea. It is never seen in the dyspnea of asthma, pneumonia, or other diseases, mediastinal, cardiac, or pulmonary, unless laryngeal complications are present.

In addition to the cardinal sign, indrawing, there are two accompanying signs that are important; but unlike the indrawing, they are not absolutely diagnostic of obstructive laryngeal dyspnea. One is *stridor*. Stridor may be loosely said to be any sound caused by the breathing that is louder



Expiration.



Inspiration.

Fig. 535.—From photographs of a child asphyxiating with obstructive laryngeal dyspnea. The indrawing at inspiration is diagnostic, and is never seen in any form of dyspnea except that due to obstruction in or close to the larynx. Failure to recognize the true meaning of this indrawing has resulted in the death of thousands for want of a tracheotomy. The indrawing is noted at the suprasternal notch and at the epigastrium on inspiration. At the left is shown how the indrawing momentarily disappears on expiration. The anxious face of the child is typical of impending death by asphyxia. There is not a moment to be lost in presence of such symptoms. To delay tracheotomy would be fatal. Sedatives sometimes given for the restlessness hasten death.

than normal. It is of less importance than indrawing, which is the danger signal. The other sign is *restlessness*. This ranks equally with indrawing as a sign of danger. It means air-hunger. As soon as the child dozes he loses the aid of his volition in deepening his breathing, and he awakens with the sense of suffocation. Cyanosis is late, and may be absent to the end. Ashy-gray pallor or "leaden" color is diagnostic. Cyanosis may be marked in an epileptic seizure, but the patient will be unconscious, and there will be convulsions and no indrawing. A man asphyxiating with a huge piece of meat in his larynx may be intensely cyanotic, and may clutch at his throat, but there will be no true convulsions; he will be conscious until respirations are about to cease, and at these last respirations there will be deep inspiratory indrawing at the suprasternal notch. In an early stage of obstructive laryngeal dyspnea a careful observer will note that the patient is making the thoracic movements of inspiration, but the air does not seem

to pass in as it should. This observation is made by inspection of the stripped patient, not by auscultation. The latter is of little use, because the rushing of the thin column of air may make vibrations out of all proportion to the small amount of air the patient is actually getting into his lungs, and because there is no differentiation between areas for comparison. Of course, thorough examination by auscultation, percussion, and palpation, and every other means should always be carried out if there is time, but nothing should be allowed to lead the examiner away from the cardinal sign of indrawing at the suprasternal notch, and in urgent cases tracheotomy



Fig. 536.—Photograph of the same child as shown in Fig. 535. The indrawing has disappeared; all the anxiety has disappeared from the face and the child has fallen asleep on the operating table after the tracheotomy. It had been fighting for air day and night until almost dead of exhaustion.

should be done first on this sign alone; the diagnosis can be completed afterward. The author has many times arrived on the scene as the patient was breathing his last; never has there been cause to regret the emergency tracheotomy done because of the indrawing without any knowledge of the history of the case or of the pathological conditions present.

**Prognosis.**—The mortality of obstructive laryngeal dyspnea is high in the unrecognized group of cases. There is no danger to life in the condition if it is recognized early and tracheotomy is promptly done. Unfortunately, its cardinal signs are not taught in our medical schools as they should be. Of a series of 126 cases admitted to the Bronchoscopic Clinic, 34 had been diagnosed asthma, 29 pneumonia, and 1 paralysis of the diaphragm! All

of these children were instantly relieved by a tracheotomy. Under such mistaken diagnoses the mortality of obstructive laryngeal dyspnea is appallingly high. Many deaths have occurred from the mistake of giving sedatives to a restless child when the restlessness was due to *air-hunger*, as the experienced eye would instantly have noticed by the cardinal signs of obstructive laryngeal dyspnea the indrawing at the suprasternal notch and at the epigastrium.

**Treatment.**—There is only one thing to do for a patient with well-marked obstructive laryngeal dyspnea, and that is, a low tracheotomy. There are only three exceptions to this rule: (1) If the case is one of laryngeal diphtheria, and there is a skilled intubator who will not only introduce the intubation tube but will remain in the house to reintubate in case the tube is expelled, intubation is worth consideration; otherwise it is not. (2) Laryngismus stridulus is accompanied by indrawing, but the dyspnea may subside without tracheotomy. (3) Retropharyngeal abscess may require only evacuation.

Apart from the matter of prevention of asphyxia there are two great advantages of tracheotomy, as compared to intubation, namely: (1) drainage of secretions and (2) putting the larynx at rest. Pulling the air at high speed through the narrowed chink is irritating. The narrowing of the glottis is not the only impediment to expulsion of secretions. The glottic movements are necessarily impaired in every case in which the glottic chink is diminished in area of cross-section; as a consequence glottic co-ordination in bechic expulsion is lacking. Objective evidence of this is presented at every tracheotomy done for prolonged laryngeal dyspnea: as soon as the trachea is opened a mass of accumulated secretions is expelled. The increasing accumulation of the secretions often makes the death for want of tracheotomy essentially one of *drowning of the patient in his own secretions*.<sup>1</sup> The question as to whether the tracheotomy should be done at once or postponed until the symptoms become more urgent often arises, and it must be acknowledged that the decision is nearly always wrong. "We always preach early tracheotomy, but practically always do it late—dangerously late."<sup>2</sup> At best postponement usually results in hasty tracheotomy, done high and badly, with resultant difficult decannulation and chronic laryngeal stenosis if, indeed, the child's life is saved. The risks of postponement depend upon the circumstances. If the patient is in a hospital, with the operating room "set up" with sterile tables, oxygen tanks, and personnel all in readiness, it is justifiable to wait longer than when the patient is in a dwelling. Any patient with signs of beginning obstructive laryngeal dyspnea should be moved to a hospital for observation if possible; and whether in the hospital or not he should never be allowed to draw an unwatched breath. Eternal vigilance is the price of life in such cases, from the onset of the indrawing at the suprasternal notch until the patient is decannulated. During the observation period in the postponed cases the intern and the nurse should be instructed that no sedatives are to be given. *Never give morphine or any other sedative when the patient shows signs of obstructive laryngeal dyspnea* is a rule that should be taught to every medical student, and it should be printed in the form of a motto in the room of every hospital intern. It is the routine to order morphine or codeine for every restless patient. A child with the air-hunger of beginning dyspnea is always restless, and has reason to be. He is depending in

part on his voluntary respiratory muscles to pull in enough air to keep him alive. When fatigue and loss of sleep begin to overcome him he dozes, and as soon as he does so he loses the aid of his voluntary muscular mechanism. Impending suffocation wakens him with a start. He tosses about. The nurse reports him to the intern as restless. The intern uninstructed in the signs of obstructive laryngeal dyspnea asks if the child is blue. The nurse reports negatively and the intern orders an opiate. The nurse is correct, because a dyspneic child is not blue till nearly dead, and not usually even then. By this time the child is worn out. He is rapidly reaching the stage where the overpowering demand for sleep will overcome him and he will sleep away in *listless asphyxia*, without the slightest fight or struggle for air that characterizes *acute asphyxia*. It is obvious that what is needed by the child restless with obstructive laryngeal dyspnea is *tracheotomy, not opiates*. It is well known that opiates are powerful respiratory paralyzers. The author has vivid recollections of an experience as one of a group of seniors who took turns, in pairs, supporting and "walking" up and down the hallway a patient who had swallowed a lethal dose of laudanum. The heart was doing nobly at about 20 pulsations to the minute, but the respiratory center was paralyzed; the breathing stopped every time we stopped because the patient fell asleep. A modern plan of treatment would have included bronchoscopic oxygen insufflation; but our crude and ultimately successful efforts taught me a fundamental lesson that has resulted in the saving of the lives of hundreds of children with obstructive laryngeal dyspnea, by forewarning against opiates in these cases. Hundreds of times it has been a pleasure to see a little child worn out by too long a fight for air drop off into a quiet, restful sleep on the operating table after a tracheotomy and continue to sleep for hours, often a day and a night, after being carried to bed.

**Laryngismus stridulus** presents a typical picture of obstructive laryngeal dyspnea. The direct laryngoscopic appearances are elsewhere herein described. The indrawing and the apparently impending asphyxia, if due to any condition other than the temporary sucking in of relatively normal tissue, would call urgently for prompt tracheotomy. In a few cases tracheotomy may be required to prevent asphyxia, but it is very rarely indeed that it is required in this disease. If a bronchoscope is available, its insertion will obviate the necessity for tracheotomy.

CHEVALIER L. JACKSON and CHEVALIER JACKSON.

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#### TRACHEOTOMY

**Definition.**—The operation of making an opening into the trachea.

**Purposes.**—The purpose for which tracheotomy is most often done is to prevent asphyxia by enabling a patient to breathe through his neck when obstruction above prevents him from breathing through his mouth.

This pathological state of obstructive laryngeal dyspnea arises in the course of many different diseases, as enumerated in the previous chapter. When indicated for obstructive laryngeal dyspnea tracheotomy should be done early. The patient has everything to lose and nothing to gain by waiting until he is *in extremis*. Hasty tracheotomy is usually done too high and laryngeal stenosis usually results<sup>1</sup> (Fig. 537). Formerly *tracheotomy* was done to remove a foreign body from the larynx or trachea, but direct laryngoscopy has rendered it much easier, safer, and better to remove the foreign body through the mouth. The introduction of a bronchoscope through a tracheotomy wound has been rendered obsolete by improved instruments and methods, which make peroral introduction easier and better. Tracheotomy was also done formerly for the intratracheal administration of an anesthetic in bloody operations about the upper air-passages requiring packing of the pharynx; this has been rendered entirely obsolete by the ether insufflation methods of Melzer, Auer, Elsberg, and George P. Muller,

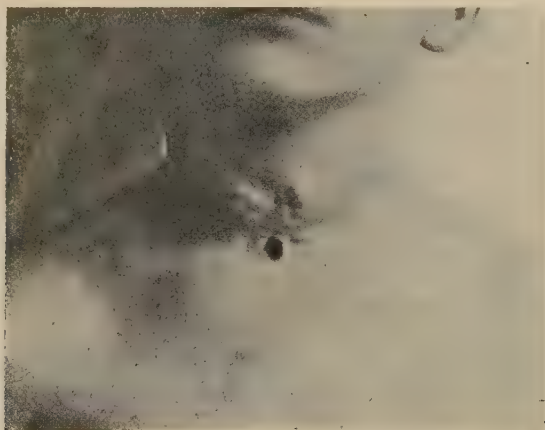


Fig. 537.—Boy, aged three years. The upper scar shows the site of the tracheotomy through the larynx that resulted in laryngeal stenosis. Below the scar is seen the cannular fistula in its proper place in the suprasternal notch. This second tracheotomy was done as the first step in the cure of the laryngeal stenosis.

and by the simple intubation method of Flagg<sup>4</sup> which was mentioned under the head of Direct Laryngoscopy.

**Anatomical Considerations.**—Anatomical text-books portraying with minute and from the anatomist's standpoint necessary detail, all the blood-vessels and other structures in the front of the neck, do not lay a foundation of self-confidence in him who for the first time is confronted with the life-saving duty of doing an emergency tracheotomy on an asphyxiated patient. Therefore, it seems best that after he has learned all the anatomist can teach him, he memorize the following points, and rehearse them bloodlessly on every patient whose neck he palpates for any purpose whatever.

Adam's apple is always easy to find. It is attached to the upper end of the trachea. The trachea extends in the center line of the neck down to the suprasternal notch. When you cut through the overlying tissues you can run your left index-finger from Adam's apple down along the trachea all the way to the suprasternal notch. There is no seriously large vessel or any other important structure in the middle line of the front of

the neck between the skin and the trachea. All of the structures you must not cut are at the sides; keep them there! The center line is safe; keep it prominent. It ceases to be a safety line at the level of the supra-

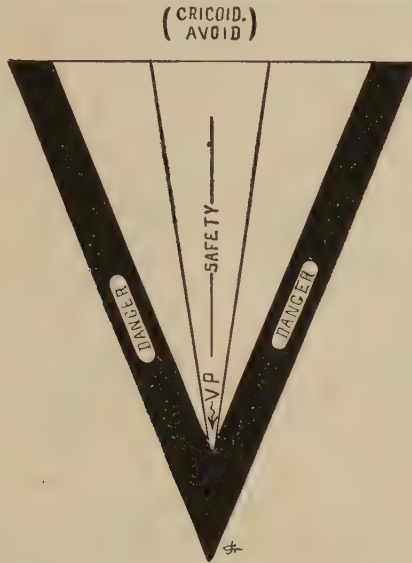


Fig. 538.—Schema of practical gross anatomy to be memorized for emergency tracheotomy. The middle line is the safety line, the higher, the wider. Below, the safety line narrows to the vanishing point VP. The upper limit of the safety line is the thyroid notch until the trachea is bared, when the limit falls below the first tracheal ring. In practice the two dark danger lines are pushed back with the left thumb and middle finger as shown in Fig. 544, thus throwing the safety line into prominence. This is generally known as Jackson's tracheotomic triangle. (From Peroral Endoscopy and Laryngeal Surgery, Text-book, 1914.)

sternal notch. The cricoid cartilage is the only complete ring in the lower air-passages, and should not be unnecessarily cut.

These points are the fundamentals of the tracheotomic triangle (Fig. 538), a clear conception of which will give self-confidence to the operator

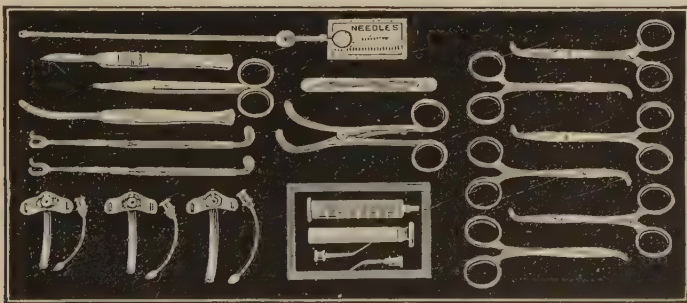


Fig. 539.—Illustration of the Ellen J. Patterson tracheotomic set of instruments. They are packed in sterile copper box, wrapped in sterile wrappers, ready for immediate use in emergencies.

in the quick, safe, and unflinching performance of emergency tracheotomy after the patient has become black in the face and has ceased to breathe.

**Instruments.**—In an emergency tracheotomy has many times been

done with a pocket knife, but in every hospital, accident ward, dispensary, and physicians' consulting room there should be a sterile package containing at least a minimum tracheotomic outfit. Many years ago Dr. Ellen J. Patterson prepared for the Bronchoscopic Clinic the outfit shown in Fig. 539. One of these sets is kept in the operating room, ready for instant use, to avoid the delay of gathering up the scattered component parts in cases of emergency. Any pupil nurse can hand out the box. The following are its contents:

Headlight	Tracheal cannulæ (six sizes)
Scalpels	Curved needles
Curved, probe-pointed bistoury	Needle-holder
2 retractors	Hypodermic syringe for local anesthesia
Trousseau dilator	with bent reinforced needle
6 hemostats (special)	No. 1 plain catgut ligatures
Tenaculum (special)	Linen tape
Scissors (dissecting)	Gauze sponges

These are sterilized, packed sterile in a copper box with lid. The box is doubly wrapped and pinned up in sterile towels.

For the after-care of the patient the following are kept in the patient's room after the operation:

Sterile gloves	1 hemostat
Sterile new gauze	Trousseau dilator
Scissors	Duplicate tracheotomy tube
Silver probe	Basin of bichloride of mercury solution, 1 : 10,000

Where there are many tracheotomic patients to be dressed the tray and outfit illustrated under the head of Chronic Laryngeal Stenosis is very convenient. Each patient should have his own tray and outfit.

**Cannulæ.**—It is appalling to contemplate the huge collection of old junk cannulæ taken from patients that have been sent in for treatment of post-tracheotomic laryngeal stenosis. When one thinks of the millions and millions of dollars that are spent in erecting beautiful hospital buildings it is sad to think that there has been in so many instances no one to see that a set of cannulæ of the various sizes, made of proper material and of proper form, is among the essentials of equipment. Cannulæ of soft rubber are irritating and soon become dangerously friable whether used or not. Hard-rubber tubes are thick walled, small lumened, cannot be boiled without losing shape, and are dangerously friable. Aluminum tubes corrode on boiling, and even in the secretions; corrosion means irritation. Plated tubes soon lose their plating. Alloyed metals are undesirable materials except for temporary use. Sterling silver is the ideal material. The shape of the tube is of utmost importance. If too short it will not reach the trachea after the reactionary swelling of the wound has increased the distance from the skin to the trachea. It had better be too long; the extra length can be taken up with a few more layers of dressings under the shield. Proper cannulæ that have been tested in hundreds of cases during the past twenty years are shown in Fig. 540. These cannulæ are sufficiently long to prevent the accident illustrated in Fig. 541.

**Selection of Sizes of Tracheotomic Cannulæ.**—The selection of the proper size of cannula for the particular patient is important. There is an individual variation in the size of the trachea so that any tabulation of sizes in relation to age is subject to modification to suit the patient. A closely fitting cannula, that is, one that allows no air-space between its outer sur-

face and the wall of the trachea, should not be worn continuously except after laryngectomy; even then a tight fit is objectionable. In all other cases a by-passage for air is necessary. The following table gives the sizes

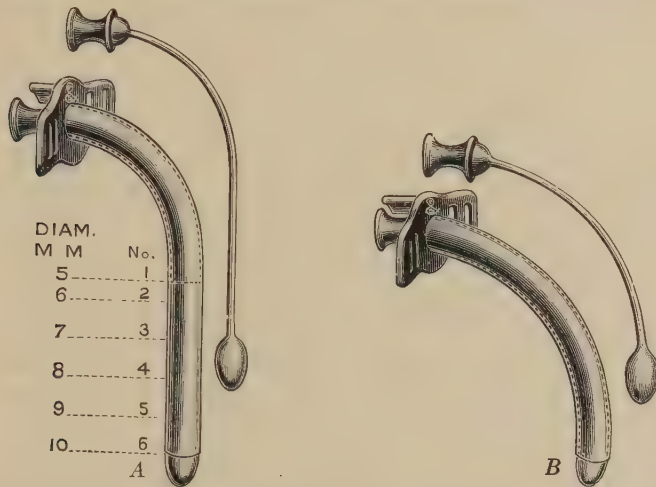


Fig. 540.—The author's tracheotomic cannulae. *A*, Cane-shaped cannulae for use in intrathoracic compressive or other stenoses. *B*, Full curved cannulae for regular use. Pilots are made to fit the outer cannula; the inner cannula not being inserted until after withdrawal of the pilot. For all ordinary purposes the cannulae, *B*, are best. A full set consists of 6 sizes. (From Peroral Endoscopy and Laryngeal Surgery, Text-book, 1914.)

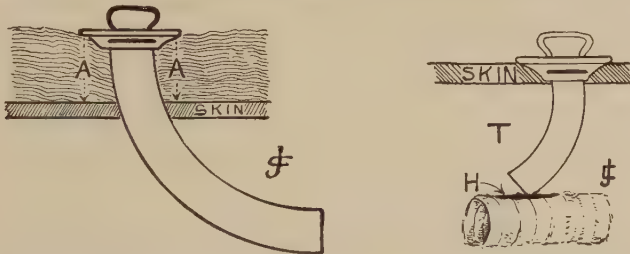


Fig. 541.—Schema showing thick pad of gauze dressing, filling the space *A*, and used to hold out the author's full-curved cannula when too long, prior to reactionary swelling, and after subsidence of the latter. At the right is shown the manner in which the ordinary cannula of the shops permits a patient to asphyxiate, though some air is heard passing through the tracheal opening (*H*), after the cannula has been partially withdrawn by swelling of the tissues (*T*). The cannula may even have been pushed into the tissues of the neck, where it will go more easily than into the trachea in the first few days after operation. Mortality after tracheotomy is most often due to failure to pipe the air into the lungs and to see to it that it gets there. (From Peroral Endoscopy and Laryngeal Surgery, Text-book, 1914.)

that in an average child, whose trachea is not swollen, are best suited to the respective ages, but the proper fit should be determined by trial in each patient:

	Number
Newborn infant.....	1
Infancy to one year.....	1
One to three years.....	2
Three to six years.....	3
Six to twelve years.....	4
Twelve to twenty years.....	5
Adults, women.....	5
Adults, men.....	5 or 6
Laryngectomized patients.....	7 or 8

In infants it is necessary, obviously, to use the small tube, but it must be remembered that the baby is at a dangerous disadvantage in being compelled to use this small tube, because it takes very little to clog it. The disadvantage is doubled by the fact that the comparatively weak muscles of a baby cannot produce a strong expulsive blast to clear the cannula.

**Anesthesia.**—For many years it has been our rule at the Bronchoscopic Clinic that no dyspneic patient should be given a general anesthetic, because any patient dyspneic enough to need a tracheotomy for dyspnea is depending largely upon the action of the accessory respiratory muscles. When this action is stopped by beginning unconsciousness respiration ceases. If the trachea is not immediately opened, artificial respiration instituted, and oxygen insufflated, the patient dies on the table. Skin infiltration along the line of incision with a very weak cocaine solution (1/10 of 1 per cent.), apothesine (2 per cent.), novocaine, (1/2 of 1 per cent.), or other local anesthetic, suffices to render the operation painless. If the patient is very dyspneic we keep him in the sitting position on the operating table during the infiltration; the recumbent posture, which increases dyspnea, is not assumed in these extremely dyspneic patients until we are ready to make the incision. Overextension of the head is avoided because it increases dyspnea. It has been advocated that an interannular injection of cocaine solution with a hypodermic syringe be done just prior to incision of the trachea for the purpose of preventing cough after the incision of the trachea and the insertion of the cannula. It would seem, however, that this introduces the risk of inspiratory infective processes, by permitting the inspiration and clotting of blood in the small bronchi, followed by subsequent breaking down of the clots. The cough reflex is the "watchdog of the lungs," and if not drugged asleep by local or general anesthesia, it can safely be relied upon to prevent inspiration into the deeper air-passages of blood or the pus which nearly always is present in acute or chronic conditions calling for tracheotomy. Cocaine in any form, by any method, and in any dosage, is dangerous in very young children.

**Technic.**—The patient should be placed in the recumbent position, with the extended head held in the midline by an assistant. The shoulders, not the neck, should be slightly raised with a sand-bag. The head should be somewhat lower than the feet to lessen the danger of inspiration of blood. A midline incision dividing the skin and fascia is made from the thyroid notch to just above the suprasternal notch. The cricoid is now located, and the deeper dissection is continued from below this point. The ribbon muscles are separated with dissecting scissors or knife, and held apart with retractors. If the isthmus of the thyroid gland is in the way, it may be retracted upward; if large, however, it should be ligated and divided, for it is apt to slip over the tracheal incision afterward, and render difficult the quick finding of the incision during after-care. This covering of the tracheal incision by the slipping back of the drawn-aside thyroïdal isthmus is one of the most frequent avoidable causes of mortality, because it deflects the cannula off into the tissues when it is replaced after cleaning in the early postoperative period. The corrugated surface of the trachea can be felt, and its exact location determined, by the index-finger. If the tracheotomy is proceeding in an orderly manner all bleeding points should be caught and tied with plain catgut before the trachea is opened. Because of distention of vessels during cough, all but the tiniest vessels

should be ligated. Side-cut veins are particularly treacherous. They should be freed of tissue, the divided ends ligated, and cut across.

The *incision in the trachea* should be as low as possible (Fig. 542) and should never be made through the first ring. The incision should be through the third, fourth, and fifth rings. Only in cases of *laryngoptosis* will it be necessary to incise the trachea higher than this. The incision must be made in the midline and in the long axis of the trachea, and care



Fig. 542 — In the suprasternal notch is seen a scar which, being red, appears dark in the photograph. In the center of the scar is seen the fistula from which the cannula has just been removed. This shows the proper location of a tracheotomy. Incidentally it may be added this is the same child as illustrated in Figs. 535, 536, three weeks later. The placid face, the full epigastrium, and the full suprasternal notch are in marked contrast to the indrawing and the anxious face shown in the preceding illustrations taken on admission. This life-saving work depends upon prompt recognition of the cardinal signs of obstructive laryngeal dyspnea.

must be exercised that the point of the knife does not perforate the posterior tracheal wall. Stab incisions are always to be avoided. If the incision in the trachea is found to be of insufficient length, the original incision must be found and elongated. A second incision must not be made, for the portion of cartilage between the two incisions will die, and will almost certainly make a site for future tracheal stenosis. The cricoid should never be cut, for stenosis is almost sure to follow the wearing of a cannula in this

position. A Trousseau dilator should now be inserted in the tracheal incision and its blades gently separated. With the tracheal lumen thus opened a cannula of proper size is introduced with absolute certainty of its having entered the trachea. A quadruple-folded square of gauze in the



Fig. 543.—The proper way to introduce a tracheotomic cannula. The cannula is held with both hands; the right thumb holds the pilot in place. The cannula is given a circular motion corresponding to the radius of the curve of the cannula. At *A* the slit in the trachea is found and the entrance of the pilot and inner end of the cannula into the trachea is assured. This is vitally important. At *B* the circular motion is continued. At *C* the cannula is in place ready for tying of tapes and insertion of gauze pad (Fig. 545). The pilot has been blown out by cough when the thumb was taken off it as the cannula reached its resting place. Failure to execute properly the movement here illustrated may result fatally during the postoperative period by misplacing the tube in the mediastinum instead of the trachea. Later, when a strongly walled fistula has been established, the danger of a false passage is largely eliminated.

form of a pad about 4 inches square is moistened with mercuric chloride solution (1 : 10,000), and is slit from the lower border to its midpoint. This pad is slipped from above downward under the tape-holder of the cannula, the slit permitting the tubal part of the cannula to reach the central part

of the pad (Fig. 545), which completely covers the wound. No attempt should be made to suture the skin, for this tends to form a pocket in which



Fig. 544.—Illustration of corks used to occlude the cannula in training patients to breathe through the mouth again, before decannulation. The corks allow air leakage, the amount of which is regulated by the use of different shapes. A smaller and still smaller air leak is permitted until finally an ungrooved cork is tolerated. A central hole is sometimes used instead of a slot. *A*, One-third cork; *B*, half cork; *C*, three-quarter cork; *D*, whole cork. These corks are made by grinding pure rubber cord to shape on an emery wheel. After grinding the taper, if a partial cork is desired a groove is ground on the angle of the emery wheel. If a half cork is desired, half of the cork is longitudinally ground away on the side of the wheel. Reliable corks made in this way are now obtainable from Messrs. George P. Pilling & Son Company. A bark cork should never be used in a tracheal cannula, and the ordinary rubber corks of commerce are likewise dangerously friable.



Fig. 544a.—Schema showing the author's method of rapid tracheotomy. First stage: The hands are drawn ungloved for the sake of clearness. The upper hand is the left, of which the middle finger (*M*) and the thumb are used to push aside the sternocleidomastoid muscles, the finger and the thumb being close to the trachea in order to press backward out of the way the carotid arteries and the jugular vein. This throws the trachea forward into prominence, and one deep slashing cut will incise all of the soft tissues down to the trachea. Full length and depth of this primary incision are fundamental. (From *Peroral Endoscopy and Laryngeal Surgery*, Text-book, 1914.)

lodge the bronchial secretions that escape alongside the tube, resulting in infection of the wound. Furthermore, it renders the daily changing of the

tube much more difficult. In fact, it prevents the attendant from being certain that the tube is actually placed in the trachea. Suturing of the skin to the trachea should never be done, for the sutures soon tear out, and often set up a perichondritis of the tracheal cartilages, with resulting difficult decannulation.

**Emergency Tracheotomy.**—Stabbing of the cricothyroid membrane, or an attempted stabbing of the trachea, so long taught as an emergency tracheotomy, is a mistake.<sup>1, 2, 3</sup> This has been abundantly corroborated.<sup>5, 6</sup> The author's "two-stage, finger-guided" method is safer, quicker, more



Fig. 544b.—Illustrating the author's method of quick tracheotomy. Second stage: The fingers are drawn ungloved for the sake of clearness. The whole wound is full of blood, and the rings of the trachea are felt with the left index-finger, which is then moved slightly to the patient's left, while the knife is slid down along the left index-finger to exactly the middle line, when the trachea is incised. If any difficulty is encountered in feeling the trachea the index-finger should locate the thyroid cartilage and then follow on downward to the cricoid and trachea, with which it is, of course, continuous. Incision is then made in the tracheal rings, preferably below the first ring. (From *Peroral Endoscopy and Laryngeal Surgery*, Text-book, 1914.)

efficient, and not likely to be followed by stenosis. To execute this promptly the operator is required to forget his text-book anatomy and memorize the schema (Fig. 538).

**First Stage.**—The larynx and trachea are steadied by the thumb and middle finger of the left hand, which at the same time push back the important nerves and vessels which parallel the trachea, and render the central safety line more prominent (Fig. 544a). A long incision is now made from the thyroid notch almost to the suprasternal notch, and deep enough to reach the trachea.

**Second Stage.**—The entire wound is full of blood and the trachea cannot

be seen, but its corrugations can be very readily felt by the tip of the free left index-finger, which is used to follow down along the trachea from the always easily found Adam's apple. The left index-finger is now moved a little to the patient's left in order that the knife shall come precisely in the midline of the trachea, and three rings of the trachea are divided from above downward (Fig. 544b). The Trousseau dilator should now be inserted, the head of the table lowered, and the patient turned on his side to allow the blood to run away from the wound. If respiration has ceased, a cannula is slipped in and artificial respiration is begun. Oxygen insufflation will aid in the restoration of respiration, and a pearl of amyl nitrite should be crushed in gauze and blown in with the oxygen. In all such cases excessive pressure of oxygen should be avoided because of the danger of producing ischemia of the lungs. Hope of restoring respiration should not be abandoned for half an hour at least. One of the author's assistants, Dr. Phillip Stout, saved a patient's life by keeping up

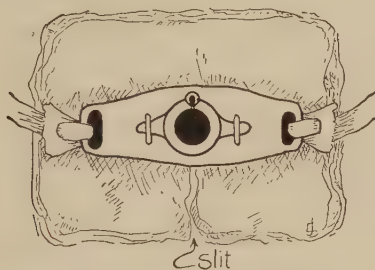


Fig. 545.—Method of dressing a tracheotomic wound. A broad, quadruple, infolded pad of gauze is cut to its center so that it can be slipped astride of the tube of the cannula, back of the shield. No strings, ravelings, or strips of gauze are permissible because of the risk of their getting down into the trachea.

artificial respiration for twenty minutes before the patient could do his own breathing.

The *after-care* of the tracheotomic wound is of the utmost importance. A special day and night nurse are required. The inner tube of the cannula must be removed and cleaned as soon as it contains secretion; not less often than every half-hour in the first few days. Ordinary decent cleanliness would call for changing the whole apparatus as often as one would clean an artificial denture. Secretion coughed out must be wiped away quickly, but gently, before it is aspirated. The gauze dressing covering the wound must be changed as soon as soiled with secretions from the wound and the air-passages. Each fresh pad should be moistened with very weak bichloride of mercury solution (1 : 10,000). The outer tube must be changed every twenty-four hours, and oftener if the bronchial secretion is abundant. Student-physicians who have been taught these methods and who have seen the patients in care of our nurses have often expressed amazement at the neglect unknowingly inflicted on such patients elsewhere, in the course of ordinary routine surgery. It is not unusual for a patient to be sent to the Bronchoscopic Clinic who has worn his cannula without a single changing for many months. The dermatitis, foul crusts, and exuberant granulations showed a lack of decent cleanliness, to say nothing of surgical care. Worst of all is the building up of fibrous tissue by

the chronic inflammation. In some cases the tube had broken, and a portion had been aspirated into the trachea.

The dry overheated air of a hospital with a modern heating installation is not good for tracheotomic breathing; it is especially bad in case of a child with weak or absent cough-reflex, and still worse when there is a tendency to crust and plug formation. Outdoor air is best for such patients, but a croup-tent properly managed will help prevent the formation of plugs (Fig. 546).

If the respiratory rate increases, instead of attributing it to pulmonary complications the entire cannula should be removed, the wound dilated with the Trousseau forceps, the interior of the trachea inspected, and all

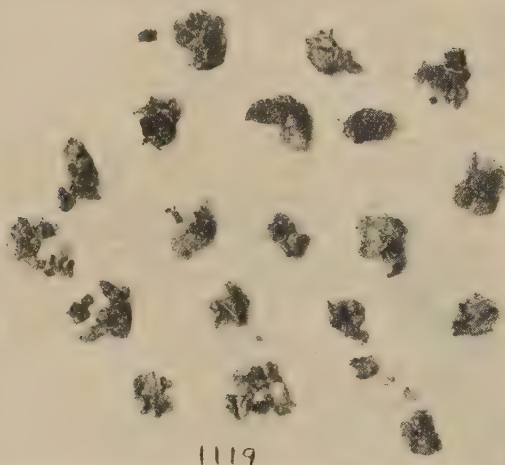


Fig. 546.—Plugs and crusts removed with the bronchoscope in the after-care of a tracheotomized child. Seventeen times the child was in a state usually fatal because of erroneous diagnoses of "pneumonia," "edema of the lungs," "filling," etc. Each time the physical signs on which such diagnoses are usually based disappeared completely after the bronchoscopic removal of one of the dried plugs of secretion here shown from the bifurcation or both main bronchial orifices. It is not often that such plug formation is encountered; but this case emphasizes the life-saving fundamental fact that the after-care of tracheotomic cases is one of good "plumbing"; that is, keeping the "pipes," natural and artificial, clear of obstruction. Air must be piped not merely into the trachea, but into the lungs, and it must be the constant duty of someone to know by seeing, feeling, and hearing, that it gets there. Only too often at the first replacement of the cannula it is piped into the tissues of the neck (Fig. 541). (From *Bronchoscopy and Esophagoscopy*, Text-book, 2 ed., 1927.)

secretions cleaned away. Then the tracheal mucosa below the wound should be gently touched with a sterile bent probe to induce cough in order to rid the lower air-passages of accumulated secretions. In many cases it is a life-saving procedure to insert a sterile long malleable aspirating tube to remove secretions from the lower air-passages. When all is clear, a fresh sterile cannula which has been carefully inspected to see that its lumen has been thoroughly cleaned, is inserted, and its tapes tied. Good "plumbing," that is, the maintenance at all times of a clear, clean passage in all the "pipes," natural and artificial, is the reason why the mortality in the Bronchoscopic Clinic has been less than half of 1 per cent., while in ordinary routine surgical care in all hospitals collectively it ranges from 10 to 20 per cent. The difference is not in the performance of the operation,

but in the after-care. Hundreds of times we have been called by an intern with the statement that the patient was "developing pneumonia," only to find that the increased respiratory rate and impaired percussion note were due simply to misplacement of the cannula, or obstruction of the cannula, the trachea, or the bronchi by easily removable secretions.

**Bronchial Aspiration.**—As mentioned above, bronchial aspiration is often necessary. When the patient is unable to get up secretions, he will, as demonstrated by the author many years ago, "drown in his own secretions." In some cases bronchoscopic aspiration is required (Peroral Endoscopy, page 483). Occasionally very thick secretions will require removal with forceps (Fig. 546). Pus may become very thick and gummy from the administration of morphine. Opiates should not be given because they not only obtund the cough reflex, which is the watchdog of the lungs, but they also lessen the normal secretions that ordinarily increase the quantity and fluidity of the pus though they do not lessen pus formation. When to this is added the desiccating effect of the air inhaled through the cannula, unmoistened by the upper air-passages, the secretions may be so thick as to form crusts and plugs that are equivalent to foreign bodies and require removal with forceps (Fig. 546). Diphtheritic membrane in the trachea may, likewise, require removal with bronchoscope and forceps. Thinner secretions may be removed by sponge-pumping. In most cases, however, secretions can be brought up through an aspirating tube, connected to a bronchoscopic aspirating syringe, an ordinary aspirating bottle, or preferably, a mechanical aspirator electrically driven. In this, combined with bronchoscopic oxygen insufflation (*q. v.*), we have a life-saving measure of the highest efficiency in cases of poisoning by chlorine and other irritant and asphyxiating gases. An aspirating tube for insertion into the deeper air-passages should be of copper, so that it can be bent to the proper curve to reach into the various parts of the tracheobronchial tree, and it should have a removable copper-wire core to prevent kinking and collapse of the lumen. The distal end should be thickened, and also perforated at the sides, to prevent drawing in of the mucosa and trauma thereto. A rubber tube may be used, but it not so satisfactory. A very small soft-rubber catheter is often useful in infants.

**Complications Following Tracheotomy.**—Pneumonia and edema of the lungs are exceedingly rare. Their preponderance in statistics is due to errors of diagnosis and faulty after-care (*q. v.*). Subcutaneous emphysema may occur and is of no consequence unless the error of stitching up the wound has been committed. If so, all the stitches should be removed; otherwise mediastinal emphysema may develop from working of the air along between the tissue layers under the deep fascia, aided by the normal negative pressure at each inspiration; it may even reach the pleural cavity, causing pneumothorax. A wound packed widely open is the best preventive of these complications. Sepsis need not be feared if dressings are changed every half-hour or oftener.

**Decannulation.**—When the tracheal incision is placed below the first ring no difficulty in decannulation should result from the operation *per se*. When by temporarily occluding the cannula with the finger it is evident that the laryngeal aperture has regained sufficient size to allow free breathing, a smaller sized tracheotomic tube should be substituted to allow free passage of air around the cannula in the trachea. In doing this the amount

of secretion and the handicap of impaired glottic mobility in the expulsion of thick secretions must be borne in mind. Babies labor under a special handicap in their inefficient bechic expulsion and especially in their small cannulæ which are so readily occluded. If breathing is not free and quiet with the smaller tube, the larger one must be put back. If, however, there is no trouble with secretions, and the breathing is free and quiet, the inner cannula should be removed, and the external orifice of the outer cannula firmly closed with a rubber cork. If the laryngeal condition has been acute, decannulation can usually be safely done after the patient has been able to sleep quietly for three nights with a corked cannula. If free breathing cannot be obtained when the cannula is corked, the larynx is stenosed, and special work will be required to remove the tube. Children sometimes become panic-stricken when the cannula is completely corked at once and they are forced to breathe through the larynx instead of the easier short-cut through the neck. In such a case the first step is partially to cork the cannula with a half or two-thirds plug made from a pure rubber cord fashioned in the desired shape by grinding with an emery wheel. Thus the patient is gradually taught to use the natural airway, still feeling that he has an "anchor to windward" in the opening in the cannula. When some swelling of the laryngeal structures still exists, this gradual corking has a therapeutic effect in lessening the stenosis by exercising the muscles of abduction of the cords and mobilizing the crico-arytenoid articulation during the inspiratory effort. The forced respiration keeps the larynx freed from secretions, which are more or less purulent and hence irritating. After the cannula is removed, in order that healing may proceed from the bottom upward, the wound should be dressed in the following manner: A single thickness of gauze should be placed over the wound and the front of the neck, and a gauze wedge should be firmly inserted over this to the depths of the tracheotomic wound, all of this dressing being held in place by a bandage. If the skin wound heals before the fibrous union of the tracheal cartilages is complete, exuberant granulations are apt to form and occlude the trachea, perhaps necessitating a new tracheotomy for dyspnea.

It is so important to fix indelibly in the mind the cardinal points concerning tracheotomy that I have appended to this chapter the teaching notes that I have been for years giving my classes of students and practitioners, hundreds of whom have thanked me for giving them the clear-cut conception of tracheotomy that enabled them, when their turn came to do an emergency tracheotomy, to save human life.

### RÉSUMÉ OF TRACHEOTOMY

#### *Instruments:*

Headlight

Sand-bag

Scalpel

Hemostats

Small retractors

Tenaculum

Tracheotomic cannulæ (proper kind)

Tracheobronchial aspirator

Probe

Tapes for cannulæ

Trousseau dilator

Sponges

Infiltration syringe and solution

Oxygen tank

{ Long, half area cross-section trachea.  
 { Proper curve: radius too short will press anterior tracheal wall; too long, posterior wall.  
 { Sterling silver

*Indications.*—Laryngeal dyspnea.

(Indrawing guttural and clavicular fossæ and at epigastrium. Pallor. Restlessness. Drowning in his own secretions.)

Do it early. Don't wait for cyanosis. The onset of asphyxia is usually sudden; the last stage is short.

Never use general anesthesia on dyspneic patient.

Forget about "high" and "low" distinctions until trachea is exposed.

Memorize Jackson's tracheotomic triangle.

Patient recumbent, sand-bag under shoulders or neck. Nose to zenith.

Infiltration (intradermatic).

Incise from Adam's apple to guttural fossa.

Hemostasis.

Keep in middle line.

Feel for trachea.

Expose isthmus of thyroid gland.

Draw it upward or downward or cut it.

Ligature, torsion, etc., before incising trachea.

Hold trachea with tenaculum.

Incise trachea below first ring.

Avoid cutting cricoid or first ring. Cut three rings vertically. Don't hack. Don't cut posterior wall, which almost touches the anterior wall during cough. Spread carefully, with Trousseau dilator.

Insert cannula; see it enter tracheal lumen; remove pilot; tie tapes.

Don't suture wound. Dress with large squares.

Don't give morphine or any other antiepileptic.

Sedatives are unnecessary, always harmful, and often fatal.

Decannulation by corking partially, after changing to smaller cannula.

Do not remove cannula permanently until patient sleeps without indrawing with corked cannula.

#### RÉSUMÉ OF EMERGENCY TRACHEOTOMY

The following notes should be memorized:

1. Essentials: Knife and pair of hands (but full equipment better).
2. Don't do a laryngotomy, or stabbing.
3. "Two-stage, finger-guided" operation better.
4. Sand-bag or substitute.
5. Press back danger lines with left thumb and middle finger, making safety line and trachea prominent.
6. Memorize Jackson's tracheotomic triangle.
7. Incise exactly in middle line from Adam's apple to sternum.
8. Feel for tracheal corrugations with left index-finger in pool of blood, following trachea with finger downward from superficial Adam's apple.
9. Pass knife along index-finger and incise trachea (not too deeply, may cut posterior wall).
10. Don't mind bleeding; but keep in middle line and keep head straight; keep head low; don't bother about thyroid gland.
11. Don't expect hiss when trachea is cut if patient has stopped breathing.
12. Start artificial respiration.
13. Amyl nitrite. Oxygen.
14. Practice palpation of neck until tracheal landmarks are familiar.

15. Practice above technic, up to point of incision, at every opportunity.

16. *Jackson's tracheotomic triangle*: A triangulation of the front of the neck intended to facilitate a proper emergency tracheotomy.

Apex is at suprasternal notch.

Sides correspond to the anterior edge of the sternocleidomastoid muscles.

Base horizontal line lower edge cricoid.

#### RÉSUMÉ OF AFTER-CARE OF A TRACHEOTOMIC PATIENT

1. Always bear in mind that tracheotomy is not an ultimate object. The ultimate object is to pipe air down into the lungs. Tracheotomy is only a means to that end.

2. Sterile tray beside bed should contain duplicate (exact) tracheotomy tube. Trousseau dilator, hemostat, thumb forceps, silver probe, scissors, scalpel, probe-pointed curved bistoury. Sterile gloves ready.

3. Special nursing necessary for safety.

4. Laxative.

5. Sponge away secretions before they are drawn in.

6. Cover wound with wide large gauze square slit so it fits around cannula under the tape holder. Pull off ravelings. Keep wet with 1 : 10,000 bichloride solution.

7. Change dressing every hour or oftener.

8. Abundance of fresh air, temperature preferably about 70° F. If there is much secretion no moistener is needed; if there is little secretion a steaming vessel in the room is advisable; if there is crusting the air must be kept constantly at the saturation point by keeping a large vessel constantly boiling hard. Modern heating systems in the United States render the air, in winter, very dry.

9. *Nurse should remove inner cannula as often as needed and clean it with pipe-cleaner before boiling.*

10. Outer cannula should be changed every day by the surgeon or long-experienced tracheotomy nurse. A pilot should be used and care should be taken not to injure the cut ends of the tracheal cartilage.

11. A sterile bent probe may be inserted downward in the trachea with both cannulae out to excite cough if necessary to expel secretions. An aspirating tube should be used when necessary.

12. A patient with a properly fitted cannula free of secretions breathes noiselessly. Any sound demands immediate attention.

13. If the respiratory rate increases it is much more likely to be due to obstruction in, malposition of, or shortness of, the cannula than to lung complications.

14. Be sure that:

(a) The cannula is clear and clean.

(b) The cannula is long enough to reach well down into the trachea. A cannula that was long enough when the operation was done may be too short after the cervical tissues swell.

(c) The distal end of the cannula actually is deep in the trachea. The only way to be sure is, when inserting the cannula, to spread the wound and the tracheal incision with a Trousseau dilator, then *see* the interior of the tracheal lumen and *see* the cannula enter therein.

15. If after attending to the above-mentioned details there are still signs of obstructive dyspnea, a bronchoscopy should be done for finding and removing the obstruction in the trachea or main bronchi.

16. If all the "pipes," natural and instrumental, are clear, there can be no such thing as obstructive dyspnea.

17. Pneumonia and pulmonary edema may exist before tracheotomy, but they are rare sequelæ. Practically all deaths attributed to these conditions are simply unrecognized and easily removable obstructions in the "pipes," natural or artificial.

18. Decannulation, in cases of tracheotomy done for temporary conditions, should not be attempted until the patient has slept at least three nights with his cannula tightly corked. A properly fitted cannula (*i. e.*, one not larger than half the area of cross-section of the trachea) permits the by-passage of plenty of air. A partial cork should be worn for a few days first for testing and "weaning" a child away from the easier breathing through the neck. In cases of chronic laryngeal stenosis a prolonged test is necessary before attempting decannulation.

19. A tracheotomized patient may be aphonic, hence unable to call for help.

20. The foregoing rules apply to the postoperative periods. After the wound has healed and a fistula is established the patient, if not a child, may learn to care for his own cannula.

21. Do not give cough sedatives or narcotics. The cough reflex is the watch-dog of the lungs.

#### NOTES ON NURSING TRACHEOTOMIZED PATIENTS

The following notes were formulated by Dr. Ellen J. Patterson and the author many years ago. Revisions since have been based on experience in teaching hundreds of nurses how to care for these patients. A copy of these notes should be given to every nurse when she takes charge of a tracheotomized patient:

Bedside tray should contain	{	Duplicate cannula	Scalpel
		Trousseau dilator	Hemostat
		Dressing forceps	Sterile vaseline
		Scissors	Tape
		Probe	Gauze sponges
		Probe-pointed curved bistoury	Gauze squares

1. Room should be abundantly ventilated, as free from dust and lint as possible, and the air should be moistened by steam in winter.

2. Keep mouth clean. Tooth brush. Rinse alcohol 1 : 10.

3. Sponge away secretion after the cough before drawn in.

4. Remove inner cannula (not outer) as often as needed. Not less often than every hour. Replace immediately. Never boil a cannula until you have thoroughly cleaned it.

5. Obstruction of cannula calling for cleaning indicated by: Blue or ashy color; indrawing at clavicles, sternal notch, epigastrium; noisy breathing (learn sound).

6. Surgeon (in our cases) will change outer cannula once daily or oftener. Nurse should not remove it unless specially instructed.

7. Duplicate cannulæ.

8. Be careful in cleaning cannula not to damage it.

9. Watch for loose parts on cannula.

10. Change dressings (in our cases) as often as soiled. Not less often than every hour. Large squares. Never narrow strips.

11. Watch color of lips and ears and face.

12. Report at once if food or water leaks through wound. (Coughing and choking.)

13. Never leave a tracheotomized patient unwatched during the first days or weeks, according to case.

14. Remember the Trousseau dilator or a hemostat will spread the tracheal wound or fistula when the cannula is out.

15. Remember life depends on a clear cannula if the patient gets no air through the mouth.

16. Remember it takes very little to clog the small cannula of a child.

17. Remember a tracheotomized patient cannot call loudly for help.

18. Decannulation. Testing by corking partially. Watch that corks are not too small or broken. Attach them by braided silk thread. Pure rubber cord ground down makes the best cork; bark corks are dangerous.

19. An uncorked cannula requires a warning to the patient when he is to do his own bathing. Inspiration of small quantities of water is dangerous; an attempt at swimming would be promptly fatal.

CHEVALIER JACKSON and CHEVALIER L. JACKSON.

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#### THE HISTOLOGICAL DIAGNOSIS OF LESIONS OF THE LARYNX

##### (From Biopsy)

**Inflammatory Lesions.**—Acute inflammatory lesions of the larynx rarely have to be considered from the histological standpoint, but the chronic inflammatory lesions occupy an important place among the specimens submitted for examination. Unfortunately, in many cases there is nothing characteristic in the histological picture to indicate the cause of the lesion, the mucous membrane may or may not be ulcerated, the epithelium is usually hyperplastic, the submucosa is infiltrated by leukocytes and fibrosed. However, in tuberculous lesions and some of the fungus infections characteristic changes in the tissue are observed. In chronic tuberculosis of the larynx the mucosa may or may not be ulcerated, the submucous tissue is the seat of diffuse mononuclear cell infiltration, is fibrosed, and usually contains miliary foci of mononuclear and giant-cells, forming fairly typical tubercles. As a rule there is very slight hyperplasia of the

epithelium. In fungus infections, such as blastomycosis, there are marked hyperplasia of the epithelium, ulceration, intense mononuclear and polymorphonuclear cell infiltration of the submucosa, the presence of giant cells and miliary foci. A positive diagnosis is based upon the demonstration of the fungi in the stained sections and the cultivation of the specific fungus from the lesion.

In chronic inflammatory lesions of long standing a biopsy is performed more frequently to determine whether or not there is malignant change rather than with the hope of finding the true cause of the inflammation. The relation of chronic inflammatory lesions to malignancy will be discussed in a subsequent paragraph.

**Papilloma** is the most frequent benign neoplasm of the larynx. There are two main varieties, namely, simple or single papilloma and the villous



Fig. 547.—Section from papilloma of larynx. (Slide studied by Dr. Ophüls.)

type. The former is usually composed of a single projection with a central stem of connective tissue covered by a membrane of stratified squamous epithelium of varying thickness.

The villous papilloma is composed of innumerable small projections with central stems of connective tissue covered by stratified squamous epithelium, the small projections springing from a single central base. The layer of epithelium covering the papillæ varies greatly in thickness, but the normal transition from the basal layer to the flat squamous-cell stratum on the surface is maintained.

It must be constantly borne in mind that the papillomas may undergo malignant change, especially if associated with chronic inflammatory processes. If the epithelium is irregular, and lacks normal transition from the basal layer to the surface layer, the lesion should be regarded with grave suspicion and the base of the papilloma especially should be examined

carefully for evidence of malignant change. Two cases, in which the papilloma had either undergone carcinomatous change or preceded the carcinoma, came under our observation recently. One case with villous papilloma which manifested no histological evidence of malignancy returned two years later with a definite carcinoma. In the second case a papilloma (Fig. 547) had been removed eight years prior to the development of a carcinoma at the same site (Fig. 550). However, it seems that the true villous type of papilloma rarely becomes malignant, even though it frequently recurs after removal. We have examined specimens from cases of recurrent papilloma over a period of years without observing the slightest evidence of malignant change. However, this benign lesion must not be confused



Fig. 548.—(From patient of Fielding Lewis.) Section from larynx of case of pachydermia laryngis, diffuse, revealing the markedly thickened epithelial surface with hyperkeratosis. Note the regularity of the basal layer of the epithelium. No evidence of malignancy was observed in any of the sections taken from different parts of the larynx.

with papillary carcinoma or the papilloma which is composed largely of hyperplastic epithelium and is potentially malignant. In the latter there is more irregularity in size, shape, and staining characteristics of the epithelium; many of the nuclei are undergoing mitosis, and there is total absence of normal transition of epithelium from the basal layer to the flat, squamous-cell stratum on the surface. When only the top of such a lesion is removed at biopsy it may closely resemble an ordinary papilloma, but the changes in the epithelium should at least suggest the true malignant nature of the lesion, even though definite evidence of infiltration by the epithelium cannot be demonstrated. If the base of this type of lesion is examined, definite infiltration by the epithelium is usually found. In a case of this type which came under our observation we failed to recognize the malignant

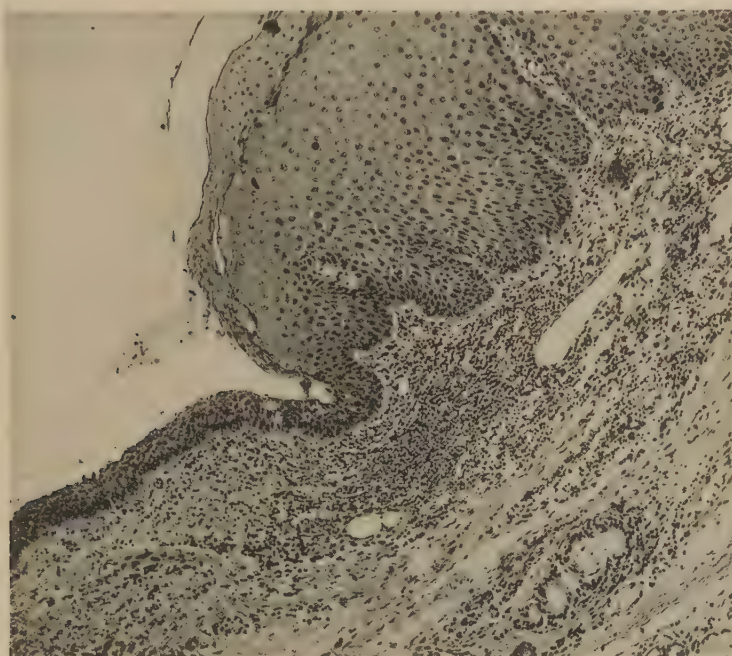


Fig. 549.—Same patient as in Fig. 548. Section from lower portion of larynx revealing the sharp line of demarcation between the thickened laryngeal epithelial surface and the tracheal epithelium. Note the inflammatory reaction in the submucous tissue.

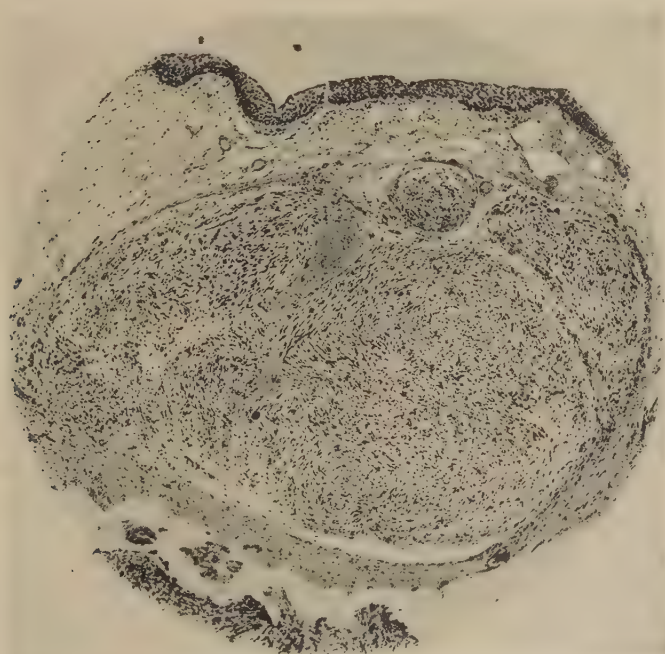


Fig. 549a.—Section from larynx revealing submucous neurofibroma (pp. 902, 903). (From patient of Chevalier Jackson.)

nature of the lesion when first examined histologically. After removal there was rapid recurrence and the histological examination of the base of the growth revealed extensive infiltration by the epithelium.

**Pachydermia laryngis** is usually a localized area of hyperplasia of the epithelium with hyperkeratosis which is frequently considered potentially malignant. There is often an associated inflammatory process in the submucous tissue. In a case of long duration recently studied, however, there was diffuse, marked thickening and hyperkeratosis of the surface epithelium of the entire larynx, in which no histologic evidence of malignancy was found (Figs. 548, 549).

**Other Benign Neoplasmas of Larynx.**—*Fibromas* occur more frequently in conjunction with papilloma and angioma than as a pure fibroma. *Fibro-an-*

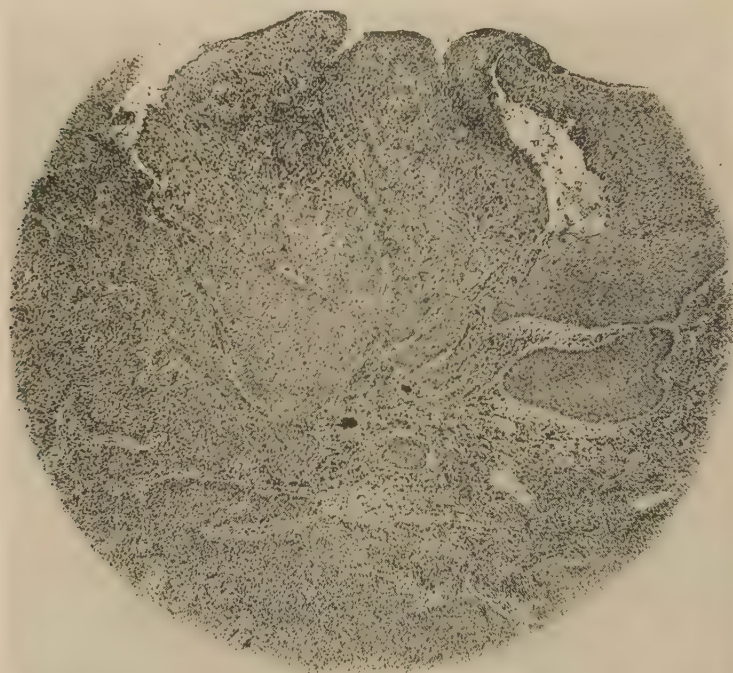


Fig. 550.—The same patient as in Fig. 547. Squamous-cell epithelioma developing at the site where the papilloma had been removed eight years previously.

*gioma* is a lesion that frequently occurs in the larynx, and involves especially the vocal cords. It probably should not be classified as a true neoplasm; however, its gross and histologic appearances are so constant, and inflammatory reaction so completely absent, that it more closely resembles a neoplasm than any other type of lesion. It usually presents as a small circumscribed but not encapsulated nodule in the submucosa, and is composed of loose connective tissue in which there are small blood-vessels and channels lined with endothelium and containing blood or granular material. The overlying mucosa is usually intact. *Chondromas* arise from cartilages of the larynx and are usually composed of rather cellular hyaline cartilage. In our series of 643 neoplasms of the larynx there were three chondromas. *Neurofibroma* is rarely observed in the larynx. We had an opportunity of studying one case.

Chevalier Jackson removed a mass of submucous nodules in the larynx, section from which presented the typical histology of neurofibroma (Fig. 549a, and Figs. 468, 469, p. 902).

**Sarcoma** of larynx has been reported by a number of authors. Ewing<sup>3</sup> states: "Sarcomas form 11 per cent. of malignant laryngeal growths (Molinié)." He further states: "It is probable that many cases recorded as sarcoma are of epithelial origin." In our series of 643 neoplasms of the larynx at the Chevalier Jackson Bronchoscopic Clinic we have not observed one of true sarcoma. We have studied a number of specimens (biopsies) in which the lesion, histologically, closely resembled sarcoma; further examination, however, proved that they were not true sarcoma, but probably belonged to the lymphoma and cytoma group.

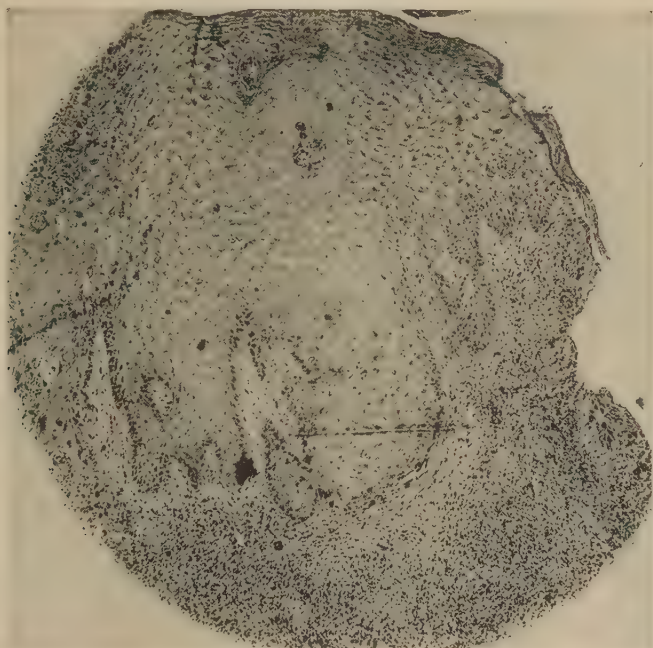


Fig. 551.—Chronic inflammatory lesion of the larynx with marked hyperplasia of the epithelium.

**Carcinoma** is the most frequent malignant neoplasm of the larynx; it is usually the squamous cell type, and is easily recognized in advanced stages. Hyperplastic and hyperchromatic epithelium, infiltrating the submucous and deeper structures, presents a typical histological picture which is not confused with other lesions. The infiltrating cells manifest marked variation in size, shape, and staining characteristics, and varying degrees of differentiation; many of their nuclei are undergoing mitosis (Fig. 553). The lesion may be ulcerative, with tumor cells infiltrating the deeper structures, or it may form a nodule, with the tumor cells piling above the surface as well, and sometimes it is definitely papillomatous. The advanced lesion needs no further discussion, and it is readily recognized histologically.

In early or borderline cases the true nature of the lesion is not easily

determined. It is recognized that long-standing chronic inflammation of the larynx is frequently followed by carcinoma. Attention has been called to this fact by Jackson<sup>1</sup> who cites a number of cases which were clearly inflammatory, clinically and histologically, when first examined, but subsequently developed carcinoma. In a case recently studied the lesion of the larynx presented the histological picture of a chronic inflammatory process with marked hyperplasia of the epithelium (Fig. 551), but the characteristic changes of malignancy could not be demonstrated. The patient did not return for examination for three years, at which time the lesion was frankly carcinomatous (Fig. 552). However, it would be entirely erroneous to consider all the lesions of chronic inflammation with hyperplasia of the epithelium, as potentially malignant, for in many instances the lesion

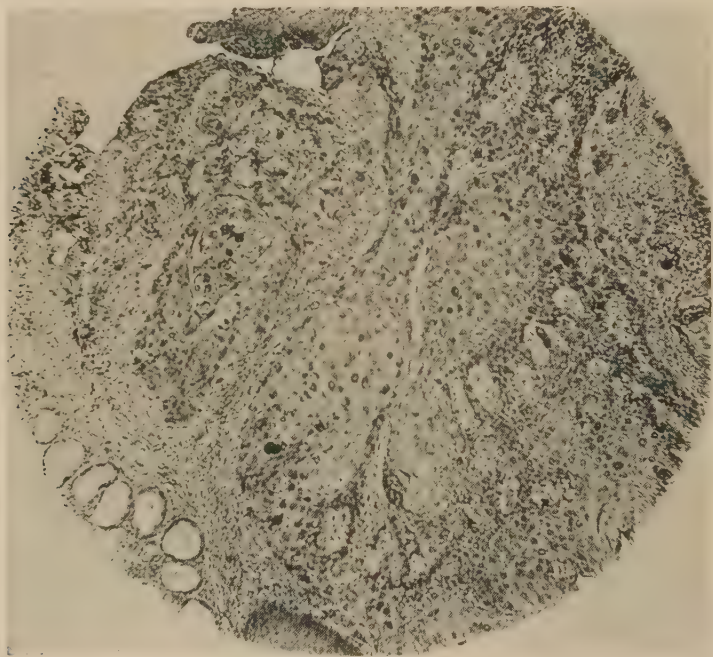


Fig. 552.—Sections from the same patient as in Fig. 551 three years later, showing definite carcinoma.

clears up entirely under proper treatment. For example, in a case of lues of the larynx which was studied some time ago, the nature of the lesion was not at first recognized, as history of lues and serological reactions were negative. Sections from the biopsy presented extensive inflammatory reaction with marked changes in the epithelium. Several capable pathologists who examined the sections regarded the lesion as undergoing early malignant change, but we did not consider it as frankly carcinomatous. The lesion promptly disappeared under antiluetic treatment, which proved that the growth was not malignant. On the other hand, it must be remembered that in any ulcerative lesion of the larynx, such as carcinoma, marked secondary inflammatory reaction is always present, and if a representative piece of tissue is not studied the inflammatory lesion may overshadow

the changes in the epithelium. Frequently the pathologist is not justified in rendering a definite opinion as to the malignancy or non-malignancy of a lesion merely from the histological picture. He can only report on the tissue submitted for study. Changes in the epithelium, such as marked irregularity in size, shape, and staining characteristics of the cell and lack of differentiation, indicate malignancy rather than the extent of hyperplasia or the location of the epithelium. After a careful study of serial sections the report should be based upon proper interpretation of actual changes in the tissue and not upon impressions or fancies. Lesions have been observed in tissue removed for biopsy, which were very suggestive of malignant change, but upon removal of entire lesion by laryngofissure no definite evidence of malignancy could be demonstrated. Unfortunately, we cannot

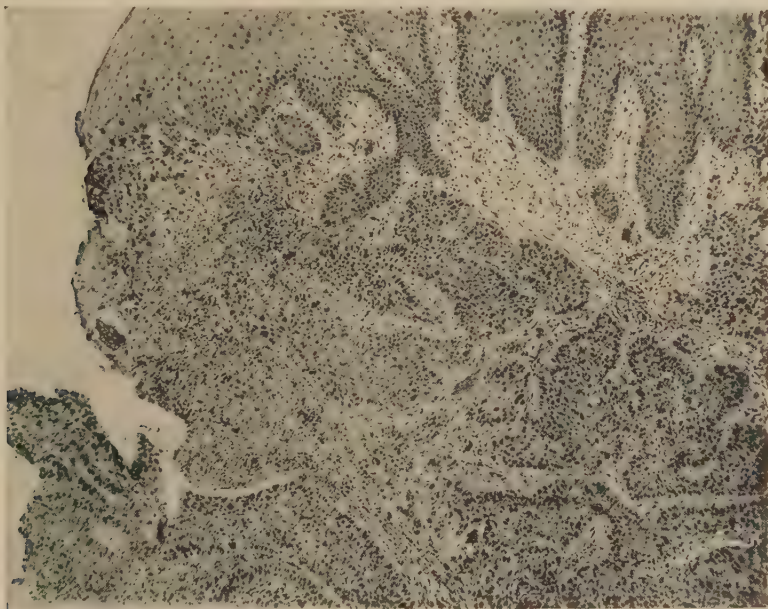


Fig. 553.—Advanced squamous-cell epithelioma of larynx. Section from the margin of the lesion, revealing the transition from the normal to the infiltrating epithelium. (From a patient of Chevalier Jackson.)

predict with any degree of accuracy whether a suggestive lesion will become malignant or not. Coplin<sup>2</sup> has aptly said, "Histology rarely, if ever, tells what a cell is going to do before it does it, although many of us are constantly deluding ourselves with the belief that we can deduce the future of the cell from a study of the past or present, or both."

**Technic.**—In doubtful cases important factors in arriving at a correct conclusion as to the true nature of the lesion are the tissue removed and the technic employed in preparing such tissue for histological study. It is very important that a representative piece of tissue be removed at biopsy and the quickest method employed for preparing the specimen which will yield satisfactory sections for a careful histological study. The frozen section method is not applicable in a majority of cases, as the specimen removed is usually very small, frequently not more than a few millimeters

in thickness and in questionable cases it is very important to have serial sections for study. Very good sections may be obtained by the rapid paraffin method which requires from six to seven hours for completion.

BAXTER L. CRAWFORD.

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### MECHANICAL DEVICES FOR OBTAINING OPERATIVE ACCESS TO THE LARYNGOPHARYNX, LARYNX, AND HYPOPHARYNX

For minor operations in these regions, such as the removal of benign growths, and specimens of tissue in cases of suspected malignant growths, most operators prefer the coöperative manipulative aid of a specular instrument held in the left hand. For more extensive procedures requiring hemostasis and other bimanual manipulations, and when the route through the mouth is preferred to external surgery, a mechanical device for exposure of the field of operation is desirable. Considering the marvelous development of the gynecologists in extensive surgical procedures by the vaginal route it seems that the oral route for extensive pharyngeal operations could be used to a greater extent than it has been, though the conditions are, of course, not the same. The possibilities of extensive operation by the oral route, however, are demonstrated by Yankauer's laryngectomies.

The Whitehead mouth-gag and the Brophy oral speculum are useful for certain procedures, but they do not give access below the fauces. For operative exposure of the laryngopharynx the suspension apparatus of Lynch is excellent.

**The Pharyngoscope.**—In suspension laryngoscopy operative exposure of the laryngopharynx is obtained by the sagging weight of the head when the patient's head is suspended on a spatular hook inserted posterior to the tongue and body of the hyoid bone. The spatular hook hangs from a gallows attached to the table. With the pharyngoscope of Haslinger the displacement forward of the base of the tongue and of all the tissues attached to the hyoid bone is obtained by a powerful screw operating a lever with the cervical spine as a point of counterpressure.

CHEVALIER JACKSON.

### SUSPENSION LARYNGOSCOPY

Suspension laryngoscopy was discovered by Professor Gustave Killian in 1909 and presented to the profession at the International Congress at Buda Pest in 1911.

It consists in converting the road from the upper teeth to the vocal cords into a straight line by means of modified tongue spatula and mouth-gag, which is suspended from an overhanging moving crane.

The spatula which I use has the advantage over the original models of Killian and others in that it is strong, rigid, and yet sufficiently adjustable to meet all conditions.

Various changes in the technic have taken place until at the present time it is simple and safe and yet affords the best view of any of the modifications.

It can be used either with local or general anesthesia, though where much operative work is to be done of a delicate nature, general anesthesia is recommended.

The technic is as follows: With the patient properly prepared as for any surgical procedure, and lying on any flat operating table where suitable rigid support can be had for the traveling crane (my table has a wooden top), the head is extended freely so that the occiput approaches the nape of the neck. An ordinary mouth-gag which fits over the molar teeth on one side will keep the mouth open during the introduction of the spatula. A small piece of sheet lead  $\frac{1}{16}$  of an inch thick and  $\frac{3}{4}$  of an inch wide and  $1\frac{1}{2}$  inches long, is molded to protect the biting edge of the teeth. This in place, the spatula is introduced (with the tongue piece following over the dorsum of the tongue) until the laryngeal face of the epiglottis is reached, at this instance the tooth plates of the mouth-gag on the spatula will fall behind the upper teeth. Being careful now to keep the tongue spatula in the middle line, the mouth-gag is opened by means of a knurled screw on the handle of the spatula, and it is best to open the gag wide at this time. The handle of the spatula is now pulled toward the operator who sits at the head of the table, and by this means the tip of the spatula is raised against the laryngeal face of the epiglottis and the larynx comes into view.

The hook on the handle is now placed over the traveling crane which has been previously fixed to the table, and is made to raise vertically which lifts the weight of the patient's head hardly free of the table. This is sufficient in most instances to give a good view of the larynx. If further view is necessary, lifting vertically increases the view or moving the crane horizontally toward the head of the table, or turning the screw which moves the worm-gear joint on the handle of the spatula, changing the angle between the point of support and tip of the spatula. All have a tendency to straighten the line from the upper teeth to the larynx and widen the view to the anterior commissure of the vocal cords, which is the goal to be accomplished. With the vocal cords held in view without the aid of an assistant or one's own hands, one is free to do whatever may be indicated. One sees the sides of the base of the tongue the glosso-epiglottic folds, a part of the base of the epiglottis, arytenoids, interior of the larynx in all its detail, trachea usually to the bifurcation, the sinuses of the larynx, upper end of esophagus, postpharyngeal wall to uvula, tonsils, and lateral pillars, etc.

These areas may not only be inspected but palpated by means of any suitable instrument and any operative procedure practised upon these parts with both hands free to work, knowing that the field of view will not be disturbed or lost by slipping instruments, aching hands, or patient's movements.

Any pathology which fixes the head with the chin flexed on the sternum, such as a cervical Potts or a thick contracted scar beneath the chin and extending to the sternum will make a view of the larynx by this means impossible. Otherwise all cases can be seen by this means.

The dangers are very few, as I have had none to record in the past five years.

R. C. LYNCH.

## PART V—PERORAL ENDOSCOPY

**Definition.**—"Peroral endoscopy" has come into general use as a convenient term to cover the examination of the interior of the larynx, trachea, bronchi, esophagus, or stomach by the procedures of direct laryngoscopy, bronchoscopy, esophagoscopy, and gastroscopy. These procedures are, essentially, specular examinations; but they differ from other specular exposures in requiring a special technic not only for their successful performance, but to avoid serious or fatal injury to the patient. The accomplishments of bronchoscopy alone, as pointed out by Keen, have "revolutionized a whole department of surgery." Esophagoscopy in the hands of experts has removed the esophagus from the realm of inference; it is no longer considered justifiable to treat the esophagus without looking into the esophagus.

The field has extended so widely that only a brief consideration of fundamental facts can be given within the limits of this book. The subject is fully treated in books devoted to the subject.<sup>1, 2, 3</sup>

**Dangers and Complications.**—When it is skilfully introduced, there is absolutely no danger whatever from the mere presence of a bronchoscope in the laryngotracheobronchial air-way, nor of an esophagoscope in the esophagus and stomach. On the other hand, when an otherwise skilful, but endoscopically untaught man starts to introduce either of these instruments into a dyspneic baby, the chances of the baby's survival are exceedingly remote. It is true that any physician or surgeon can be taught how to introduce these instruments safely; yet it is equally true that he cannot learn how to introduce them by looking on at a clinic. In principle, the bronchoscope, the esophagoscope, and the gastroscope are specula; but their introduction is highly technical as compared to the introduction of a vaginal or rectal speculum. If an esophagoscope is inserted into the pharynx and simply pushed downward, the one place it will *not* go is into the esophagus. Only a slight push is necessary to send it through the hypopharyngeal wall, after which it will meet with less resistance in its progress down into the mediastinum than it would if it were going down inside the esophageal lumen.

**Dyspnea.**—A patient dyspneic from true asthma is in no particular danger from bronchoscopy; but a patient supposed to have asthma but really dyspneic from mechanical obstruction of the air-way by disease or by foreign body, is likely to die on the table unless handled by a team of three, all trained to act together with promptness and precision. The danger in dyspneic patients, especially babies, may be as great in esophageal as in laryngotracheal cases for the reason illustrated in Fig. 554. Direct laryngoscopy should never be attempted without preparation for the insertion of a bronchoscope in case of dyspnea, whether the patient seems dyspneic or not.

**Trauma.**—Apart from the trauma of false passage of instruments by the untaught, fatal trauma may be inflicted by improper attempts to deal

with a foreign body. A trained man may harmlessly manipulate a safety-pin for half an hour, because he knows that "advancing points perforate, trailing points do not."<sup>3, 8, 10</sup> Furthermore, he not only *knows* this, but he is

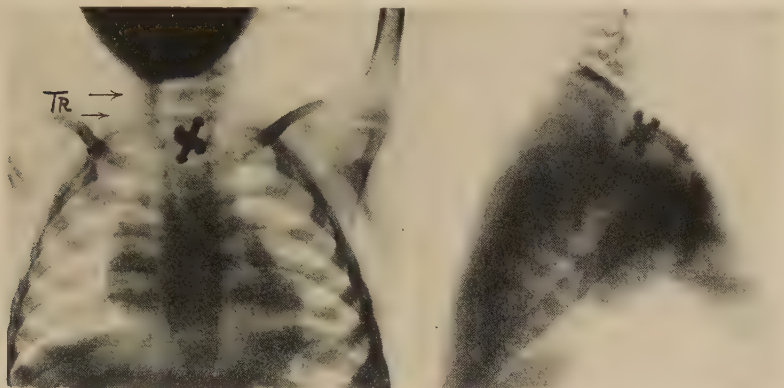


Fig. 554.—Roentgenogram of a child aged four months with extreme dyspnea from a foreign body (toy jack) in the esophagus. The displacement of the "party wall" by the foreign body in the esophagus between the trachea and the unyielding spine has nearly obliterated the lumen of the trachea (*TR*). The violent inspiratory efforts have drawn air into the *stomach* (*S*) through the *esophagus* because the trachea was so obstructed that not enough air entered to satisfy the negative pressure created by the violent inspiratory efforts.

trained until it is impressed upon his subconscious mentality that only an exceedingly slight pull is required to drive a point through the wall of either the esophagus or the bronchus. The same principle applies to all pointed objects: nails, pins, tacks, hooks, staples, etc.

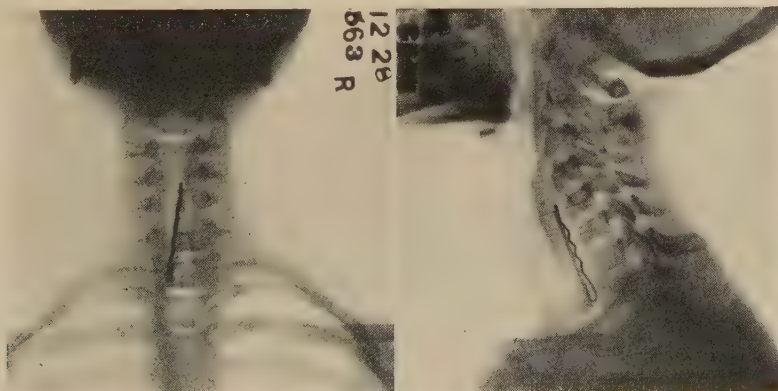


Fig. 555.—Roentgenograms, anteroposterior and lateral, of a girl aged sixteen years showing a "bobette pin" in the esophagus. Had only the anteroposterior roentgenogram been taken, the erroneous diagnosis of foreign body in the trachea would have been made. The lateral view, however, demonstrates the foreign body to be behind the trachea and in the esophagus. A lateral as well as an anteroposterior roentgenogram is necessary in every case of foreign body in the esophagus.

**Mediastinal emphysema, pneumothorax, and septic mediastinitis** are rare complications. They may occur spontaneously before bronchoscopy or esophagoscopy,<sup>3, 10</sup> or may be due to perforation by a safety-pin or other

foreign body under manipulation. The gravity of these complications should always be in the mind of the peroral endoscopist.

**Mortality.**—Considered apart from the condition for which it is done, there is no mortality attached to the skilful insertion of a bronchoscope or esophagoscope. Taking all the cases as they come, those with essentially fatal visceral diseases, such as mediastinal lymphosarcoma, advanced cancer, etc., as well as those presenting difficult mechanical problems of foreign-body extraction or foreign-body pathology, the mortality, taken over a period of ten years, is not over 1.7 per cent. In 1926, at the Chevalier Jackson Clinics, there were 4656 peroral endoscopies done by eight individual members of the personnel and course graduates. During this period there was but one death shortly after bronchoscopy, and in that case there was no autoptic evidence to indicate that death was directly or indirectly attributable to the endoscopy.

**Contraindications to Bronchoscopy, Esophagoscopy, Direct Laryngoscopy, and Gastroscopy.**—The *contraindications to peroral endoscopy for foreign body* are few, and none would absolutely contraindicate endoscopic removal of a foreign body.<sup>3</sup> High blood-pressure, advanced cardiovascular disease, aneurysm, active syphilis or tuberculosis, and other organic diseases must be weighed against the urgency of the indications for the procedure, and call for preparatory treatment of the patient. Pneumonia is no contraindication, and in foreign-body cases the supposed pneumonia is usually an error in diagnosis.<sup>14</sup> In cases of embolic abscess a moribund condition of the patient may contraindicate bronchoscopy, not because bronchoscopy would hasten the end but because it would be powerless to prevent it. In babies and in very young children bronchoscopy should not be prolonged beyond twenty-five minutes, and repetition of the procedure is contraindicated without an interval of a few days, preferably a week. The contraindications to endoscopy for disease will be found in the article on that subject.

**Preparation of the Patient.**—Asepsis of the field is impossible; but a clean mouth should be insisted upon in adults and older children. Examination of the mouth for artificial dentures, bridge work, loose crowns, deciduous teeth, etc., is essential in order to forestall accidents in the use of the bite-block and tube. Examination of the nose, the fauces, pharynx, and larynx is always essential. Mirror-examination of the larynx for a local lesion or a recurrent paralysis should never be omitted. Rest and relaxation in bed is a desirable preliminary to the first endoscopy, but is dispensed with after the first, in ambulatory patients. A laxative is advisable, especially if a sedative is to be used.

**Care of Patients After Direct Laryngoscopy, Bronchoscopy, Esophagoscopy, and Gastroscopy.**—After direct laryngoscopy the patient should remain in the hospital over night, and longer if necessary, because of the possibility of laryngeal reaction with stenosis. An exception may be made to this rule in patients wearing a tracheal cannula and attended by someone competent to care for it.

In cases of chronic disease of the esophagus, no anesthetic or sedative being used, the patients, adults or children, come in, get on the table, are treated, and then go home or back to work. In chronic suppurative disease of the lung, most patients are kept in bed a large part of every twenty-four hours as a useful adjunct to medical care. If this is carried out at home

the patients come in at the appointed time for bronchoscopic aspiration and return home immediately after treatment. Hospitalized patients rest in bed until the next morning. In patients with slight hemoptysis, rest in bed is essential. Severe hemorrhage is a contraindication to bronchoscopy, except in rare cases in which bronchoscopy is done to arrest bleeding.<sup>12</sup>

In very young children, especially in cases of vegetal bronchitis from inspired peanuts and other nut kernels, beans, peas, watermelon seeds, fruit pits, etc., there is usually as a part of the diffuse laryngotracheobronchitis a swelling of the subglottic tissues. This causes a "croupiness," and in children under two years of age the swelling may be so great as to require tracheotomy for obstructive laryngeal dyspnea. For this reason, children are always kept in bed in the hospital after bronchoscopy for two or three days for observation. In cases of lung suppuration from prolonged sojourn of a foreign body, the patient usually requires no after-treatment other than rest in bed under fresh-air conditions, and he is usually sent home within a few days after bronchoscopy for this to be carried out under the care of the family physician.

Children under treatment for suppuration of other than foreign body origin are brought in for bronchoscopic aspiration and are allowed to go home immediately afterward. Not using an anesthetic, general or local, makes this possible. Nevertheless, for its therapeutic effect, plenty of rest in bed is essential for chronic pulmonary suppuration under any method of treatment (Pritchard), and rest in bed outdoors is best.

After bronchoscopic pneumonography, we usually keep the patient under observation for a few days, though we have never seen any untoward result. A Roentgen-ray examination is usually made after a few weeks to record the disappearance of the bismuth or lipiodol. Subsequent Roentgen-ray examinations are indicated in a few cases.

**Preliminary Training of the Bronchoscopist and Esophagoscopist.**—As with all purely manual procedures, education of the eyes and fingers is essential to success; additionally, in peroral endoscopy it is essential to safety of the patient. Anyone who regards it beneath his dignity preliminarily to educate his eyes and fingers in the technic of the cataract operation by practice on sheep's or pig's eyes from butchered animals had better not attempt a cataract operation on a living human being. The dead eye affords better preliminary practice anyway, because work on it is free from anxiety or stress. And the cataract operation is done under binocular control. Peroral endoscopy is done under monocular control, which is not only unusual but involves depth perception with one eye, a thing that is difficult to acquire to a useful degree, and impossible to acquire to perfection. Absolutely nothing will take the place of education of the eye *at the tube*. Fortunately, a rubber-tube manikin affords an easy, handy, care-free, always ready means of education of the eye and the fingers in all the essentials. The man who will use his spare moments for manipulating various foreign bodies in a rubber tube under guidance of the eye will, with a little training on the cadaver as to the dangers to be avoided and how to avoid them, and on the dog for the problems presented by the movements of the living bronchi, soon make a safe and successful bronchoscopist. Of course, it is essential to have training on the living human being also; but we never permit our pupils to attempt to introduce a

bronchoscope or esophagoscope until after they have had at least two weeks of intensive instruction and practice on rubber tube, dog, and cadaver. Most of these pupils have been practicing otorhinology for

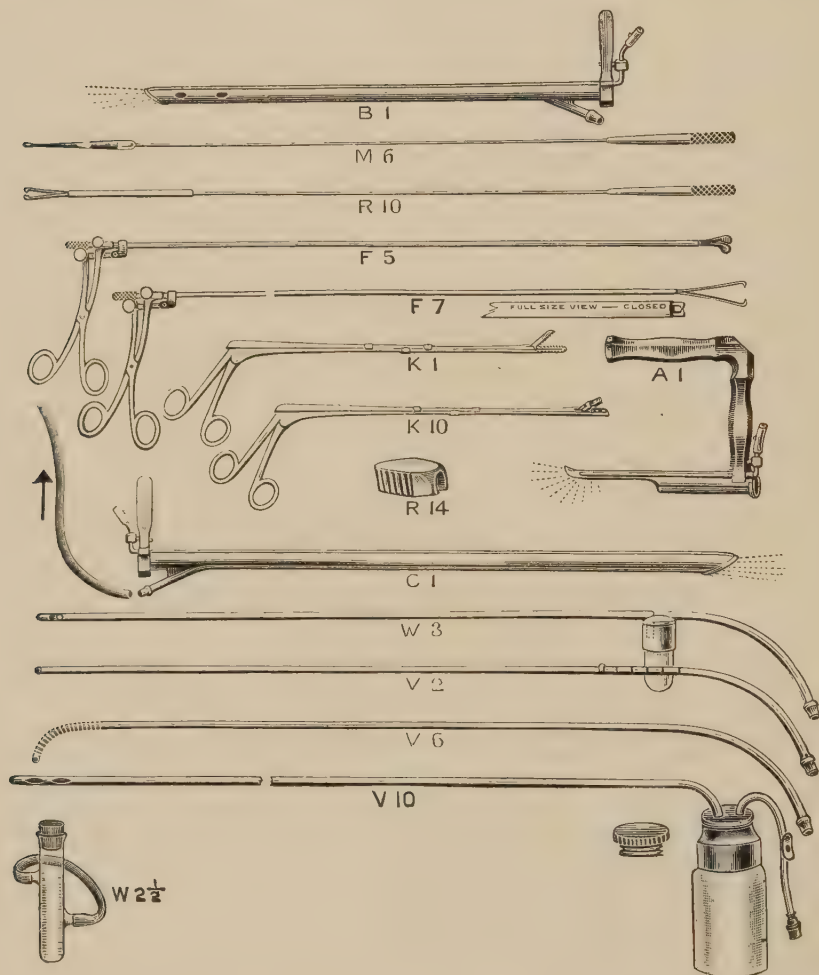


Fig. 556.—Peroral endoscopic instruments. A1, Direct laryngoscope. B1, Bronchoscope. C1, Esophagoscope with aspirating canal. M6, Esophagoscopic bougie for safe dilatation under guidance of the eye. R10, sponge carrier for gauze sponges; the sponges are not shown, but are absolutely essential—at least 4 dozen of the proper size for each size of tube to be used should be in readiness on the sterile table. F5, Esophagoscopic and bronchoscopic forceps. F7, Rotation forceps. K10, Laryngeal forceps for taking specimens of tissue; a longer form of this forceps is needed for bronchoscopic and esophagoscopic use. W3, Clerf collecting tube. W2 $\frac{1}{2}$ , Lukens' specimen collector. V2, Aspirating tube with "warning stop." V10, Chevalier L. Jackson esophageal evacuator. V6, Spiral tipped aspirating tube (Lynah). R14, Moore thimble bite-block. Adult and child sizes of instruments A1, B1, and C1 are necessary. Messrs Pilling & Sons supply two extra light carriers for endoscopic tubes which is a great convenience.

years, and hence have had hundreds of hours of practice in depth perception with one eye only. In case of a man who has had training only as a surgeon using both eyes and both hands in open wounds, we would

regard a longer preliminary practice with the rubber tube essential even for endoscopy for disease unconnected with foreign body. The early endoscopies on living human beings should be on adults, later ones on

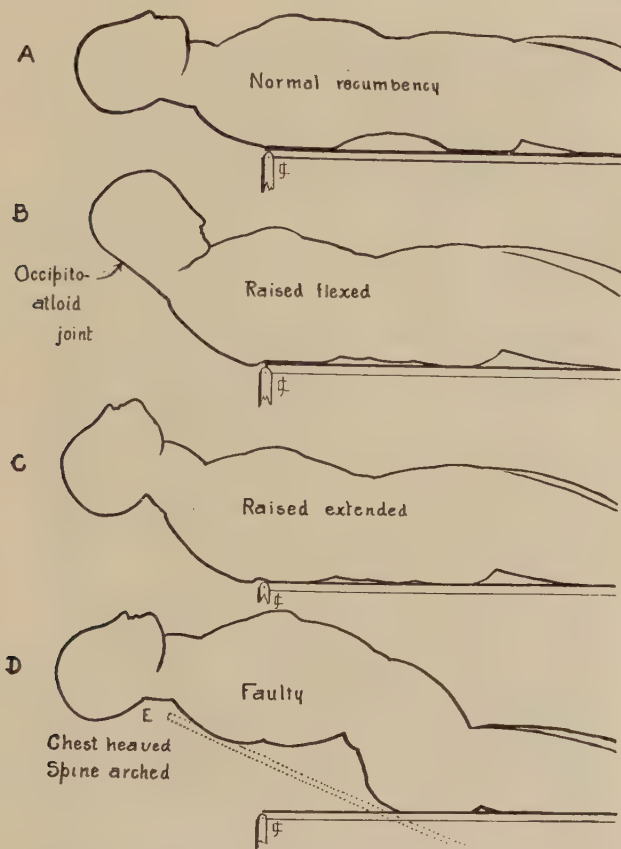


Fig. 557.—Schema of position for endoscopy. A, Normal recumbency on the table with pillow supporting the head. The larynx can be directly examined in this position, but a better position is obtainable. B, Head is raised to proper position with head flexed. Muscles of front of neck are relaxed and exposure of larynx thus rendered easier; but, for most endoscopic work, a certain amount of extension is desired. The elevation is the important thing. C, The neck being maintained in position B, the desired amount of extension of the head is obtained by a movement limited to the occipito-atloid articulation by the assistant's hand placed as shown by the dart (B). D, Faulty position. Unless prevented, almost all patients will heave up the chest and arch the lumbar spine so as to defeat the object and to render endoscopy difficult by bringing the chest up to the high-held head, thus assuming the same relation of the head to the chest as exists in the Rose position (a faulty one for endoscopy) as will be understood by assuming that the dotted line, *E*, represents the table. If the pelvis be not held down to the table the patient may even assume the opisthotonos position by supporting his weight on his heels on the table and his head on the assistant's hand. It cannot be too strongly emphasized that the whole support and manipulation of the head devolves upon the *left hand*. If any support is given by the right forearm (certain to occur if not guarded against), the cervical spine will be rendered convex and introduction will be correspondingly difficult.

older children; only after prolonged practice and experience is one justified in attempting to put an endoscopic tube down the tender passages of a baby.

**Instrumentarium.**—The commonly used instruments are shown in Fig. 556. An adequate equipment must include sizes for adults and children. Obviously instruments suitable for the removal of a bit of peanut kernel from the lung of a baby would not serve for the removal of a huge artificial



Fig. 558.—Position of patient and assistant for introduction of the bronchoscope and esophagoscope. The middle of the scapulae rest on the edge of the table; the head and shoulders, free to move, are supported by the assistant, whose *right* arm passes under the neck; the *right* middle finger inserts the bite-block into the *left* side of the mouth. The *left* hand, supported by the *left* elbow resting on the *left* knee maintains the desired degree of elevation, extension, and lateral deflection required by the operator. The patient's vertex should be 10 cm. higher than the level of the top of the table. It is essential for the assistant to have constantly in mind that the *right* forearm must not support the neck; all support must be of the head only and must come from the *left* hand; and that this *left* hand must not rest on the knee, only the *elbow*. The second assistant is holding the patient's shoulders firmly down on the table. This is very important. The sterile cover has been moved aside to expose the patient's shoulder sufficiently to demonstrate this point. The nurse is holding down the patient's wrists; her left thorax and arm keep down the patient's knees if necessary. This is the Boyce position, which has never been improved upon for bronchoscopy and esophagoscopy. Precisely this position is of the utmost importance, and drill is necessary before it can be assumed promptly in emergencies and maintained comfortably for a long time when necessary. The foot-rest is a box, 10 by 12 by 14 inches, to give choice of height, with stability.

denture from the esophagus of a muscular adult. Failures and fatalities follow attempts at endoscopy with inadequate equipment.

**Position of the Patient.**—For all peroral endoscopic procedures the patient is placed in the position shown in Figs. 557 and 558. The purposes

of this position is twofold: (1) To place the anatomical structures of the patient in the best position for entrance of straight and rigid tubes; and (2) to control the patient. The slightest deviation from precisely this position will render peroral endoscopy difficult or impossible. Only too often this position, which has been carefully worked out in every detail at the bronchoscopic clinic, has been rendered hopelessly misleading by careless redrafting, as if it were no more important than grouping a crowd for portraiture.<sup>1, 2, 3</sup>

CHEVALIER JACKSON and CHEVALIER L. JACKSON.

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#### DIRECT LARYNGOSCOPY

As mentioned in a former section, direct laryngoscopy is in principle merely a specular examination of the larynx. In practice, however, the examination requires a special technic that must be taught. It is not merely the putting in of the instrument and following a lumen. The displacement of tissues that obstruct the view of the larynx requires in some patients a certain technical skill that can be acquired only under an instructor.

**Anesthesia.**—In children, anesthesia, general or local, is quite unnecessary; and, in dyspneic patients, it is dangerous for this procedure. The entire interior of the larynx of any child, even the newborn infant, can be examined in a few minutes without any anesthetic, local or general. In adults general anesthesia is never required; even local anesthesia may be dispensed with. It is usually advisable, however, to use local anesthesia in adults, and a sedative may be added if there is no contraindication. The cocaine solution in about 8 per cent. strength is applied with a curved laryngeal applicator to the laryngopharynx and pyriform sinuses in the region of the superior laryngeal nerves. This is usually sufficient, but if the laryngeal reflexes still seem too active a little of the same or a stronger (20 per cent.) solution may be applied to the interior of the larynx with a gauze sponge in the straight applicator (R10, Fig. 556) after the larynx is exposed to view with the laryngoscope.

**Technic of Direct Laryngoscopy.**—After the application of local anesthetic or, in the case of children, without any such application, the patient is placed in the position shown in Figs. 557 and 558. The operator must be *standing* and must remain standing during the examination; crouching on the floor will totally defeat the object obtained by the proper position of the patient. The laryngoscope, held in the left hand in the

position shown at Fig. 559, is introduced back along the dorsum of the tongue, and the tip of the epiglottis is exposed to view. The lip of the laryngoscope is inserted beyond the proximal edge of the epiglottis for a distance of more than 1 cm. in a child, or 2 cm. in an adult. Then a powerful lifting motion, sufficient to sustain the weight of the patient's head, is imparted to the laryngoscope in the direction of the dart shown in Fig. 560. At this point particular care to keep the patient's shoulders down on the table is necessary. Prying on the upper teeth as a fulcrum must be avoided. The patient not being anesthetized, there will be no glottic chink until he takes a deep breath. If the patient is three years of

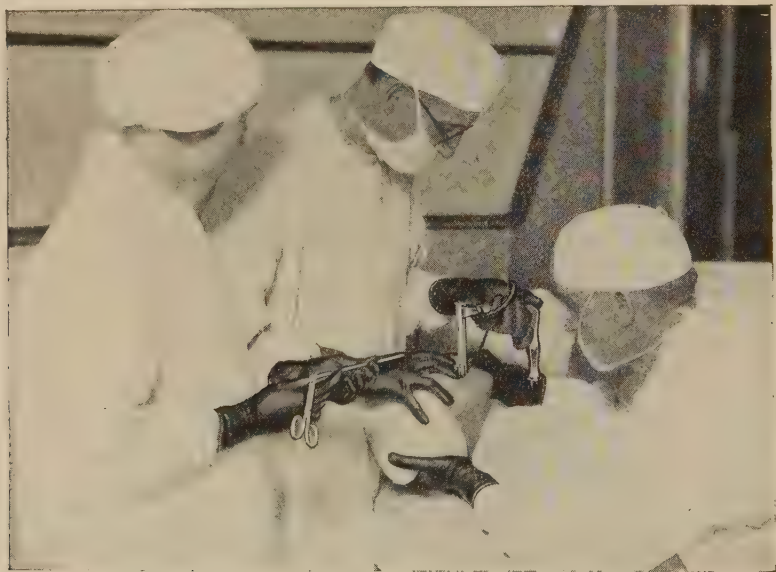


Fig. 559.—Direct laryngoscopy, recumbent patient. The second assistant and the assistant who holds down the shoulders have been asked to step aside in order to give a better view of the instrumentation. The fingers of the operator's right hand pull the upper lip out of all danger of getting pinched between the teeth and the laryngoscope. This is a precaution of the utmost importance and the trained habit of doing it must be developed by the peroral endoscopist. The instrument nurse is holding the laryngeal forceps in proper position for entering the tube. If bronchoscopy were to be done she would be thus holding the bronchoscope, with its handle out to the right.

age or over he is told to take a deep breath. If he is under that age he will soon do so without being told.<sup>1</sup>

It is absolutely essential that the laryngoscope be held in the left hand, and that the use of the instrument never be attempted even once with the right hand; to do so would give the operator a false start that would be a handicap. It is easier to expose the larynx with the left hand than with the right, even for right-handed persons. It requires no higher degree of ambidexterity to do bronchoscopy than to use the knife and fork in eating. As in any department of surgery, the bronchoscopist should develop the "lame duck" (left hand) to the greatest possible degree; and no matter how great the degree of ambidexterity developed he will, at times, wish he had a third or even a fourth hand.

**Direct Laryngoscopy for the Introduction of Ether Insufflation Tubes**

**or of Flagg's Ether Inhalation Tubes.**—The insufflation of ether by the method of Melzer and Auer, so well developed by Müller and Elsberg, has become recognized as a method of great value in operations on or near the head and for thoracic operations involving the opening of the pleura. The method of Paluel J. Flagg,<sup>3</sup> in which the patient breathes through a closely fitting rubber tube passed through the larynx, is very simple, safe, and satisfactory. It entirely eliminates the "death zone," and, furthermore, places the anesthetist out of the way of the operator and his assistants, who have the entire head of the table to themselves, unobstructed by an anesthetist or anesthetizing apparatus. In both these methods the direct laryngoscope is used to insert the tube through the larynx. Anyone familiar with the method of exposure of the larynx to view with the direct laryngoscope can insert the tube into the trachea in less than a minute. The technic is the

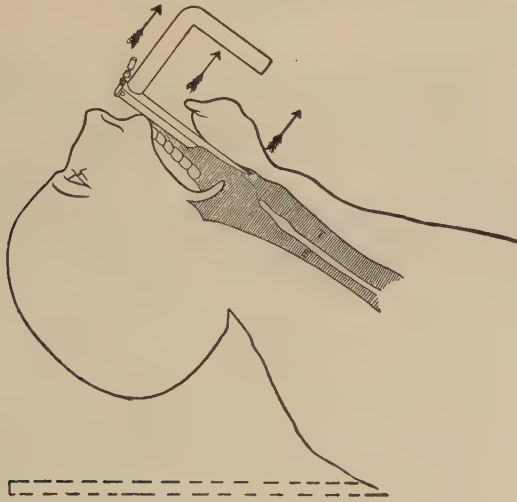


Fig. 560.—Schema illustrating the technic of direct laryngoscopy on the recumbent patient. The motion is imparted to the tip of the laryngoscope as if to lift the patient by his hyoid bone. The portion of the table indicated by the dotted line may be dropped or not, but the back of the head must never go lower than here shown for direct laryngoscopy; and it is better to have it at least 10 cm. above the level of the table. (Note that in bronchoscopy and esophagoscopy the head section of the table *must* be dropped so as to leave the head and neck of the patient out in the air, supported by the second assistant.)

same as that shown at A in Fig. 568. A strong lifting motion is imparted to the laryngoscope, sufficient to raise the patient's head off the table. Prying on the upper teeth as a fulcrum must be avoided. The procedure being for the purpose of etherization, the tube is not inserted until the patient is fully relaxed by the ether given by the open method. This relaxation is a great help to those inexperienced in the exposure of the larynx with the laryngoscope; but the experienced laryngoscopist could quickly insert the anesthetizing tube without any previous anesthetization.

**Direct Laryngoscopy for Pneumonography.**—If desired, lipiodol or bismuth may be introduced into either lung by direct laryngoscopy, without the bronchoscope. To enter the left bronchus the special "left bronchus" shape of instillation tube is necessary. The necessity for inspection of the bronchi and for the removal of obstructing granulations, granulomata,

tumors, or secretions renders the use of the bronchoscope advisable, however. (See chapter on Bronchoscopic Pneumonography.)

**Direct Laryngoscopy in Suspected Diphtheria.**—All children too young for mirror examination, and having a croupy cough without membrane in the fauces or visible pharynx should have a direct laryngoscopy for diagnosis as to the cause of the croupy cough. In many such cases the patient will be found not to have diphtheria, but a *foreign body*, or multiple *papillomata*, or *infective (influenzal) laryngotracheitis*, or perhaps *acute laryngitis*. If no foreign body or tumor is found, a swab-specimen for laboratory examination should be taken. This can easily be done without contamination with oral secretions, and will often show diphtheritic organisms when the specimens taken from the fauces in the ordinary way are negative.

**Webs of the Larynx.**—These may be congenital or cicatricial. If they cause obstruction enough to produce dyspnea they should be dealt with as shown in Fig. 561, using scissors (K7, Fig. 562). In some cases the air-

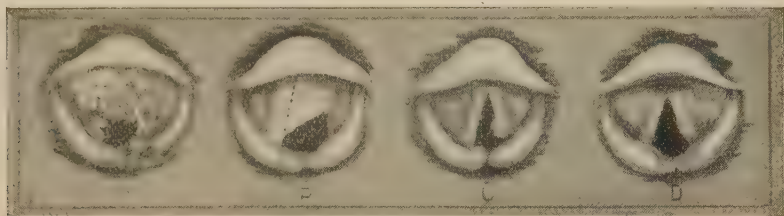


Fig. 561.—The method we have found most satisfactory for dealing with webs of the anterior commissure that bind the cords, or in absence of cords, the sides of the larynx together. Instead of excising the web (B), which would be followed by increased cicatricial contraction, the web is slit up along one side all the way to the anterior commissure as shown by the dotted line. The effect of this is to do away with tension and allow all the redundancy to go to one side. Later the redundancy may require removal, but usually it does not. Biweekly bouginage usually results in absorption of the redundancy and restoration of the normal triangular shaped glottis with the acutely angular anterior commissure. The cause of the web in this case was trauma inflicted by divers operators using indirect methods.<sup>2</sup>

way is sufficient, but the web prevents phonation. Liberation of the cords as shown will usually give very satisfactory results.

**Dilatation of Laryngeal and Tracheal Stenosis.**—In certain kinds of cases, as noted in another chapter, dilatation of laryngeal stenosis is the method of first choice. The metallic dilators are used, the triangular shape for laryngeal stenosis and the round form for tracheal lesions. No anesthesia or sedative is necessary, but there is no objection to using local anesthesia in adults if desired. It is very important that the dilators be smeared with vaseline before passing them.

**Direct Laryngoscopic Removal of Benign Growths from the Larynx.**—The precision with which benign growths may be scalped off, leaving the normal tissues intact, renders the method ideal for this purpose. The technic has been described elsewhere herein. The patient is always placed recumbent. No anesthetic, general or local, is used in children. In adults local anesthesia is used, and a full dose of morphine may be given two hours beforehand to quiet reflexes if it is desired.

The cupped forceps (K4 and 5, Fig. 562), straight or bent, are better for this purpose than the cutting forceps. The anterior commissure laryngoscope is best for this work. In case of very large growths the "extubal

method" (Fig. 563) may be used. The instruments are introduced into the mouth alongside the laryngoscope, their movements being guided by the eye looking through the lumen of the tube.

**Vocal nodules**, though not true tumors, sometimes require removal as elsewhere herein mentioned. This delicate operation can be done with all the nicety required. The vocal nodule forceps (K4, Fig. 562) and the anterior commissure laryngoscope are used.

**Removal of a Specimen of Tissue for Biopsy in Suspected Cancer of the Larynx.**—The ease with which a specimen of tissue can be removed

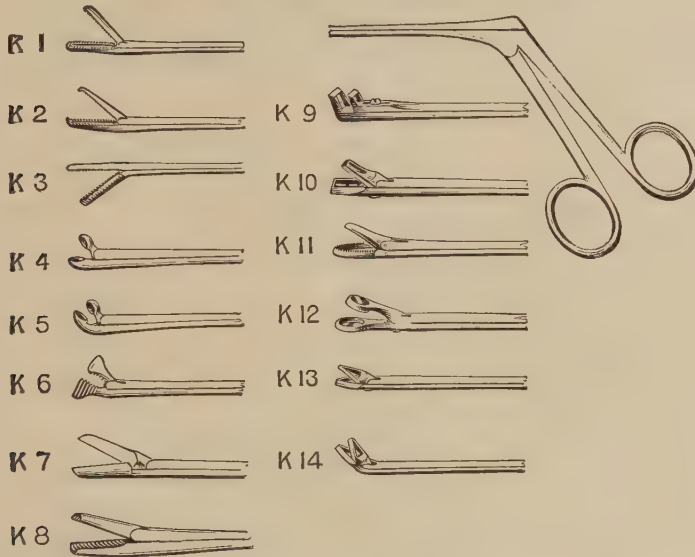


Fig. 562.—Alligator forceps. Except *K10*, these instruments are chiefly used through the laryngoscope and esophageal speculum. *K1*, Alligator grasping forceps for general purposes. *K2*, Alligator rotation forceps for version and for secure grasping of bones, dentures, peach stones, etc. *K3*, Down-jaw forceps. *K4*, Cupped forceps, made in three sizes of cups; the smallest is known as the vocal nodule forceps. *K5*, Bent-cupped; the same as *K4* except that the cups stand at an angle. *K6*, Flat forceps for scalping off papillomata without injury to underlying normal tissues; occasionally used for other purposes. *K7*, Scissors. *K8*, Cylindric forceps, made with and without spike teeth, for grasping cylindric objects, broken bougies, etc. *K9*, Clerf's sliding punch. *K10*, Tissue forceps, with shearing blades and a basket to hold the tissue punched off; the angle of the jaw is such that it will bite into a lateral wall. This is the best instrument for small specimens. Besides the laryngeal lengths it is used in 50 cm. and 60 cm. lengths through the bronchoscope, esophagoscope, and gastroscope. *K11*, Oval punch, used for operations such as excision of webs. *K12*, Basket punch forceps; the instrument of choice for excision of large specimens, or masses of tissue, as in ventriculocordectomy. The basket prevents loss of the removed tissue. *K13*, Triangular punch used chiefly for laryngeal operations. *K14*, Bent triangular punch (Imperatori).

from precisely the desired spot in the larynx by direct laryngoscopy has placed this absolute method of diagnosis in cases of suspected cancer on a plane entirely different from that which it occupied in the old days of mirror groping. The anterior commissure laryngoscope and the tissue forceps (*K10*, Fig. 562) are the best instruments. Local anesthesia is sufficient, and even this may be dispensed with. It is usually advisable to include an edge of normal tissue in order that the histopathologist may see the transition. Modern laboratory methods, dealing with the relatively small specimens taken from the larynx, render it possible to get an opinion

in eighteen hours or less. This removes the objection made to taking a specimen when nearly a week's delay was involved. If the specimen is taken immediately on the patient's entering the hospital, the report may be had by the time he is properly prepared for operation. Metastases do not travel far in eighteen hours.

**Ventriculocordectomy.**—This procedure<sup>4</sup> consists in punching out with the forceps (K 12, Fig. 562) the ventricular floor and all of the vocal cord lying anterior to the vocal process; and some of the latter may also be removed with advantage. This operation is done in cases of severe stenosis due to bilateral recurrent paralysis. It is indicated only in cases of pure paralysis. Therefore, the 9 mm. bronchoscope (in adults) should be passed prior to the operation. If stenosis other than the paralytic is found with

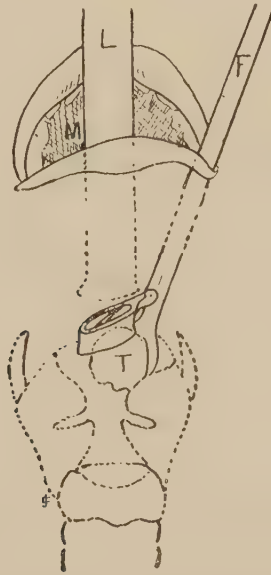


Fig. 563.—Schema illustrating removal of a tumor from the upper part of the larynx by the "extubal" method for *large* tumors. The large alligator basket punch forceps, *F*, is inserted from the right corner of the mouth, and the jaws are placed over the tumor, *T*, under guidance of the eye looking through the laryngoscope, *L*. This method is not used for small tumors. It is excellent for amputation of the epiglottis, with these same punch forceps or with the heavy snare.

this bronchoscope, ventriculocordectomy is not indicated. Subglottic stenosis, so often resulting from too high a tracheotomy, renders the patient an unsuitable one for ventriculocordectomy.

**Galvanopuncture for tuberculosis of the larynx** can be done with great accuracy through the anterior commissure laryngoscope. Great care is necessary. Indiscriminate or extensive cauterization is a mistake and may prove disastrous. For the indications and contraindications for the use of the galvanocautery see the article on Laryngeal Tuberculosis, by Dr. George B. Wood, who has used this method for many years.

**Multiple Papillomata of Children.**—These growths constitute a benign self-limited disease, and all radical removal of basal tissues is absolutely contraindicated. Destruction by radium or the Roentgen ray is usually attended by perichondritis, stenosis, and a disastrous destruction of the

larynx that would be justifiable for cancer, but for the self-limited papillomata of children it is a calamity. The growths repullulate on the surface and do not infiltrate the basal tissues. Experience in hundreds of these cases at the Bronchoscopic Clinic has taught us that superficial removal repeated as frequently as necessary ultimately results in a cure, with a good voice and perfect restoration of the airway.

**Eversion of the ventricle** may be advantageously examined with the anterior commissure laryngoscope. If any surgical procedure is deemed advisable it can be done quickly and with great precision by the direct method; but as to the advisability of surgical treatment the reader is urged to study carefully the article on this subject by Dr. Irwin Moore, who has clarified in his masterful way this heretofore much confused subject.

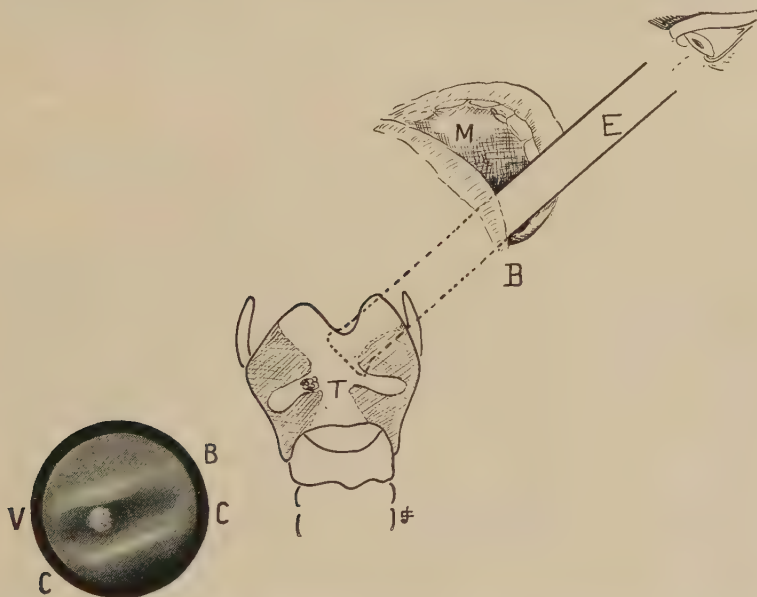


Fig. 564.—Schema illustrating the lateral method of exposing a growth in the ventricle of Morgagni by bending the patient's head to the opposite side, while the second assistant externally fixes the larynx with his hand: *M*, Patient's mouth; *T*, thyroid cartilage; *R*, right side; *L*, left. *V B*, Ventricular band. *C C*, Vocal cord. The circular drawing indicates the endoscopic view obtainable by this method. The tube, *E*, is dropped to the corner of the mouth, *B*, and the tube is inserted down to *R*. The lip of the spatula can then be used to lift the ventricular band so as to expose more of the ventricle. The drawing shows an unusually shallow ventricle.

**Laryngostasis.**—When it is desired to prop the glottic chink widely open, use is made of the laryngostat, a form of the Chevalier Jackson anterior commissure laryngoscope. The view given is shown in Fig. 565.

**Congenital Laryngeal Stridor.**—The typical form of this disease is shown at D, Fig. 566. The epiglottis is very soft and rolls over when manipulated out of the line of vision, instead of yielding to the lifting of the spatula. The aryepiglottic folds curve inward on inspiration and are obviously flabby. The stridorous sound, which is due to the vibration of the flabby upper laryngeal margin, may be prevented altogether by the propping apart of the folds. This is the typical form. Stridor present at times since birth may be due to malformation of the larynx, to papillomata, to in-

spiratory collapse into the trachea of the esophagotracheal party wall. The differential diagnosis of these conditions is made by the endoscopic examination. The prognosis is good. The treatment of papillomata is given elsewhere herein; the treatment of all the other conditions is general

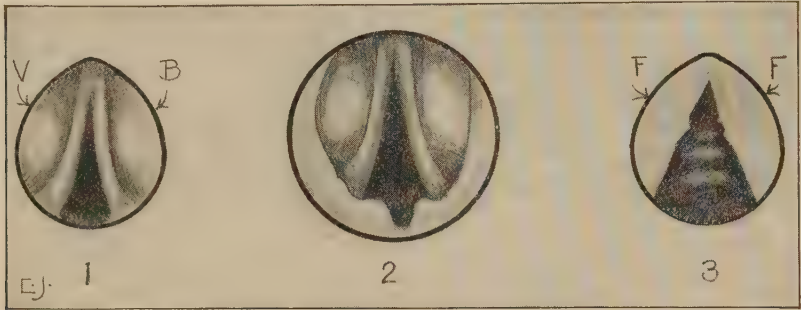


Fig. 565.—Pencil sketches, illustrating laryngostasis as contrasted with ordinary direct laryngoscopy. At 2 is shown schematically the broad, general view of the larynx afforded by the standard laryngoscope; the cords are rounded, the ventricular bands overhang the cords; moreover, in this procedure the cords move spasmodically and accurate work is impossible until the operator has learned the knack of laryngostasis. At 1 is shown schematically the view in the anterior commissure laryngoscope. The cords are rounded and the ventricular bands, *V*, *B*, overhang; there is, however, with this instrument a much less effort required to expose the anterior commissure and the knack of laryngostasis is easily acquired. At 3 is shown the exposure of the entire floor of the ventricle, *F*, *F*, obtainable with the laryngostat, with which the ventricular bands are pushed entirely to the side walls of the larynx out of sight. Moreover, the cords are held motionless in perfect laryngostasis, so that the utmost accuracy of forceps work is facilitated. A pair of vocal nodules are indicated in the sketch because of the necessity for the highest degree of precision in dealing surgically with these growths in singers. The laryngostat we find equally useful in the removal of papillomas and all benign growths, in which the utmost care to avoid injury to normal cordal tissue is imperative.<sup>4</sup>

rather than local. Rickets or other nutritional or metabolic disorders will require study and appropriate treatment.

**Laryngismus Stridulus.**—The direct laryngoscopic appearances in this disease are usually typical. The mucosa is pale or slightly grayish, and

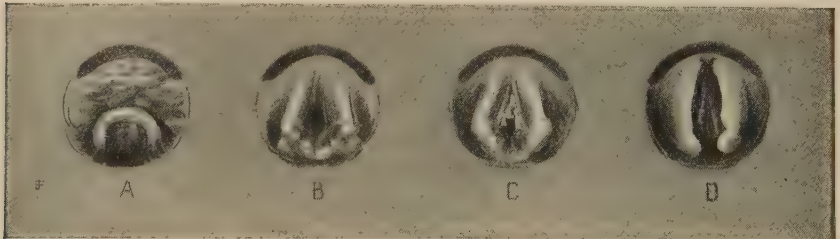


Fig. 566.—Direct laryngoscopic views in children. A, Epiglottis. B, Glottis on inspiration, prevented from a wide inspiratory excursion by normal spasm at the presence of the instrument in an examination without anesthesia. A few moments later it opened widely, and subglottic papillomata are visible as shown at C and D. Indrawing of the upper laryngeal aperture in a moderate case of congenital laryngeal stridor in an infant of eleven months.<sup>2</sup>

is distinctly non-inflammatory. The upper margin of the larynx is drawn in at each inspiration. The cords draw together at each inspiration and only slightly separate on expiration. The insertion of a bronchoscope gives immediate relief. If it is kept *in situ* for a few minutes the stridor and

dyspnea usually do not recur after its removal. The total absence not only of membrane but of inflammation is in marked contrast to the intensely inflammatory, deep red, and swollen laryngeal mucosa and the subglottic edematous masses protruding from below each cord as seen in diphtheria.

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3. Flagg, Paluel J.: *Intracheal Inhalation*, *Arch. Otolaryngology*, 5, 394, May, 1927.
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#### BRONCHOSCOPY

**Definition.**—Inspection of the interior of the trachea and bronchi with a tube that serves as speculum by displacing obstructive tissues or by dragging the tissues to be examined with a new position in the line of sight.

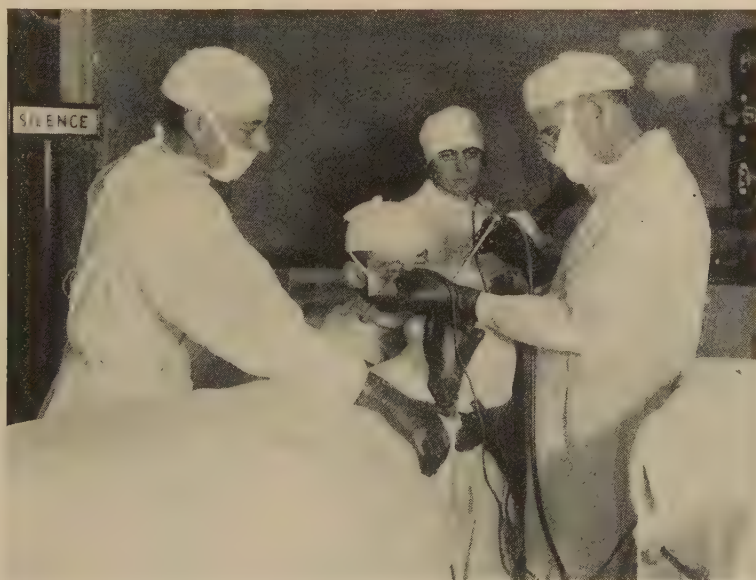


Fig. 567.—Insertion of the bronchoscope. Note direction of the trachea as indicated by the bronchoscope. The operator, having exposed the glottis with the laryngoscope in the left hand, has now transferred his eye to the bronchoscope, which he is insinuating into the glottic chink. Note the handle of the bronchoscope is to the right, and the instrument is held lightly, not grasped in the clenched fist.

For convenience the word "bronchoscopy" has come into general use as inclusive of the other peroral procedures—direct laryngoscopy, esophagoscopy, gastroscopy, and pyloroscopy. For purposes of this book, however, it is necessary to consider each of these procedures separately.

**Purposes.**—Bronchoscopy is used in the diagnosis and treatment of disease and for the diagnosis and removal of foreign bodies.

**Introduction of the Bronchoscope.**—The bronchoscope can be quickly and safely introduced without any difficulty after the larynx is properly exposed, with the laryngoscope held in the left hand.

**Anesthesia.**—General anesthesia is not necessary and is dangerous in dyspneic patients. Cocaine is dangerous and quite unnecessary in children; but is generally used in adults. Morphine in full doses may be given to both adults and children,<sup>2</sup> and should be administered hypodermically at least an hour and a half beforehand. One of the most important aids for working with local anesthesia, or no anesthesia at all, is quiet and tran-

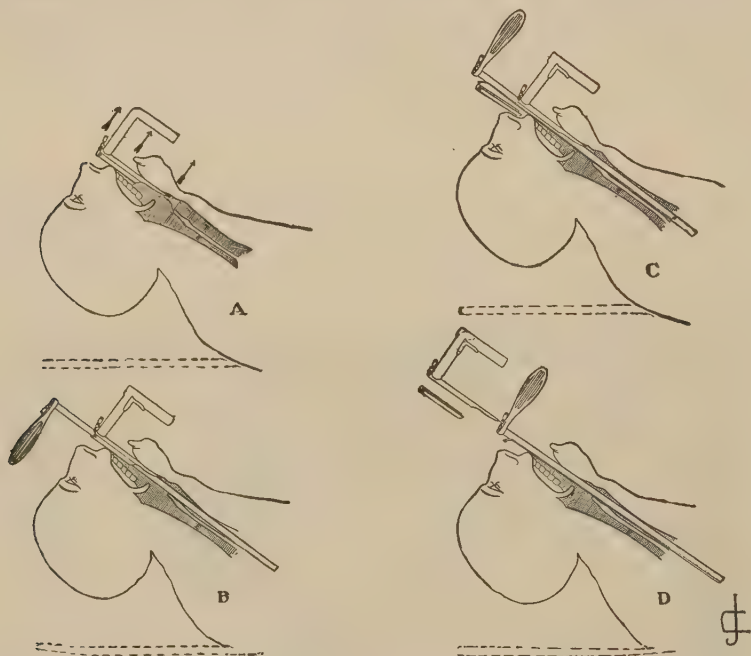


Fig. 568.—Schema illustrating peroral bronchoscopy. The portion of the table here shown under the head is, in actual work, dropped all the way down perpendicularly. It appears in these drawings as a dotted line to emphasize the fact that the head must be above the level of the table during the introduction of the bronchoscope into the trachea. A, Exposure of larynx. B, Bronchoscope introduced. C, Slide removed. D, Laryngoscope removed, leaving bronchoscope alone in position. (From *Bronchoscopy and Esophagoscopy*, by Chevalier Jackson, Text-book, 2d ed., 1927, W. B. Saunders Co.)

quility. To this end a well-trained team, a smooth technic, and the illuminated "Silence" sign (Fig. 567) contribute much.

**Position of the Patient.**—The patient should be in the position shown in Figs. 557 and 558, and the operator should stand up as shown in Fig. 567 until the distal end of the bronchoscope is in the trachea (D, Fig. 568). Later the operator may sit on a high or low stool, as necessary to present a lumen image.

**Insertion of the Bronchoscope.**—Once the vocal cords are exposed with the direct laryngoscope held in the left hand (A, Fig. 568), as described under the heading Direct Laryngoscopy, the insertion of the bronchoscope is easy. Before the bronchoscope is inserted a gauze sponge moistened

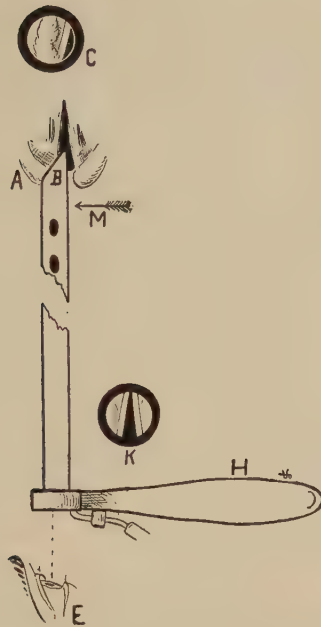


Fig. 569.—Schema illustrating the introduction of the bronchoscope through the glottis, recumbent patient. The handle, H, is always horizontally to the right. When the glottis is first seen through the tube it should be centrally located as at K. At the next inspiration the end, B, is moved horizontally to the left as shown by the dart, M, until the glottis shows at the right edge of the field, C. This means that the point of the lip, B, is at the median line, and it is then insinuated through into the trachea. At this same moment or the instant before the hyoid bone is given a quick additional lift with the tip of the laryngoscope.<sup>1, 2</sup>

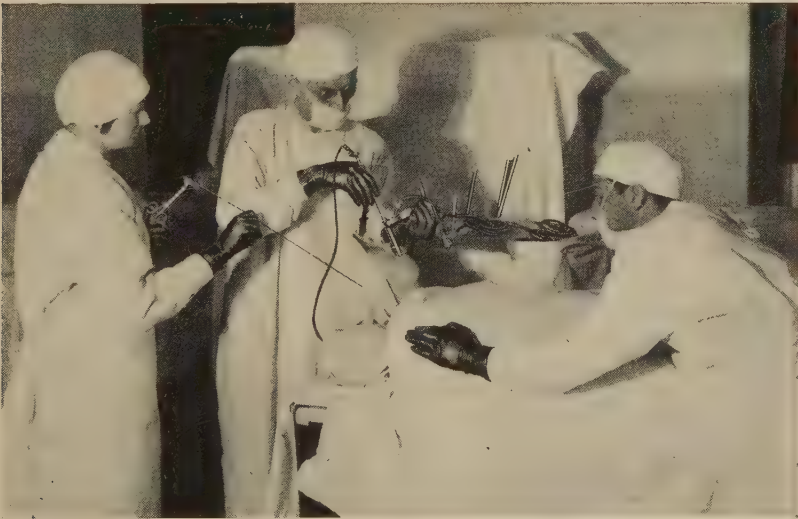


Fig. 570.—The operator has insinuated the bronchoscope through the larynx; the distal end is well down in the trachea. He is now ready to remove the heavy laryngoscope, leaving the light delicate bronchoscope alone in position. (Please note that the assistant who holds the head has stepped aside to avoid obscuring our view of the instrumentation.)

with a 10 per cent. solution of cocaine (in adults only) may be passed between the cords, down to the bifurcation, and held there for a minute or two, or the cocaine solution may be sprayed in limited quantity. The bronchoscope, illuminated with its own lamp, should be passed to the operator in exactly the proper position for insertion (point forward, "handle" to the right). The operator, after inserting the bronchoscope into the laryngoscope, transfers his eye to the bronchoscope and, making sure of the presentation of the vocal cords, insinuates the slanted end of the bronchoscope carefully between them with a slightly rotary motion (Fig. 569). The exploration of the tracheobronchial tree is a matter of following the lumen, and is greatly facilitated by the position above described, which leaves the head of the patient free to be moved about widely in every direction. Though the operator must be standing at the start (Fig. 570), following the lumen, after introduction usually requires the operator to sit. To expose anterior branches, such as the middle lobe bronchus or the anterior branches of the left upper lobe bronchus, requires lowering of the patient's head, and a low position of the operator.

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3. Jackson, Chevalier: *Teaching Bronchoscopy and Esophagoscopy*, Arch. Otol., January, 1928.

#### INTRODUCTION OF THE ESOPHAGOSCOPE

The first thing to realize is that if an esophagoscope is simply put into the pharynx and pushed upon, it will not go into the esophagus, but into the tissues of the mediastinum. Esophagoscopy is so totally unlike the introduction of a soft rubber stomach-tube that the practitioner uninformed of the difference will almost certainly cause perforation with the esophagoscope. It is essential that close attention be given to the following details. The introduction of the esophagoscope calls for the exact position of the patient described (Fig. 571). With the esophagoscope vertical, the standing operator finds the right pyriform sinus by sight, no mandrin being used. Passing downward, the operator finds the first obstacle at the bottom of the hypopharynx, in the rigid contraction of the cricopharyngeus muscle. It is necessary to wait for this to relax, but while waiting continuous *gentle* advancing pressure must be maintained and at the same time the esophagoscope must be pressed anteriorward by the left thumb, to lift it away from the posterior weak point, where it otherwise is almost certain to perforate. This must be done without lowering the head of the patient. The "handle" of the esophagoscope is not grasped in the hand. It must be *up*, by which we know that the lip of the tube-mouth is anterior and away from the danger point. The general direction of the entire esophagoscope is maintained by aiming for the median line as indicated by the midline of the sternum, notwithstanding the fact that we are starting

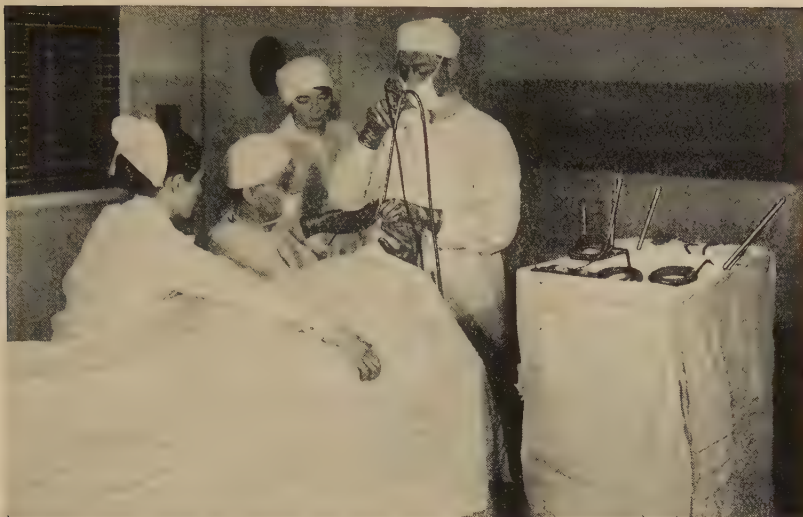


Fig. 571.—Esophagoscopy by Chevalier Jackson's "high-low" method. First stage. "High" position. Finding the right pyriform sinus. In this and the second stage the patient's vertex is about 15 cm. above the level of the table and the operator must be standing. After the esophagoscopic tube-mouth reaches the mediastinal esophagus the head is lowered as required by the operator to follow the lumen as seen through the tube. When the diaphragmatic level is reached the patient's vertex is level with the table-top.



Fig. 572.—Esophagoscopy by our "high-low" method. Stage 4. Passing the hiatus. The patient's vertex is about 5 cm. below the top of the table. The esophagoscope is pointing toward the anterior superior spine of the patient's left ilium. The patient's knees may be flexed if desired to assist relaxation. The nurse is holding the forceps in line for insertion. It cannot be too strongly emphasized that if introduction of the esophagoscope were attempted, starting with the position here shown, introduction would be impossible and fatal trauma probable. Incidentally this illustration shows the positions of the battery table (B) at the operator's left and of the instrument table (A) at his right, back of the instrument nurse. Precisely this relative position of the tables is invariably used; otherwise confusion, delay, and entanglement in handling of cords, rubber tubes, and long instruments would result.

from one side, in the right pyriform sinus. Care must be taken not to point clear across toward the left side. The opening of the lumen is watched for in the *anterior* part of the field. The relaxation of the cricopharyngeal pinch-cock is usually accompanied by a regurgitation of saliva, and the slanted end of the tube-mouth glides over anterior to the relaxing cricopharyngeus muscle. The rush of saliva is quickly and automatically removed by the aspirating canal if the handle of the esophagoscope is up as it should be. Exploration of the thoracic esophagus is simply a matter of following the lumen as it opens up ahead, a procedure easy of accomplishment when the head is held in the air, free to be moved in every direction, as indicated above. The operator follows the lumen, the assistant follows the operator with the patient's head. At the hiatus œsophageus,

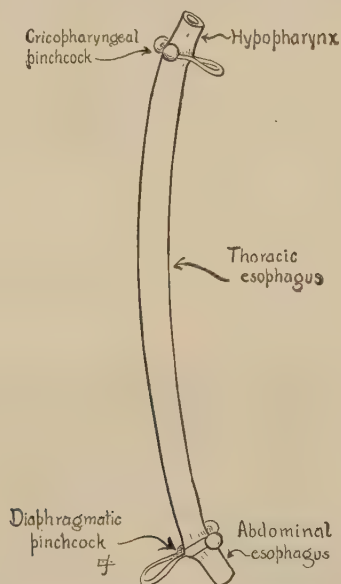


Fig. 572a.—Manikin demonstrating the two greatest difficulties in esophagoscopy. The esophagus is represented by a piece of rubber tubing pinched shut by burette pinch-cocks representing the closure of the esophagus at the respective locations by muscles *outside the esophageal wall*. The closure is constant, normal, tonic contraction, not spasm. In pre-ventriculosis (so-called cardiospasm) there is failure of co-ordinate opening of the diaphragmatic pinch-cock in swallowing, but no increased resistance to the esophagoscope unless organic disease is present.

the esophagus goes through the diaphragmatic pinch-cock. Just as the rubber tube of a buret is pinched tightly shut by the spring clip known as a pinch-cock, just so we have the esophagus pinched together at the hiatus by the crura and the muscular fibers of the diaphragm. The point in the lumen of the esophagus corresponding to this pinch-cock closure is found by lowering the patient's head to the right and aiming the esophagoscope (or gastroscope) for the anterior superior spine of the left ilium (Fig. 572). Gentle but continuous pressure on the proper place will be rewarded after a few moments of patient waiting by the relaxation of the pinch-cock. The opening of the hiatal constriction is usually accompanied by a rush of gastric fluid, which will be clear if the stomach is "empty" and normal; otherwise, it may be mixed with pus, blood, or food. Once the hiatus is passed, the esophagoscope slips so quickly and easily through

the abdominal esophagus that the existence of an abdominal esophagus is not realized. There is no constriction of any kind, functional or structural, noticed at the cardia, only a faint difference in color and a marked difference in the form of the folds.

**Difficulties in the Introduction of the Esophagoscope.**—The first real difficulty in passing an esophagoscope arises at the cricopharyngeal pinch-cock, which often remains pinched tightly shut for minutes that seem almost to be hours. The esophagoscopist seeking admission to the esophageal hallway is rudely and arbitrarily kept waiting in the hypopharyngeal vestibule a long time for the cricopharyngeal door to open. The foregoing refers only to the delay. We all have to wait a varying length of time for the cricopharyngeal pinch-cock to open. Independent of this matter of delay there is difficulty in finding exactly the right place. If difficulty

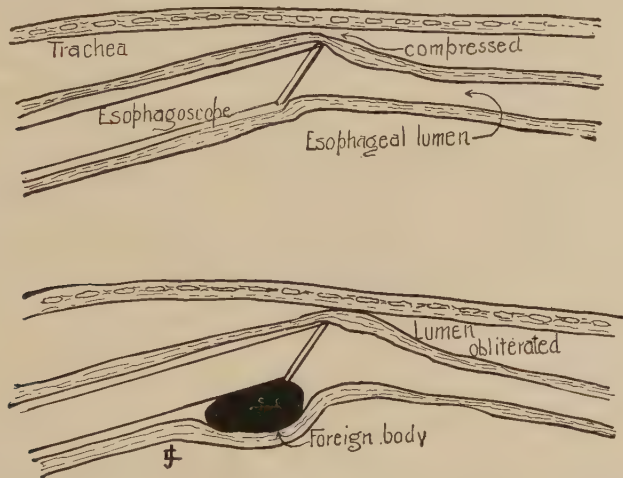


Fig. 573.—Serious obstruction of the trachea from crowding of the party-wall into the trachea during esophagoscopy, especially in babies and very young children. Asphyxia will soon result if this condition is not promptly recognized. A proper position of the patient and careful work will serve to avoid all danger of this complication. Even without a foreign body the bulk of an esophagoscope may cause dyspnea in babies unless a very small tube is used. (From *Bronchoscopy and Esophagoscopy*, by Chevalier Jackson, 2d ed., W. B. Saunders Co., 1927.)

is experienced at the cricopharyngeal or hiatal pinch-cocks, the filiform (*M*, Fig. 556) may be used carefully through the tube by sight to make sure of the lumen.

**Dyspnea** may be so great as to require cessation of the introduction of the esophagoscope. This may be from laryngeal edema secondary to the foreign body, or to previous instrumentation; but most frequently it is due to causes illustrated in Fig. 573.

**Esophagoscopy in Cases of Retention.**—The drainage canal in the esophagoscope automatically removes all fluid secretions when the tube is held in the handle-up position. If solid foods, floating in the fluid, clog the inlet at the distal end, it is in most cases quickly cleared by substituting the positive pressure tube. In preventriculosis (so-called cardiospasm) huge collections of food are quickly removed with the esophageal evacuator of Chevalier Jackson (V10, Fig. 556). The bottle conveniently collects the food for examination.

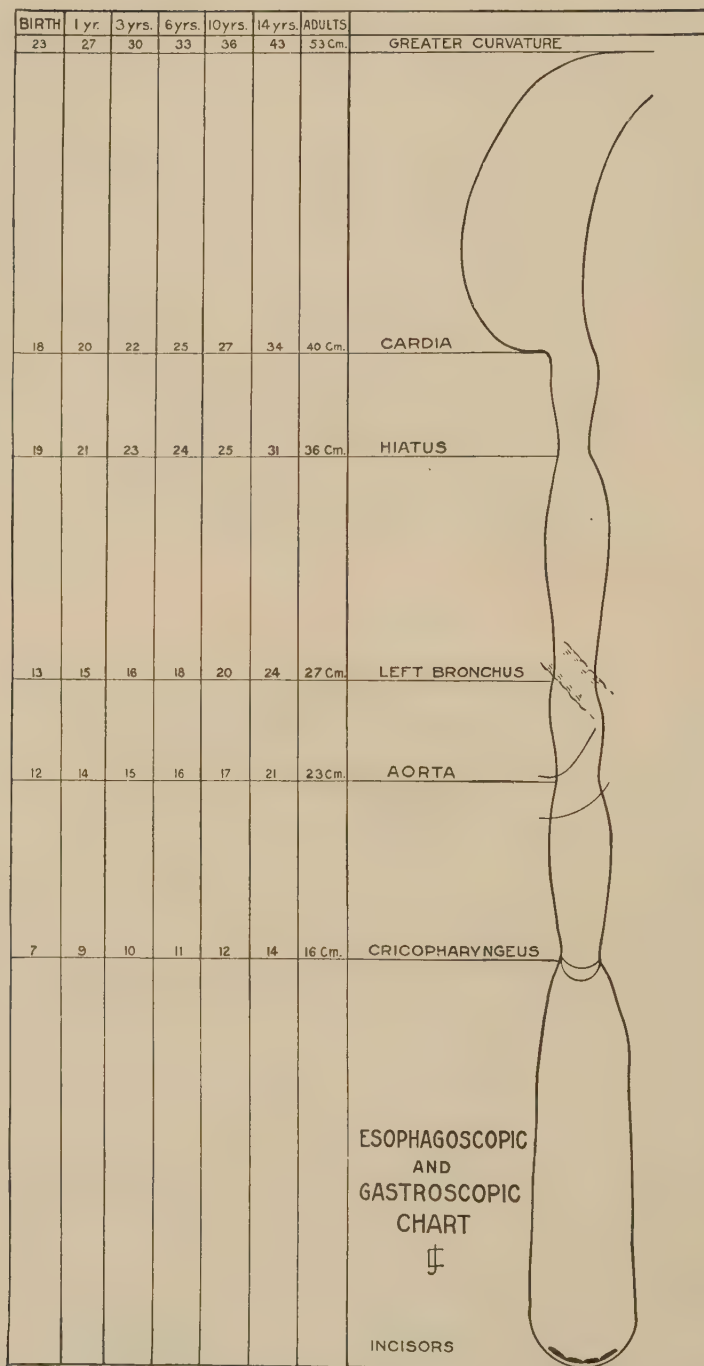


Fig. 574.—A chart of approximate distances of the esophageal narrowings from the upper incisor teeth, at various ages, arranged for convenient reference during esophagoscopy in the dorsally recumbent patient. Copies of this chart suitable for framing may be obtained gratis from the Chevalier Jackson Bronchoscopic Clinic, Graduate Hospital, University of Pennsylvania.

**Technic of Esophagoscopic Removal of a Specimen of Tissue.**—The only certain way of making a diagnosis of cancer of the esophagus early enough to be of any avail is by esophagoscopy and removal of a specimen. All other methods are inferential, are late at best, and often erroneous, even fatal, at worst. To warrant a transthoracic esophagotomy on a man in the good general condition necessary to survive the major operation requires an absolutely positive diagnosis, and this only the histologist can give. In endo-esophageal cancer a specimen can be safely taken through the esophagoscope under guidance of the eye with the long form of the forceps (K10, Fig. 562). We have never seen any ill results from taking a specimen of tissue. Obviously it is unwise to penetrate normal esophageal wall to search for a specimen of peri-esophageal cancer. In cases of suspected cancer high in the esophagus it is well to inspect the hypopharynx and upper end of the esophagus with the laryngoscope (A, Fig. 556).

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#### GASTROSCOPY

**Peroral Gastroscopy.**—The peroral gastroscope, a straight and rigid instrument (C1, Fig. 556), can be passed into the stomach of any patient who can open his mouth, and this can be done without any anesthetic, general or local. It is an invaluable procedure for diagnosis and treatment of disease and for removal of foreign bodies. The insertion of the tube through the mouth into the stomach is the same procedure as esophagoscopy (*q. v.*). Exploration of the explorable area of the stomach is carried out with the organ in the collapsed state, with the open-tube esophagoscope, by traversing it upward and downward, moving the tube laterally to a new location at the end of each traverse, as with the mechanical stage of the microscope. If desired, a lens system may be inserted in the open tube, and the stomach inflated for inspection. The greatest obstacle to reaching the pylorus, especially in adults, is the forward bulging of the spinal column. In children it is not very difficult to reach the pylorus; we have a number of times removed perorally foreign bodies jammed in it. In either adults or children the pylorus can be manipulated by an assistant in such a way as to bring it over far enough to the left for the gastroscopist to inspect the pyloric antrum and the pylorus itself.

**Peroral Gastroscopy for Disease.**—The usefulness of direct inspection of the stomach is obvious. The exploration of the left two-thirds of the stomach is a common procedure at the Bronchoscopic Clinics. The technic is the same as that of esophagoscopy (*q. v.*) up to the point of reaching

the stomach. After that the gastric folds are explored by vertical traverse in successive tracks, starting at the extreme left; each successive track is a centimeter toward the right of its predecessor until the entire field is covered. In some cases a window-plug is placed in the proximal end of the gastroscope to retain air pumped in through the drainage canal for inflation. A lens-system for angular vision of the inflated stomach may be used if desired, in which case it is introduced into the open tube after inflation. With an open tube carefully watched during introduction is the only safe method of traversing the esophagus. The gastroscope has opened up an enormous field for endoscopic study; but the diseases of the stomach are not within the scope of this book. The reader interested in the subject is referred to the appended bibliography. Peroral gastroscopy for foreign body is elsewhere herein considered.

**Peroral pyloroscapy** with an ordinary open-tube gastroscope is a practical procedure in all infants and very young children. The displacement required to bring the pylorus over to the tube mouth is easily accomplished by an external abdominal manipulator in adults also if there are no adhesions. If there is a palpable mass, it can be brought from an unreachable area and placed in front of the tube mouth for inspection. A foreign body can be brought in front of the tube mouth by the fluoroscopist. The reader interested in this subject is referred to the appended bibliography. Peroral pyloroscapy is elsewhere herein considered.

**Retrograde esophagoscopy, gastroscopy, pyloroscapy, and duodenoscopy** are useful procedures that can be carried out in patients on whom gastrostomy has been necessitated by atresia of the esophagus. In such cases a cure of the esophageal occlusion, unless malignant, is greatly facilitated by the retrograde esophagoscopy. The first portion of the duodenum is accessible to the gastrostomic gastroscope.

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## PART VI—DISEASES OF THE HYPOPHARYNX, ESOPHAGUS, AND TRACHEO- BRONCHIAL TREE

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### DISEASES OF THE HYPOPHARYNX

THE hypopharynx as a site of disease has not been accorded its full degree of importance in the literature. Inasmuch as the anterior wall of the hypopharynx is grossly a "party-wall" in common with the posterior wall of the larynx, it is intimately associated with all posterior mural extensions of laryngeal disease as elsewhere herein considered; but it is also subject to a number of diseases entirely independent of the larynx. These will be considered here, because of the clinical fact that every peroral esophagoscopy necessarily includes examination of the hypopharynx as the first stage.

The hypopharynx is subject primarily or secondarily to various ailments, which may be classified as listed below (the diseases are listed categorically for ready reference in diagnosis; no attempt is made to eliminate overlapping): Dilatation, diverticulum, congenital stenosis, "spasmodic" stenosis, acute inflammatory stenosis, cicatricial stenosis, compression stenosis, abscess, acute hypopharyngitis, chronic hypopharyngitis, ulceration, erosion, trauma, malignant neoplasm, benign neoplasm, tuberculosis, syphilis, paralysis, hysteria, angioneurotic edema, actinomycosis, varix, anomaly, foreign body.

Most of these conditions will, because of the limitation of space imposed upon a text-book, be considered in connection with similar conditions in the esophagus.

The **symptoms of hypopharyngeal disease** may be in the initial stages only a slight, vague, ill-localized sensation, spontaneous or on swallowing. At this stage all hypopharyngeal diseases may be mistaken for hysteria because of the classic "globus hystericus," or sensation of "a lump rising in the throat." The latter phrase is usually forced on the patient by the physician, because the patient is unable to describe the vague sensations he feels. The symptom when occurring in hysterical patients is a spasmodic contraction of one or both the inferior constrictor muscles. Much more often, however, hypopharyngoscopy will reveal a lesion, and it may be a lesion of serious character. In hundreds of cases of cancer of the hypopharynx that have come to the Bronchoscopic Clinic, the diagnosis of a neurosis or hysteria had been made; and in many other cases, where we expected negative findings we have ourselves been surprised to find a serious lesion. In the later stages the symptoms are dysphagia and odynphagia. The patient may be unable to swallow or swallowing may be so painful that he refuses to swallow. Spontaneous pain may be slight or very severe and may be local or referred to the ear. There may be tenderness to external palpation, but more often the tenderness is really located in the lymph-nodes secondarily involved.

**Diagnosis of Disease of the Hypopharynx.**—Inferential diagnosis is nearly always erroneous. Objective methods alone are of value. They are three:

(1) Palpation of the neck deeply under the sternomastoid muscles near the manubrium often yields important information and should never be neglected; but the most dependable methods are (2) Roentgen-ray examination, first without, then with an opaque mixture, and (3) hypopharyngoscopy. The former should always precede the latter and should include the chest and esophagus. Hypopharyngoscopy gives all the certainty of direct vision and affords opportunity for biopsy, which is the final arbiter in all infiltrative, fungative, and ulcerative lesions.

**Hypopharyngeal inflammation, ulceration, erosion, and stricture** are usually secondary to trauma, instrumentation, swallowing of lye, acids, or other caustics, or to foreign body. Milder degrees may be secondary to stasis. The diagnosis, prognosis, and treatment are the same as in similar conditions in the esophagus. *Traumatic perforation* as from a blindly passed bougie is usually followed by subcutaneous emphysema, cervical cellulitis, mediastinal emphysema, or mediastinal abscess. Any or all of these may coexist. The prognosis is serious, and in most instances death follows within a few hours or a few days.

**Abscess of the hypopharynx** is usually part of a retropharyngeal abscess that has started higher up. The chief causes are infection from the tonsil, disease of the vertebra, trauma from blind passage of a bougie, suppurating lymph-nodes, and foreign body. The diagnosis is made by hypopharyngoscopy. In children, the finger of the examiner may reach the lesion; if so the diagnosis can be made by palpation. Roentgenograms should always be made in both the anteroposterior and lateral planes, to show the abscess and to determine the condition of the cervical spine. It is usually the lateral view that will show the abscess best. The abscess should be evacuated with the patient in the so-called Jackson position, with the table lowered at the foot and elevated at the head, the patient's head being lowered over the end of the table so that the pus will not be inspired into the lung. The best instruments are the laryngoscope (A1, Fig. 556) and the scissors (K7, Fig. 562) or the laryngeal forceps (K13, Fig. 562). The aspirating tube should be held at the patient's mouth in readiness for instantaneous insertion as soon as an opening is made for it. Reaccumulation may require re-evacuation, but unless there is vertebral disease prompt recovery is usual.

**Foreign body in the hypopharynx** will be considered in connection with esophageal foreign body. In most cases the foreign body passes below the cricopharyngeal fold, which is the lower border of the hypopharynx.

**Paralysis of the hypopharynx** is really paralysis of the inferior constrictors. The patient is unable to swallow and will die of starvation if not fed with a stomach-tube, because gravity alone will not usually carry food or liquids into the stomach. The paralysis may follow diphtheria; in which case prognosis is good. When associated with bulbar lesions, such as myasthenia gravis, as is usually the case, the prognosis is that of the general condition, usually bad. When occurring as a part of a "*syndrome of Avellis*," the prognosis is fairly good, though as with any syndrome the prognosis will vary with the gravity of the pathological conditions present in the particular case.

**Diverticulum of the hypopharynx** is so closely allied to esophageal disease in its diagnosis and treatment that it will be considered under the head of esophageal disease. Anatomically, however, *pulsion diverticulum* is a hernia of the wall of the hypopharynx. Its only real connection with the esophagus is secondary, usually a compression of the esophagus by the sac when filled.

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### DISEASES OF THE ESOPHAGUS

It is not within the scope of this book to consider exhaustively the subject of the esophagus and its diseases. It is essential, however, that the fundamentals be presented in useful form. For additional information the reader is referred to the text-book, *Bronchoscopy and Esophagoscopy*, published by W. B. Saunders Company. The various pathological states of the esophagus encountered in the *Bronchoscopic Clinic* are listed below. For convenience in reference no attempt has been made to avoid overlapping. It must be remembered in clinical work that two or more of these conditions may be found to coexist: Anomaly; acute esophagitis; chronic esophagitis; erosion; ulceration; trauma; congenital stricture; spasmodic stricture; inflammatory stricture; cicatricial stricture; local dilatation; diffuse dilatation; diverticulum; compression stenosis; mediastinal tumor; mediastinal abscess; mediastinal glandular mass; aneurysm; malignant neoplasm; benign neoplasm; tuberculosis; lues; actinomycosis; varix; angioneurotic edema; hysteria; paralysis; foreign body: (a) pharynx, (b) larynx, (c) trachea, (d) esophagus; antiperistalsis. The most important of these diseases will be considered separately in the following pages. The others will be found to be considered *in extenso* in the works listed in the references.

**Diagnosis of Diseases of the Esophagus.**—There are only two methods of esophageal diagnosis that are worthy of a moment's consideration, and neither should be omitted in the case of any patient complaining of the slightest abnormal sensation in swallowing. These two methods are Roentgen-ray examination and esophagoscopy. The bougie as a diagnostic instrument is inferential and inconclusive at best, and dangerous in any case. A history should always be taken, but no inferences drawn therefrom should justify dispensing with or postponing application of the two essential methods of objective examination—the Roentgen ray and the esophagoscope. After a history is taken, careful examination of the mouth, nasopharynx, pharynx, and larynx is made by the laryngologist. He may find local painful lesions such as tuberculous ulceration of the epiglottis, or

cancer anywhere in the area explorable by the laryngoscopical mirror. The diagnosis may then be completed by the Bordet-Wassermann test, the physical and Roentgen-ray examination of the chest, sputum examination, biopsy, etc. The laryngologist may find impaired laryngeal motility, which is, except in case of aneurysm, a strong indication for diagnostic esophagoscopy. He may see the pyriform sinuses full of bubbling secretions. This is known as Chevalier Jackson's sign (lack of esophageal drainage) and calls for esophagoscopy unless there is obvious painful ulceration of the larynx. The patient is now ready for esophagoscopy, the final step in esophageal diagnosis. While final in point of sequence it is first in importance and should never be omitted.

In esophageal diagnosis it is essential to remember the following points:

1. Regurgitation is usually called vomiting by the patient, and especially by the parents when the patient is a child.
2. Never make a diagnosis of "globus hystericus" until everything else is excluded.
3. "Globus hystericus" is more often cancerous than hysterical.
4. Always determine the presence or absence of aneurysm before esophagoscopy. In some cases it is a contraindication.

The various steps in diagnosis are listed categorically here for convenience.

*Diagnostic Steps in Cases of Suspected Diseases of the Esophagus:*

1. Anamnesis.
2. General physical examination.
3. General examination of nose and throat.
4. Mirror examination of larynx and pyriform sinuses.
5. Systemic tests.
6. Fluoroscopy.
7. Fluoroscopy with opaque mixture and capsule.
8. Roentgenogram with opaque mixture.
9. Esophagoscopy.
10. Biopsy in certain cases.

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#### ANATOMY AND ANOMALIES OF THE ESOPHAGUS

**Comparative Anatomy.**—In examining vertebrates generally it is found that the esophagus may be very short and wide and indistinguishable from the stomach with which it is directly continuous without any marked constriction as in fishes. In serpents the esophagus is very distensible. It may have large conical papillæ which are directed backward (Chelonians).

In birds at its lowest part there is a special sac-like dilatation called the crop. Nothing like a crop exists in man's class except in the dormouse. In this animal the lower part of the esophagus is symmetrically enlarged. In birds the first or the cardiac part of the stomach (preventriculus) resembles more a dilatation of the lower end of the esophagus than the stomach proper.

The esophagus may be more muscular than it is in man; or it may be rather valvular as in the dugong, and still more so in the porpoise.

**Congenital Anomalies.**—The esophagus may be divided or double. It may open into the trachea, the lower part being absent. *Fistulæ* between the trachea and the esophagus occur, and annular strictures have been found. Diverticula of congenital origin have never been reported.

**The Esophagus at Birth.**—A study of the casts of the esophagus and the stomach in 65 babies dead at birth showed that it was not uncommon in babies of the same size for the esophagus of one to be twice the width of the esophagus of another. In 3 of these 65 babies a stricture was present at the cardiac end of the esophagus, and in 2 of these 3 there was also a narrowing of the pyloric opening of the stomach. At birth it was found that the left crus as it passes behind the esophagus often constricts it and, at the same time, makes a notch in its left border. It is common also to have the edge of the left lobe of the liver make a crease in the front face of the esophagus, and above this the esophagus has a spindle-like dilatation. In other words, these findings at birth are the same as those found in the adult. An S-shaped twist was found in one instance, the main bend being to the right. In a fetus of about three months an enlarged spleen pressed on the lower end of the esophagus and markedly narrowed it. So far I have never found a diverticulum of the esophagus at birth.

**Anatomy.**—The esophagus is a nearly straight membranous muscular tube. It is the continuation downward of the inferior constrictor muscle of the pharynx, and starts from the back of the cricoid cartilage opposite the sixth cervical vertebra. At the mouth of the esophagus the lower border of the inferior constrictor projects like a mound into its lumen, and acts as a sphincter.

The sphincter is especially strong in children. Foreign bodies often hide below the posterior half of the mound made by the sphincter. In removing a foreign body, such as a coin, from the mouth of the esophagus it must be held firmly or the sphincter will pull it from the grasp of the forceps.

**The Vertebral Bed.**—According to the formation of the bodies of the vertebra the esophagus has a secure or an insecure bed. If the vertebral bodies are flat and broad the esophagus rests securely. If, however, as often happens, the bodies of the thoracic vertebræ are narrow and pointed the esophagus is readily unseated. It always twists or falls to the right. I have found but four instances in which the esophagus lay on the left side of the vertebral column. The descending aorta usually monopolizes the left side of the spine. The backward pressure of the heart steadies the esophagus from the front. Once the esophagus has left its bed the pressure of the heart prevents it from getting back.

**Structure.**—The esophagus has an outer muscular coat of two layers and an inner glandular coat covered with pavement epithelium. A connective-tissue layer joins the two. The outer layer of the muscular part

consists of longitudinal fibers, and the inner layer of circular ones. The thickness of the esophagus is 3 to 4 mm. The longitudinal fibers are attached to the back of the cricoid cartilage. The inner layer of the circular muscular fibers is a continuation downward of the fibers of the inferior constrictor muscle. The upper end of the esophagus is the lower end of the pharynx, so that the voluntary muscular fibers predominate. From this it happens that a foreign body arrested at the entrance of the esophagus may be thrown back into the pharynx and into the mouth.

**Relations.**—The esophagus has the vertebral column behind it and the trachea in front and lying on it. It runs in the posterior mediastinum. At the fourth thoracic vertebra the arch of the aorta makes a transverse constriction in it, and a vertebra lower down at the fifth thoracic, the left main bronchus, makes an oblique line across its front face. Below this point the heart lies on it like a weight. In the lower part the right and left pneumogastric nerves are found on the sides of the esophagus, and back of the arch of the aorta the thoracic duct crosses from right to left behind it, on the front of the vertebral column.

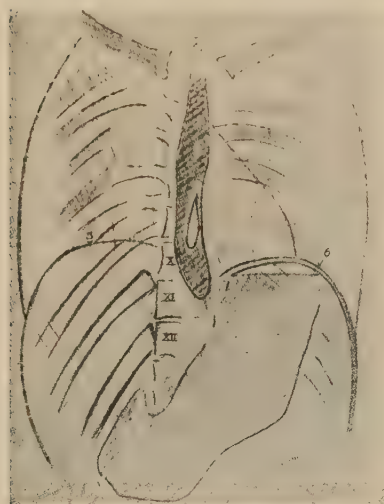


Fig. 575.—Tracing from a Roentgen-ray film of a normal adult male to show the relation of the esophagus to the spine.

**Direction.**—The esophagus is placed for the most part a little to the left of the middle line of the body. Half-way down in its course, at the fourth thoracic vertebra, it swings to the middle line, back of the arch of the aorta, but at once goes to the left again and enters the stomach to the left of the spine and in front of the descending aorta, at a point placed somewhere between the middle of the tenth and the middle of the eleventh thoracic vertebræ. This deviation from the middle line does not interfere with the passing of bougies or tubes except at the lower part where the esophagus

passes through the diaphragm, and here only slightly.

Casts of the adult esophagus show that its position often varies from the text-book description just given. If the vertebral bed is broad the esophagus does lie for the most part to the left of the middle line of the spine. If, however, the bodies of the vertebræ are narrow the esophagus covers the whole front face of the spine or may overhang its right edge and lie in part on its right side.

**The Postesophageal Space.**—The esophagus runs in the posterior mediastinum. Behind the esophagus there is a space—the postesophageal space—which appears as a light streak in the fluoroscope. This space amounts to but little above the arch of the aorta. Below this the space may be  $\frac{1}{4}$  to  $\frac{1}{2}$  inch wide. It gradually increases in width downward until at the diaphragm it may measure an inch. Tumors of the esophagus grow into the space and darken it. In perforation of the esophagus by instrumentation the space infects very readily.

When the thoracic and cervical spine bows forward, as in people with round shoulders, the postesophageal space becomes much wider, measuring at the bottom as much as 2 or 3 inches. I have two casts of humpbacks in which the aorta follows the curves of the spine, but the esophagus runs straight down in a comparatively normal course. In such cases the postesophageal space reaches its greatest width.

**The Aortic Crescent.**—The arch of the aorta projects backward into the postesophageal space. Barium in passing the mound of the arch assumes a crescent form—called the aortic crescent. This crescent is normal, but

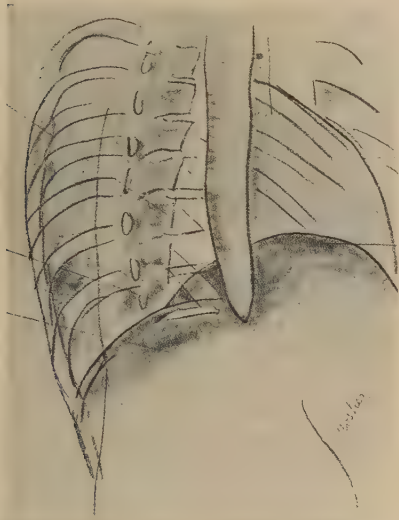


Fig. 576.—Tracing of a Roentgen-ray film of the esophagus of an adult male. The posterior mediastinum—the space between the esophagus and the vertebral column—when normal shows on the film as a light vertical streak. Pathology in the posterior mediastinum shows as a dark area. Invasion of the posterior mediastinum is very common in malignant disease of the esophagus. The esophagus is shown momentarily closed in the center of the cone of the diaphragm. This, as has been shown, is normal.



Fig. 577.—Tracing of a Roentgen-ray film of the esophagus. It shows the compression of the esophagus by the arch of the aorta. The crescentic form which the barium takes opposite the arch of the aorta—the aortic crescent—is normal. It is more marked, of course, when the arch of the aorta is enlarged.

it has very naturally been thought in some cases to indicate an aneurysm of the arch of the aorta.

**Length of the Esophagus.**—In men the distance from the incisor teeth to the beginning of the esophagus is 15 cm.; and in women, 14 cm. The distance from the incisor teeth to the bifurcation of the aorta is 26 cm. in men, and 24 cm. in women. In men the length of the esophagus from the incisor teeth varies from 36 to 59 cm., the normal or average distance being 40 cm. In women the figures are a little smaller, 32 to 41 cm., the average being 38 cm. When flexible bougies are used for measuring, from 1 to 3 cm. should be added to these measurements.

## LENGTH OF THE ESOPHAGUS AT DIFFERENT AGES\*

Teeth to Cricoid.		To Bifurcation.
Birth.....	7 cm. ( $2\frac{3}{4}$ in.).....	12 cm. ( $4\frac{3}{4}$ in.)
1 year.....	10 cm. (4 in.).....	14 cm. ( $5\frac{1}{2}$ in.)
2 years.....	10 cm. (4 in.).....	15 cm. (6 in.)
5 years.....	10 cm. (4 in.).....	17 cm. ( $6\frac{3}{4}$ in.)
10 years.....	10 cm. (4 in.).....	18 cm. (7 in.)
15 years.....	14 cm. ( $5\frac{1}{2}$ in.).....	23 cm. (9 in.)
Adult.....	15 cm. (6 in.).....	26 cm. ( $10\frac{1}{4}$ in.)

To Cardia.	Length of Whole Esophagus.
18 cm. ( $6\frac{3}{4}$ in.).....	10 cm. (4 in.)
22 cm. ( $8\frac{3}{4}$ in.).....	12 cm. ( $4\frac{3}{4}$ in.)
23 cm. (9 in.).....	13 cm. ( $5\frac{1}{4}$ in.)
26 cm. ( $10\frac{1}{4}$ in.).....	16 cm. ( $6\frac{3}{4}$ in.)
28 cm. (11 in.).....	18 cm. (7 in.)
33 cm. (13 in.).....	19 cm. ( $7\frac{1}{2}$ in.)
40 cm. ( $15\frac{3}{4}$ in.).....	25 cm. (10 in.)

\* From Stark.

For memorizing the length of the esophagus at different ages the following approximate figures are given: Birth, 7 inches; five years, 10 inches; fifteen years, 13 inches; twenty-five years or adult, 16 inches (Stark).

Add 3 inches for every five years.

Diameters of tubes for different ages:

To 8 years.....	9 mm.
From 9 to 15 years.....	11 mm.
From 17 years.....	12 to 14 mm.
Adults.....	14 mm. (average)

The esophagus begins 6 inches from the incisor teeth, back of the cricoid cartilage at the sixth cervical vertebra. It is 10 inches long, and goes through the diaphragm at the tenth thoracic vertebra, 16 inches from the teeth. It is crossed by the arch of the aorta back of the middle of the first piece of the sternum, 10 inches from the teeth. The measurements to be remembered in connection with it are, then, 6 and 10.

**The Diameter.**—Only in the region of the mouth of the esophagus is the diameter relatively fixed. The esophagus is constricted at four points. Of these the upper and the lower are the most important. The upper one is caused by the projection backward of the cricoid cartilage; the lower, by the encircling fibers of the diaphragm. The upper one hinders the introduction of the examining tube; the lower obstructs the passage of the esophagoscope into the stomach. The first constriction is a transverse slit slightly less than an inch wide; the second is about the same width. The long axis of this constriction is from right to left from behind forward. The lumen of the esophagus at this point is subject to wide variations, which depend upon the relaxation or the contraction of the diaphragm. In addition to these two important constrictions there are two others. Often they are not seen unless watched for, and they disappear completely if large tubes are used. The first of these supplemental constrictions corresponds to the arch of the aorta, and is found behind the junction of the first and second pieces of the sternum and in front of the fourth thoracic vertebra. The last constriction to be described, which is the third from above downward, is made by the crossing of the left bronchus in front of the esophagus, and occurs at the level of the fifth thoracic vertebra.

The following tables are compiled from Stark. They are of use for reference.

#### DIAMETERS OF THE ESOPHAGUS AT THE FOUR CONSTRICTIONS

Constriction.	Diameter.	Vertebra.
Cricoid.....	Transverse 23 mm. (1 in.) Anteroposterior 17 mm. ( $\frac{3}{4}$ in.)	Sixth cervical
Aortic.....	Transverse 24 mm. (1 in.) Anteroposterior 19 mm. ( $\frac{3}{4}$ in.)	Fourth thoracic
Left bronchus.....	Transverse 23 mm. (1 in.) Anteroposterior 17 mm. ( $\frac{3}{4}$ in.)	Fifth thoracic
Diaphragm.....	Transverse 23 mm. (1 in. +) Anteroposterior 23 mm. (1 in. →)	Tenth thoracic

**The Weak Triangle of the Esophageal Wall.**—The longitudinal fibers which form the external of the two muscular layers of the esophageal wall split posteriorly, turn right and left, and mark off a V-shaped space at the beginning of the esophagus. Having separated, the two bundles of fibers sweep forward and upward round the esophagus laterally to gain an attachment anteriorly to the vertical ridge in the center of the seal of the cricoid cartilage. This leaves the circular fibers of the upper posterior part of the esophageal wall unsupported by longitudinal fibers. The V-shaped gap is about an inch long, that is, it equals the height of the cricoid cartilage, and is directly behind it. This gap or triangle has proved to be a weak spot. It is through this that the diverticula of the upper end of the esophagus may herniate.

**The Subdiaphragmatic Esophagus.**—Dissection shows that there often is no subdiaphragmatic esophagus. In such cases the edges of the crura lie on the fundus of the stomach itself.

**The Cone of the Diaphragm.**—The two halves of the diaphragm meet in the midline and in front of the spine dip down in a cone, the tip of which is made by the crura. Through this cone runs the terminal part of the esophagus, the cone intervening between the esophagus and the liver. The cone is packed with loose connective tissue which binds the esophagus more firmly on the front and sides than behind. The median basal tips of the lower lobes of the lungs lie in the cone on the right and left of the esophagus. The cone of the diaphragm acts as a sleeve which keeps the terminal part of the esophagus in line. With falling of the diaphragm the cone of the diaphragm opens and flattens out. When this happens the terminal part of the esophagus is set afloat. The esophagus bobs about aimlessly on the flattened diaphragm and turns and twists as it pleases.

**The Liver Tunnel.**—The terminal part of the esophagus is not only surrounded by the cone of the diaphragm but it passes through a tunnel of surrounding liver. Between the esophagus and the liver the cone of the diaphragm and the crura which make the point of the cone intervene.

The liver is chiefly responsible for the shape of the lower end of the esophagus. According to the closeness of the investing liver the terminal part of the esophagus is either trumpet shaped or cone shaped. The lower end of the esophagus has liver on the right, in front, and in many cases a thin tongue of liver hooks round its left edge like a sickle. Behind the esophagus is the descending aorta which separates it from the vertebral column. The liver tunnel, as studied on the cadaver, varies greatly in length in different subjects. Owing to the extreme size of the liver at birth the liver tunnel in the baby is much longer and wider than in the adult.

A reconstruction of the lower end of the esophagus from frozen sections shows that the terminal portion within the cone of the diaphragm consists of two parts, a vertical and a horizontal. Where the two join, the esophagus twists on itself and turns to the left. At the twist the vertical part or arm comes to a point. Seen from within, the esophagus at the turn has in some cases a small central opening, in others there is an oblique slit. This is the clinical hiatus and is at the beginning of the horizontal arm. The anatomical hiatus is at the lower end of the horizontal arm where the esophagus actually passes through the diaphragm. This also is an oblique

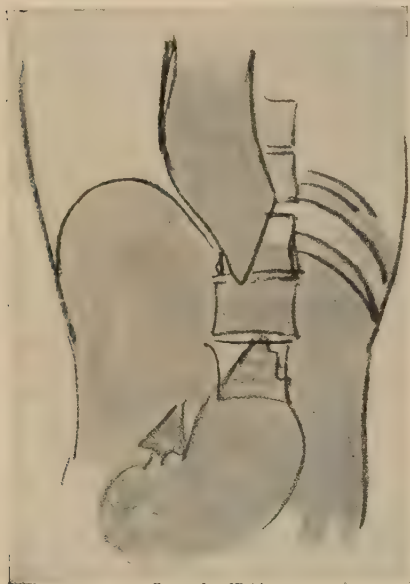


Fig. 578.—Case of stricture of the terminal portion of the esophagus of four years' standing. Female, aged thirty-two years. Old tuberculosis of left apex. The plate was taken with the diaphragm up. Only the right half shows. Notice the support which it gives to the terminal portion of the esophagus. Other plates show that the left half of the diaphragm supports the esophagus in the same way. When the diaphragm is up the cone of the diaphragm determines the shape of the lower end of the esophagus. In this instance the esophagus is dilated and the cone of the diaphragm is wider than normal.

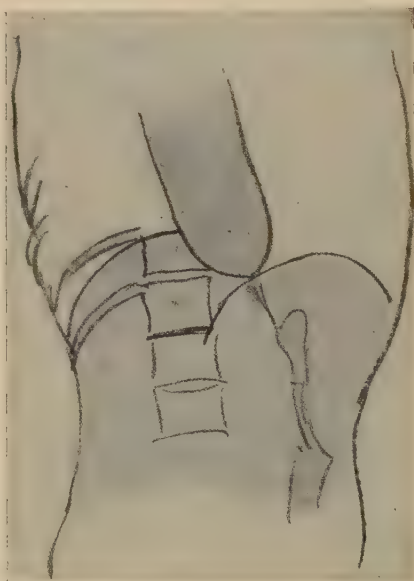


Fig. 579.—Same patient as Fig. 578. The diaphragm is down. When the diaphragm opens up and the terminal portion of the esophagus loses its support. Notice the change in the shape of the terminal portion of the esophagus.

slit like the clinical hiatus and is bounded by the two crura. The left crus crosses behind the esophagus on a slant, running forward and downward from the left above to the right below. The upper edge of the left crus mounds into the esophagus and makes the most conspicuous boundary of the clinical hiatus.

The horizontal arm of the terminal portion of the esophagus is imprisoned between the left crus behind and the edge of the posterior surface of the left lobe of the liver in front. The right crus comes in between the liver and the horizontal arm in front, but the right crus is thin and does

not leave its impression on the esophagus as does the left crus. Frozen sections show that the horizontal arm of the esophagus is flat and practically closed. Backward pressure on the liver will actually close the lumen of the horizontal arm, whereas pulling the liver forward or downward opens it. Tipping the liver forward and its posterior surface backward also closes the horizontal arm. As we study the anatomy of the esophagus on the dead the patency of the horizontal arm is seemingly controlled by the position of the liver.

**The Relation of the Lungs to the Esophagus.**—From the bifurcation of the trachea to the tip of the cone of the diaphragm the lungs are in close rela-

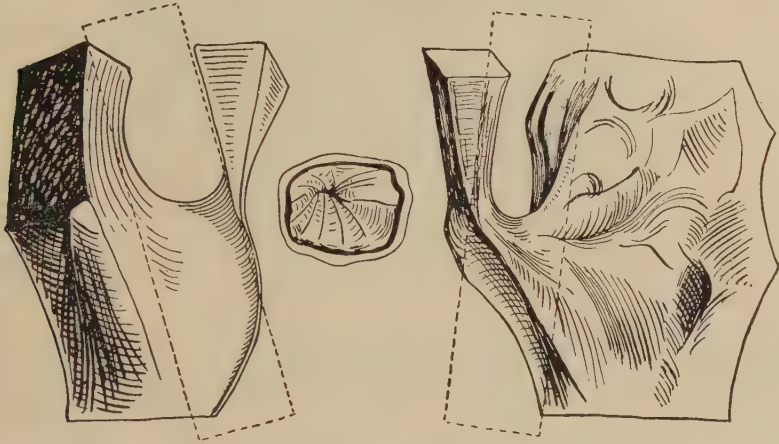


Fig. 580.—Wax reconstruction from frozen sections of the terminal portion of the esophagus of an adult cadaver. The first cut shows the esophagus from behind. The terminal portion of the esophagus is divided into a vertical part and a horizontal part. The vertical part or arm is pyramidal in shape and ends in a point. Below this the esophagus turns on itself to the left and makes the horizontal arm of the terminal portion. The dotted lines represent the posterior or left crus as it crosses the esophagus from behind. Notice that it lies behind the horizontal arm and that where it crosses the esophagus the vertical arm comes to a point. The second large cut shows the same reconstruction from the front. The vertical and horizontal arms of the terminal portion of the esophagus are shown as before. The dotted lines again show the position of the posterior crus. The small central cut shows the lumen of the vertical arm. The surgical hiatus occurs at the junction of the vertical and the horizontal arms of the terminal portion of the esophagus. When the horizontal arm of the esophagus is closed the surgical hiatus appears as a small round central opening. When the horizontal arm is open the hiatus becomes an oblique slit which follows the direction of the left crus and into the lumen of which the left crus often makes a distinct mound.

tion with the esophagus. The anterior median basal tips of the inferior lobes fill the cone of the diaphragm. The anterior median basal tips of the lower lobes of the right and left lungs when distended with air press upon the esophagus and momentarily close it. The anterior median tip of the left lobe when inflated extends forward like a thumb and makes a deep notch in the left side of the esophagus. This action of the lung tip can be seen through the fluoroscope, and the films of the lower esophagus often show the circular impression of the expanded lung tip. Just above the point where the lung tips come in contact with the esophagus its axis is transverse. But where the lung tips press on the esophagus the axis changes and becomes anteroposterior.

Small ligaments run from each of the anterior median lung tips to the sides of the esophagus and join across its front face around the margin of the hiatus.

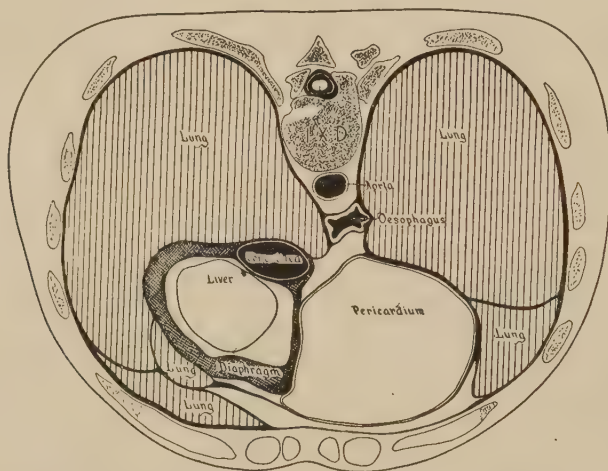


Fig. 581.—Drawing from a tracing of a frozen section of an adult cadaver through the ninth dorsal vertebra. The drawing shows the intimate relationship between the lungs and the esophagus at this level.

**The Behavior of the Terminal Portion of the Esophagus.**—Fluoroscopic observations to show the behavior of the terminal portion of the esophagus are best made on a case in which there is a slight stricture of the lower end, that is, in a case of so-called “cardiospasm.” (See Fig. 584.)

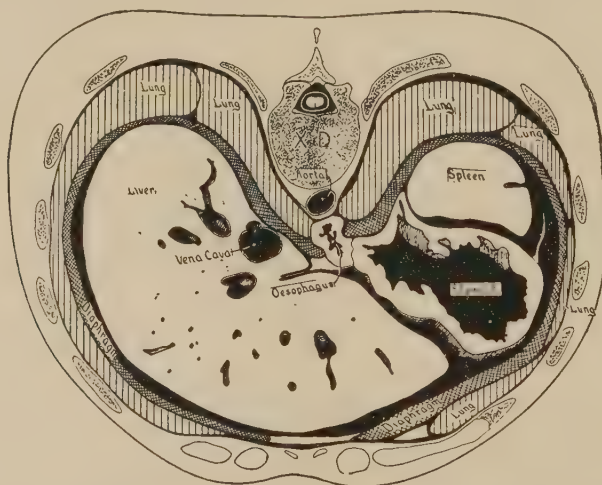


Fig. 582.—Drawing from a frozen section of the same cadaver shown in Fig. 581. The section is made a vertebra lower down. The drawing shows the intimate relationship between the basal lung tips and the esophagus. Notice how the long axis of the esophagus has become anteroposterior instead of transverse, as in the section above. This the writer believes is due to the pressure of the expanded lung tips.

When the diaphragm rises the terminal portion of the esophagus, within the cone of the diaphragm, rises with it and the end of esophagus bows slightly to the right. At the beginning of the descent of this diaphragm

the esophagus appears to end in a nipple-like point in the cone of the diaphragm. As the diaphragm continues to descend the anterior median lung tips of the inferior lobes are seen to fill with air. Immediately about  $\frac{1}{2}$  inch of the esophagus seems to be amputated, and the esophagus ends bluntly. Films show that when the diaphragm goes down the stomach descends

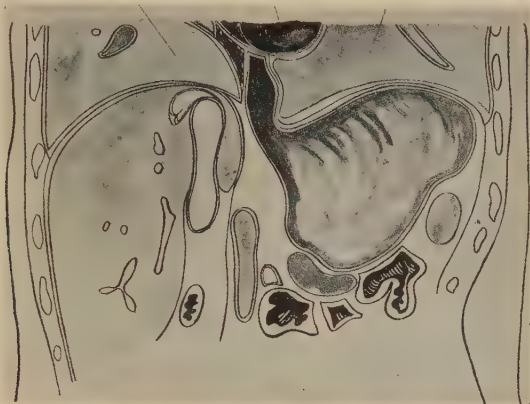


Fig. 583.—Drawing copied from Testut to show the relation between the lungs and the terminal portion of the esophagus.

and swings toward the median line. The stomach carries the horizontal arm of the esophagus with it, straightens it out, and brings it into line with the vertical part of the esophagus, thus undoing the kink and opening the esophagus.

The action of the lung tips is naturally brought out best by forced inspiration. When the diaphragm starts to ascend the esophagus opens and barium pours into the stomach.

**The Pleated Segment.**—The terminal portion of the esophagus which has just been rather minutely described can be described more simply by



Fig. 584.—Retouched tracing from Roentgen-ray film. Male, twenty-five years. This is from a case of cardiospasm.

calling it the pleated portion, as it contains many deep vertical folds which nearly close the esophagus in its resting condition. These, of course, twist with the esophagus from right to left. This pleated segment of the esophagus is strikingly like the common rubber tobacco-pouch, the mouth of which is closed by folds which have a permanent spiral twist. From the description of

the terminal portion of the esophagus which has just been given it is easy to see that slight pathological conditions at this point can cause trouble.

**The Appearance of the Normal Esophagus.**—Under good illumination the color of the mucous membrane of the esophagus is a whitish pink like that of the mouth. Poorly lighted or when inflamed the color changes to a red of varying depth. After trauma the mucous membrane soon becomes edematous. When examined with small tubes the walls of the esophagus are thrown into large longitudinal folds. These folds are seen indenting the circumference of the central dark area which represents the lumen. The folds are especially numerous at the mouth of the esophagus behind the cricoid cartilage. They make it hard to be sure of the pathological lesions

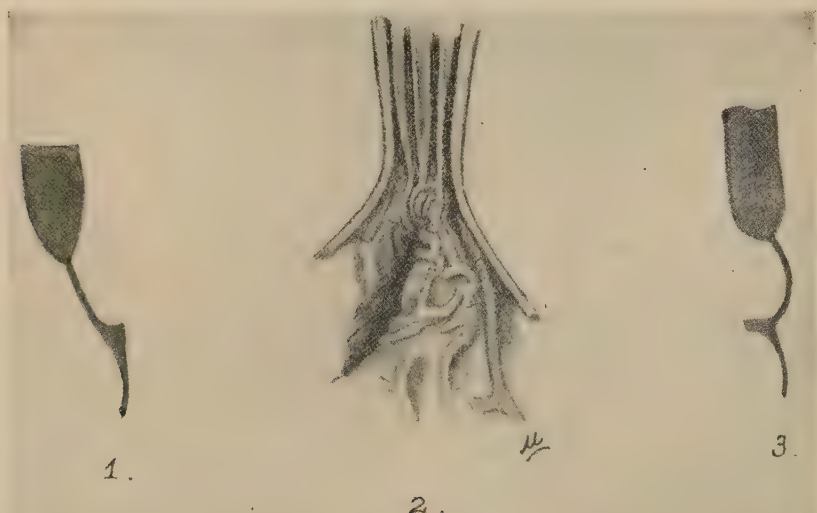


Fig. 585.—The esophagus of a child. The illustration is to show the vertical pleats at the lower end of the esophagus. They are continuous for a short distance with the rugæ of the stomach. On the anterior wall of the esophagus one or two pleats are continuous with long rugæ which skirt the lesser curvature of the stomach. One of these rugæ is shown in the illustration. In the resting or collapsed state of the terminal portion of the esophagus the barium runs in the grooves made by the pleats. This accounts for the pictures given by the barium meal. Figures 1 and 2 are such pictures. In these the esophagus is seen to come to a point. This is the point of occlusion due to the inflated lung tips. Below this there is a narrow ribbon of barium. This represents a channel between the pleats in the closed esophagus.

in this locality. Below the cricoid cartilage and in the cervical portion, the lumen is seen to enlarge with inspiration and to close down again, but not entirely during expiration. When a large tube is used the examiner can often look a long way down the esophagus. As the esophagoscope reaches the first piece of the sternum the pulsation of the arch of the aorta can be seen through the anterior wall. A little lower the heart mounds into the anterior wall on the left. The beating of the heart is visible and when the tube has passed beyond and the heart lies on it, the tube often vibrates in unison with its beating. The hiatus of the esophagus appears as a slit or a rosette. The axis of this opening through the diaphragm is oblique, running from right to left from behind forward. The horizontal arm of the terminal portion of the esophagus usually shows no lumen, but opens as the tube

passes through it. The mucous membrane here is so much like that of the stomach that it is hard to tell where the esophagus ends and the stomach begins. The mucous membrane of the stomach, however, is a darker red than that of the esophagus and the longitudinal folds of the esophagus give place to the familiar *rougie* of the stomach.

**The Movements of the Esophagus.**—When the esophagus is watched through the fluoroscope it is seen that during the act of swallowing the larynx moves quickly upward for about an inch and carries the upper end of the esophagus with it an equal distance. Pouch cases demonstrate this very prettily. When the diaphragm ascends the terminal portion of the esophagus, that is the part within the cone of the diaphragm, also moves upward about an inch and the terminal portion of the esophagus bends to

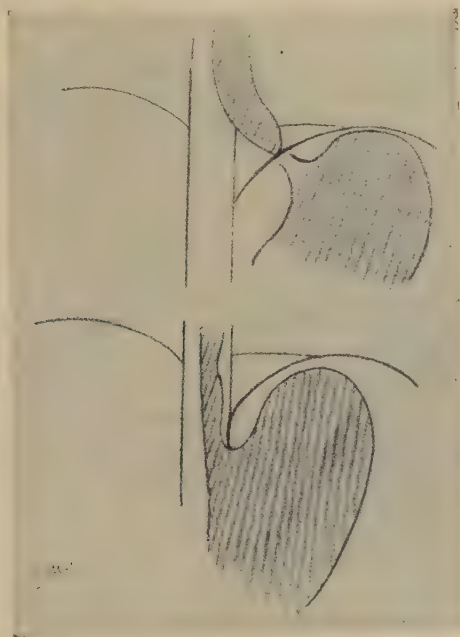


Fig. 586.—Diagrammatic drawing to show the changes in the horizontal arm of the terminal portion of the esophagus as the position of the diaphragm changes. Upper drawing: Diaphragm up, esophagus closed. Lower drawing: Diaphragm down, esophagus open.

the right. It is an exaggeration of this physiological bend occurring with expiration which gives the familiar bend to the right when the esophagus slips from its vertebral bed in cases of stricture of the lower end of the esophagus. Waves of peristalsis are occasionally seen to pass along the esophagus from above downward. They are noticeable only when the esophagus is moderately distended and even then only infrequently. They are conspicuously infrequent in pathological conditions. In pathological conditions it is not uncommon to see the lower end of the esophagus hammered in a lively fashion by the heart beats. When seen through the esophagoscope the esophagus is never twice alike even in the same individual. At the level of the fourth thoracic vertebra (24 cm. from the teeth) the throbbing of the arch of the aorta can be detected if watched for, and a

little lower, at the level of the seventh and eighth thoracic vertebræ (30 cm. from the incisor teeth), the backward mounding of the heart and its bearing are visible.

If a relatively small esophagoscope is used for the examination the esophagus opens with inspiration and partly closes with expiration. These changes occur chiefly in the thoracic portion, and are due to the negative intrathoracic pressure. If a large tube is used the esophagus stands wide open after the cricoid cartilage has been passed, and the respiratory changes nearly disappear.

During swallowing peristaltic movements pass along the esophagus from above downward, while in vomiting the movements are reversed.

**Distensibility.**—All the constrictions of the esophagus are distensible. The upper constriction is less so than the others, so that this is the one which gives the greatest trouble in esophagoscopy. The normal wall of the thoracic esophagus in the adult, according to Jackson, will

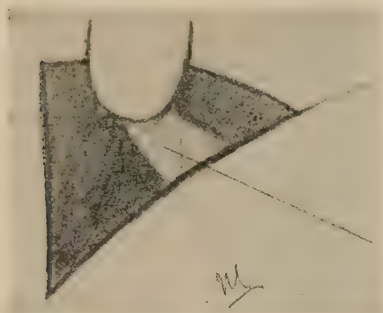


Fig. 587.—Tracing of the left lung triangle with the lung tip inflated. The esophagus was seen to be momentarily occluded and to end in a blunt point in the middle of the lung triangle. The shutting off was not absolute because there is a faint shadow in the esophagus below the point of constriction.



Fig. 588.—Tracing of a Roentgen-ray plate of stricture of the terminal portion of the esophagus. Female, aged thirty-two years. The diaphragm is down and the lung tips expanded. There is a gap in the esophagus which corresponds accurately to the dark lung triangle.

stretch 2 cm. without rupture. At times foreign bodies stretch it more than this. In infants a tube 7 mm. should pass readily, and in the adult a tube which has a diameter of 14 mm. In infants a flexible bougie 8 mm. could pass, and in adults, one that measures 14 mm. The distensibility of the esophagus is much greater in the living than in the dead.

With light stretching the transverse diameter of the esophagus is 23 mm. at the cricoid cartilage and 17 mm. anteroposteriorly. The diameter of the esophagus as it goes through the diaphragm is 24 to 25 mm. Two stomach-tubes can be passed side by side. Bruenings states that the esophagus at its mouth can be dilated to 30 mm. without danger.\*

At the lower end of the esophagus von Mikulicz in his operation for

\*Any attempt at routine clinical use of the maximum distended diameter of the esophagus will be attended with a high percentage of mortality. [EDITOR.]

cardiospasm stretched the lumen to 7 cm. so that the hiatus had a circumference of 16 cm.

On the dead, when the esophagus is stretched transversely only, it dilates to 40 mm., or  $1\frac{1}{2}$  inches. The ordinary full-sized tooth plate is  $2\frac{1}{4}$  inches (57 mm.) broad. A fifty-cent piece is  $1\frac{1}{8}$  inches (30 mm.) wide. Since the transverse diameter of the esophagus hovers round 1 inch it would seem as if this coin should pass readily in an adult. The direction in which the esophagus will stretch the most is from side to side. For this reason oval tubes take up the slack in the esophagus along anatomical lines better than round ones.

**Cadaver Findings in Casts of the Adult Esophagus.**—In a series of thirty-five wax casts of the esophagus and the stomach made on adult cadavers the following were the conspicuous findings: The upper edge of the left lobe of the liver often made a crease across the front face of the esophagus and above this the esophagus had a spindle-shaped dilatation. The left crus as it runs upward behind the esophagus frequently made a deep furrow in the posterior wall of the esophagus and a deep notch in its left wall. Between these two constrictions the esophagus in a number of cases had a ball-like dilatation. In four such specimens the upper constriction was replaced by an annular stricture. So far the annular strictures which have been found in dissecting room cadavers were all at this level, that is, at the upper border of the left lobe of the liver. The lower part of the esophagus seems to be caught between the liver edge above and the left crus below. The globular dilatation of the esophagus just described may occur, however, without an actual stricture at the upper limit. The pressure of the liver edge and the left crus alone seems to be able to produce it. The casts show three specimens of this.

**Cadaver Experiments in the Adult.**—If backward pressure is made on the left lobe of the liver, the abdominal wall being flaccid, and the lumen of the esophagus is watched through the esophagoscope a crescentic mound appears in the right quadrant of the field. Very strong pressure will practically close the esophagus. This mounding into the field of the esophagoscope is caused by the liver. Anatomically it may be either the upper edge of the left lobe or the edge of the spigelian lobe where this fits into the lesser curvature of the stomach and bounds the terminal portion of the esophagus on the right. If downward traction is made on the liver the lumen of the esophagus increases in size.

**Lymphatics.**—The lymphatics of the esophagus enter both the mediastinal and the cervical glands.

**The Nerve Supply.**—The swallowing center is situated in the medulla just above that of respiration, and stimuli proceed to this from the pharynx by the fifth, the superior laryngeal, and vagus nerves. The afferent fibers in the glossopharyngeal nerve exercise a powerful inhibitory influence on the deglutition center as well as on that of respiration.

**The Cardiac Sphincter.**—In all physiologies the statement is found that the lower end of the esophagus is closed by the cardiac sphincter. This is held to be in a permanently contracted state but the force of the closure is slight. From time to time there are rhythmic alternations of contraction and relaxation. The tonic condition of the sphincter is controlled by the vagus nerve, stimulation of which causes relaxation with an after-effect of strong contraction.<sup>1</sup>

According to Jackson,<sup>2</sup> the presence of this sphincter is doubtful and anyway is not the chief agency through which the regurgitation of food is prevented. This observer maintains that the kinking of the esophagus below the opening of the diaphragm and the increase of this twist by distention, plus the pinching by the crura have much more to do with keeping the food in the stomach than the presence of the cardiac sphincter. Fluoroscopic observations support this view.

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#### CARDIOSPASM

**Definition.**—The term “cardiospasm” is a relic of the days before the esophagus was examined by sight. It means an obstruction of the lower or cardiac end of the esophagus by spasm. Recent studies with the esophagoscope have placed stricture above spasm as a cause of the condition. And in addition to stricture another cause has been found in bends and twists of the terminal portion of the esophagus. In the majority of the writer’s cases there was either a stricture or a twist. In order to determine accurately pathologic conditions at the lower end of the esophagus it is necessary, in my opinion, to examine the patient under a general anesthetic and with a large esophagoscope. Webs and small strictures are missed with small tubes.

**Symptoms.**—The patient has difficulty in swallowing and feels that food lodges at the bottom of the esophagus. There is a feeling also of pressure at the tip of the sternum. If the case is of mild degree after the feeling of pressure has lasted a short time it lets up and the food passes into the stomach. Or the patient takes a drink, preferably warm, and the food passes on. A cold drink generally aggravates the trouble. As the closure of the esophagus progresses the patient restricts his diet more and more, until finally he comes to live on semisolids and liquids. Vomiting of food retained in the esophagus now becomes a symptom. Finally, when the closure is nearly impassable, the patient becomes almost a living skeleton. He is continually eating and continually vomiting. He is ostracized from the table. On lying down the full esophagus overflows into the larynx, and the patient’s nights are made hideous by coughing and strangling. If one of the advanced cases yields to the temptation to eat solid food like meat or swallows the pulp of an orange then the narrowed esophagus becomes completely stopped and a surgical emergency ensues.

**Course.**—Cases of cardiospasm are seldom seen before twelve years of age. Most of the patients are between thirty and fifty. The majority of the cases progress slowly and run on for years. Many patients seek relief only in the terminal stages. One such patient came to the writer who had had symptoms for sixteen years.

**Cause.**—The theory which has long been accepted as the cause of car-

diospasm is that spasm starts the condition and that spasm finally is superceded by stricture. Spasm is a peculiarity of the alimentary canal when it is irritated. In examining the esophagus without an anesthetic spasm delays the introduction of the tube at the mouth of the esophagus and delays its further progress at the clinical hiatus. The esophagus has the habit of spasm, especially at the places just mentioned. The writer, however, has yet to see



Fig. 589.—Tracing from a Roentgen-ray plate of a typical case of stricture of the terminal portion of the esophagus. Female, aged fifty years. Symptoms of obstruction for three years. The plate is taken slightly from the side-right oblique. The diaphragm is up. The left lung triangle shows clearly. The esophagus is dilated and bends slightly to the right. The esophagus tapers to the typical nipple. It appears to touch the diaphragm. This is due to the rotation of the body to the right. The nipple is really a little above the diaphragm. The horizontal arm is closed and is represented by a gap between the nipple of the esophagus and the fundus of the stomach. Under ether a central narrowing of the esophagus was found at the level of the esophageal nipple and below this there was a crescentic web or fold mounding into the right quadrant of the esophagus. The narrowed esophagus dilated easily. A large bougie can now be passed readily. The patient came to the hospital starving. She went home eating normally. The tube pictures are shown in the small diagrams at the bottom of the plate. The advocates of the spasm theory would maintain that the central narrowing was due to spasm. It persists, however, under ether and a large esophagoscope is halted by it and has to be coaxied through slowly by steady pressure. In most instances the withdrawal of the large tube discloses one or two breaks in the mucous membrane of the esophagus in the region of the central narrowing, giving the observer the feeling that an actual gluing of the esophagus was present at this point or that a web was present which was broken by the passing of the esophagoscope. If a small tube is used for the examination and this is followed by dilatation with the water bag or the mechanical dilator no accurate idea can be gained of the actual condition. Even when the esophagus is put on the stretch by as large a tube as can be safely introduced it is not easy to interpret correctly the tube picture.

what he could call a pure case of cardiospasm. All his cases so far have been due to some degree of stricture or twist of the terminal portion of the esophagus. The writer makes it a routine to try the effect of antispasmodics like atropine or benzyl benzoate. So far these remedies have given only insignificant relief.

The cone of the diaphragm which sheaths the terminal portion of the esophagus can pick up infection from within and from without. Within

from the thorax and without from the upper abdominal cavity and the organs located there, namely, the stomach, the liver, and the gall-bladder. It has been known for a long time that cancer of the fundus of the stomach, infection of the gall-bladder, and even disease of the appendix are at times associated with what was called spasm of the lower end of the esophagus.

The writer has one specimen of annular stricture of the terminal portion of the esophagus opposite a tubercular lesion in the thoracic spine, one case associated with old lumbar Pott's disease, and three cases in the living where the stricture was associated with tuberculosis of the lung.

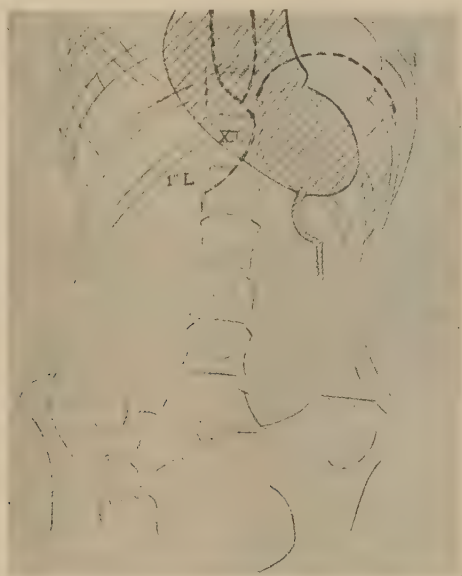


Fig. 590.—Tracing from a Roentgen-ray plate of a long-standing case of so-called "cardiospasm." The patient was a woman of fifty. The condition has existed some sixteen years. Notice how the esophagus is dilated and swings to the right. On examination under ether the opening through the diaphragm was found to be a small fibrous opening which could be dilated only to the size of a No. 22 French bougie. Notice how the opening is displaced downward two vertebrae and a half and displaced to the left the width of one vertebra. The horizontal arm of the esophagus is seen as a narrow perpendicular ribbon. In this case there was a general visceroptosis and the greater curvature of the stomach was at the bottom of the pelvis. The downward pull of the fallen and distended stomach has straightened out the horizontal arm of the terminal portion of the esophagus. The position of the normal esophagus is shown by the dotted lines. The esophagus is shown diagrammatically with the diaphragm up. It ends in the typical physiological point which I have come to feel is the site of election for the strictures which are the cause of the majority of cases of cardiospasm.

In one reported case of cardiospasm which came to abdominal section the liver tunnel was obliterated by adhesions.

It is not always necessary to look outside of the esophagus for the pathology responsible for the stricture at the bottom of the condition so long called cardiospasm. The configuration of the terminal portion of the esophagus predisposes to trouble. For instance and first of all there is the twist of the esophagus. Then there is the momentary closure when the diaphragm is down, followed by the secondary closure caused by the pressure of the lung tips on forcible breathing. The upward movement of the terminal portion of the esophagus is accompanied by a bend to the right.

One form of cardiospasm seems but an exaggeration of this normal happening which occurs eighteen times a minute.

There is an analogy between the upper end of the esophagus and the lower end, which throws some light on the ease of the formation of stricture in both places.

The posterior surface of the left lobe of the liver reinforces the front face of the terminal portion of the esophagus in the same manner that the cricoid cartilage stiffens the upper part of the esophagus. In both places



Fig. 591.—Tracing from a Roentgen-ray plate of a case of stricture of the terminal portion of the esophagus. Symptoms for three years. Mrs. C., aged fifty years. The diaphragm is up and there is a marked bend of the esophagus to the right. There is a large lung triangle. Under ether a central narrowing was found with a crescentic mounding into the right quadrant. Dilatation up to 30 French was accomplished with bougies. A metal olive on a metal staff could not be passed, neither could the mechanical dilator. The patient came to the hospital with pernicious vomiting associated with acidosis. Since the examination a flexible bougie cannot be passed. The patient left the hospital swallowing normally and continues to do so after a period of four months. Figure 592 shows that the terminal portion of the esophagus is reduced to a narrow ribbon. Under ether the flexible bougies followed the bend of the narrow esophagus, the metal instrument would not. Without ether even flexible bougies will not follow it. As the patient is swallowing normally it would seem as if the anteroposterior diameter of the ribbon-like narrowing was greater than the vertical. It is possible that this narrowing is due in part to the pressure of the lung tips, because if the shadow of the breast is excluded it is seen that there is a very large lung triangle.

the vertebral column is a rigid wall behind. On entering the esophagus a bougie has to displace the cricoid; on leaving it must displace the liver. When food enters the esophagus the cricoid cartilage moves forward and upward. When it leaves the esophagus the liver moves forward and downward.

The liver tunnel differs from the cricoid tunnel—if I may use this term for the moment—in that the esophagus in the middle of the liver tunnel twists on itself and narrows to a point at the site of the twist or kink. This produces the esophageal nipple in normal swallowing.

The posterior face of the left lobe of the liver, especially its upper edge, which so often actually indents the esophagus, produces a narrowing of the esophagus which accentuates any form of trauma to which the lower part of the esophagus is subjected. Foreign bodies have to squeeze by the liver edge and through the liver tunnel. The passage of hot fluids or caustics is



Fig. 592.—Stricture of the terminal portion of the esophagus. Female aged fifty years. Tracing of a Roentgen-ray plate. Diaphragm down. The oval drawings are the tube pictures obtained during the examination under ether. In order to make them legible they are drawn out of scale and slightly larger than actual size. Each oval drawing has a pointer leading to the portion of the esophagus where it was obtained. First tube picture, counting from above downward. The esophagus shows a small central opening. On the right there is a crescentic mounding into the lumen of the tube. This mounding into the tube is very common. It is usually found below the central narrowing. Anatomically this may be caused by the pressure of the edge of the left lobe of the liver or by the edge of the spigelian lobe. It can be due also to the inward folding of the right quadrant of the esophagus. In some cases this mounding acts like a true web comparable to the webs found in the pyriform sinus. There is still another possible explanation of this mounding into the right quadrant. The horizontal arm of the terminal portion of the esophagus turns sharply to the left. At the beginning of the turn backward pressure would tend to fold the right quadrant inward. This folding can be imitated by approximating the thumb to the first finger. The fold between the base of the thumb and the index-finger behaves like the folding of the right quadrant of the horizontal arm.

The second tube picture shows the esophagus narrowed to a small central opening.

In the third tube picture the central opening has given way to an anteroposterior slit. The long axis of the esophagus which previously has been transverse has become anteroposterior owing to the pressure of the lung tips on the sides.

The fourth tube picture was obtained by advancing the esophagoscope into the horizontal arm and then on into the stomach. On withdrawing the tube a small slit was seen in the mucous membrane of the esophagus in the middle line posteriorly. This slit I take to be due to the breaking of a web or the ungluing of a vertical fold. One or two slits in the mucous membrane are common after the dilatation of a stricture of the terminal portion of the esophagus by the gradual advance of the large esophagoscope.

delayed. The upper edge of the liver acts as an intermittent clamp on the esophagus and its constant hammering seems to reinforce ulcerative processes and to localize strictures at this level.

In 9 cases of cardiospasm the esophagoscope showed that below the stricture the rest of the esophagus was open and normal. In one, however,

the whole of the liver tunnel was narrowed. My conception of cardiospasm, therefore, is that it is due in many cases either to a stricture of the esophagus at the beginning of the liver tunnel or in the liver tunnel or to a narrowing of the liver tunnel as a whole. Such strictures may be due to causes acting within the esophagus—trauma or caustics—or to causes acting from without.

In the writer's cases the chief cause of cardiospasm has been stricture of the terminal portion of the esophagus somewhere in the cone of the diaphragm. There is usually a single stricture. However, the whole liver tunnel and the cone of the diaphragm which it encloses and supports may be narrowed. The commonest sight of the stricture is at the twist between the vertical and horizontal arms of the terminal portion of the esophagus. Many of the strictures at this point are but a slight gluing together of the folds of the mucous membrane of the esophagus because the steady pressure of the end of the esophagoscope soon forces the esophagus open and the

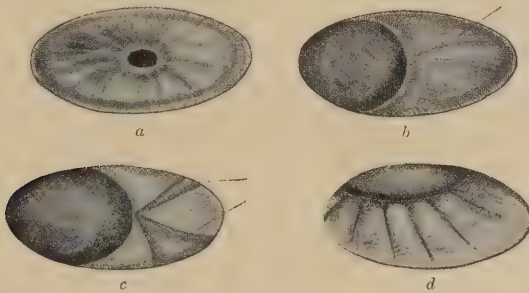


Fig. 593.—*a*, Small central opening at the beginning of the liver tunnel found in 4 of 10 cases of cardiospasm. *b*, Crescentic web-like stricture found in 5 of 10 cases of cardiospasm. This was placed at the beginning of the liver tunnel. On introducing the mechanical dilator, spreading it until marked resistance was felt, and then withdrawing it closed, it was found that these strictures acted like webs at the upper end of the esophagus; that is, they were readily divulsed. On withdrawing the dilator one or two slits were usually found in the mucous membrane of the esophagus and the esophagoscope would then slip into the stomach without meeting obstruction. Below these web-like strictures the subdiaphragmatic esophagus was normal. I feel that the upper edge of the liver is probably glued to the esophagus and reinforces these strictures. *c*, This plate shows the slits in the esophageal mucous membrane after divulsion of the crescentic stricture. *d*, This plate shows the normal subdiaphragmatic esophagus usually found below the crescentic stricture.

tube proceeds without further obstruction into the stomach. On the withdrawal of the tube, however, a slit will be found in the mucous membrane at the point where the tube halted. The same thing is found when the tube or the dilator breaks a web in the upper part of the esophagus behind the cricoid cartilage. It takes but a very slight inflammatory process to produce a gluing of the folds of the esophagus at the point of the twist, in fact, the twist predisposes to such a happening.

The second great cause of cardiospasm is falling of the diaphragm and the release of the terminal portion of the esophagus from the support of the cone of the diaphragm. This results in the formation of a trap at the lower end of the esophagus. Most but not all cases of cardiospasm have a fallen or ptotic diaphragm. Associated with this there is often a general visceroptosis. In the type of case now under discussion the diaphragm is always ptotic. The falling of the diaphragm opens up the cone of the diaphragm and sets the terminal portion of the esophagus free to sag and

twist as it may. Mobilization and displacement, therefore, of the terminal portion of the esophagus are the cause of the second type of case.

That there is no obstruction in the esophagus is shown by the fact that that not only the full-sized esophagoscope but a No. 40 bougie will pass into the stomach of the patient without difficulty.

In many cases of so-called "cardiospasm" there is a ptosis of the diaphragm or even a general visceroptosis. In the normal individual the downward excursion of the diaphragm is the same as the upward; that is, both are about  $1\frac{1}{2}$  inches. In a markedly ptotic diaphragm the downward excursion is either nothing at all or only  $\frac{1}{4}$  inch. The upward excursion

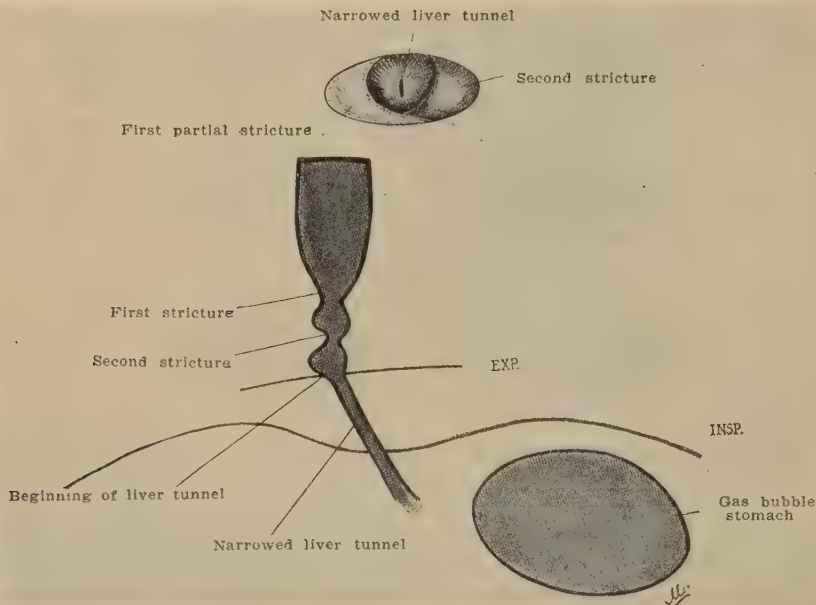


Fig. 594.—Male aged forty-five years. Preoperative diagnosis—cardiospasm. Upper figure: The esophagoscope showed two crescentic strictures. The first was on the left and about an inch above the liver edge; the second was  $\frac{1}{2}$  inch lower down and  $\frac{1}{2}$  inch above the liver edge. The second partial stricture was on the right. The liver tunnel was narrowed to a central opening, which could be dilated to take only a No. 36 French elastic bougie. Lower figure: Roentgen-ray plate taken before operation. This plate shows the whole of the liver tunnel narrowed to a ribbon. The two strictures above the liver tunnel are shown in the plate to be really annular, not crescentic strictures, as they appeared through the esophagoscope.

remains the same as in the normal (Holmes). The Roentgen-ray picture of such a diaphragm shows that the two halves are nearly flat.

In cases of cardiospasm due to mobilization and displacement of the terminal portion of the esophagus the passing of a bougie temporarily brings the thoracic part of the esophagus into line with the terminal portion. When it is withdrawn, however, the previous condition is re-established and little, if any, improvement results.

**Prognosis.**—What is the practical application of the foregoing facts? In other words, what is the prognosis of a given case of cardiospasm? If the Roentgen ray shows a stricture in the region of the liver tunnel and the examination with the esophagoscope shows this to be a lateral web, the

web can be divulsed with the mechanical dilator under ether. Normal swallowing is restored and the occasional passing of a bougie for a short period makes the cure permanent. If the whole liver tunnel is narrowed, a gain in the patient's ability to swallow can be secured if the stricture is not too tight. Such a patient, however, must lead a bougie life. In the cases in which the diaphragm is flattened and the thoracic esophagus makes a marked trap, dilatation offers but little help unless a stricture has been superimposed by inflammatory reaction within or without the esophagus.

**Treatment.**—The treatment of stricture of the terminal portion of the esophagus is to find and dilate the stricture. When it is due to a lateral web, or to a light gluing of the esophageal folds at the point of the twist, the treatment is easy. Cases with this simple pathology are not necessarily light cases or early cases; often a long-standing case will be found to have an easily remediable pathological condition behind it. This is why such dramatic relief

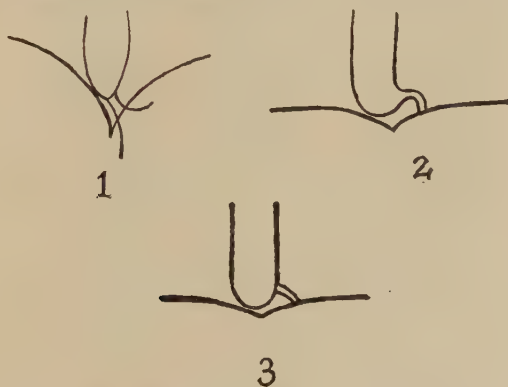


Fig. 595.—Diagrammatic drawings to show the twisting of the terminal portion of the esophagus when the diaphragm falls and the support of the cone of the diaphragm is withdrawn from the esophagus.

1, The normal cone of the diaphragm with the esophagus ending in the physiological point.

2, The diaphragm is flat and the terminal portion of the esophagus becomes a trap.

3, The diaphragm is flat. The terminal portion of the esophagus has swung round so that a twist has been added to the trap. (See Fig. 597.)

is obtained in many of these cases, why, for example, a patient who is actually starving is restored to normal swallowing and to a normal diet in a matter of a week. When, however, there is a thick annular stricture, say, at the beginning of the liver tunnel, or the whole of the liver tunnel is narrowed, or the hiatus has become a firm fibrous ring, the problem is far from easy. In other words, the surgeon's patience, judgment, and skill are severely taxed by cases which fall into this class.

There are a number of methods of dealing with these cases and more than one method is successful. The writer's routine is as follows: The fluoroscope and the Roentgen-ray film are called upon to give all the information they can as to the nature and the location of the obstruction in the esophagus. If, as often happens, the patient comes to the hospital semi-starved, giving a history of frequent vomiting, the urine is tested for acetone because these patients not infrequently are in a condition of acidosis. The almost pernicious vomiting of some cases is due in great

part to this condition. Glucose is given intravenously and by rectum until the acidosis has cleared. Then the patient's esophagus is washed out to free it from barium and remnants of food and the patient given a general anesthetic and sent to the examining table.

The examination is made with a full-sized oval esophagoscope. The tube should be 21 inches long, otherwise it will not pass beyond the stricture when it is divulsed and give the condition of the esophagus from this point on to the stomach. Two accessories are essential—suction for cleaning the esophagus and a window plug for ballooning it and bringing the lumen of the stricture into view. The writer prefers a large suction tube introduced through the esophagus, the small suction tube incorporated in the esophagoscope has proved inadequate in his hands. There is no pathological



Fig. 596.—Male aged seventeen years. The diaphragm is down and the bend of the esophagus which was present when the diaphragm was up has straightened out. The terminal portion of the esophagus appears to be twisted on its vertical axis. Notice the lung triangle.

condition at the mouth of the esophagus as a rule,\* so the esophagoscope is passed beyond the cricoid with the plunger. Once well in the esophagus the plunger is removed and the esophagoscope is carried down to the point of obstruction by sight through the tube. The field is made clean by suction and the character of the obstruction is determined as well as it can be at a distance of 20 inches and from the eye. The esophagoscope shows usually either a crescentic web filling the right half of the field or the esophagus is contracted to a small central opening.

When the esophagoscope is halted by the narrowed esophagus the end of the tube is held firmly against the strictured area and an attempt is made to advance the tube by gentle pressure, working the end of the tube slightly to the left in order to follow the long axis of the horizontal arm of

\* It might be well to make sure of this by preliminary examination. [EDITOR.]

the esophagus. In the cases in which the maneuver succeeds it is a matter of only half a minute or a minute before the end of the tube passes the stricture and continues on into the stomach. On withdrawing it a vertical slit is often found in the mucous membrane of the esophagus in the right quadrant of the tube. It is evident that in cases where the tube can be advanced in the manner just described and the obstruction removed there cannot be a very firm stricture. The best explanation is that there was a light gluing of the esophageal folds at the point of the twist. The crescentic web so often seen on the right may be simply a folding inward of the right half of the beginning of the horizontal arm which is caught in this position by some inflammatory process.

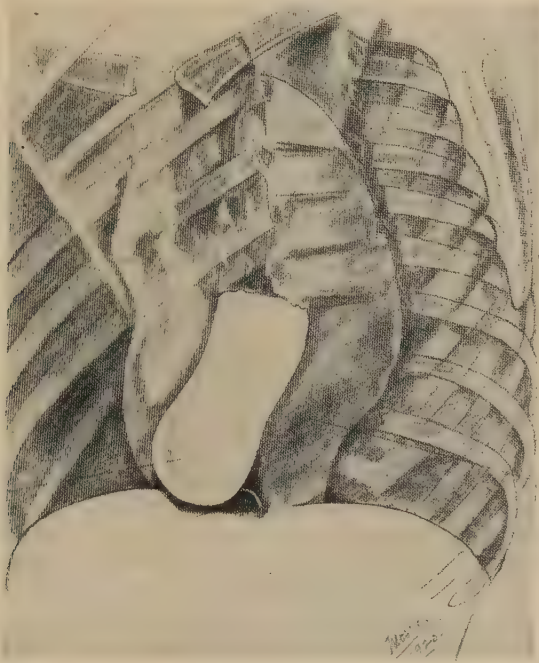


Fig. 597.—Retouched tracing of a Roentgen-ray film of the esophagus of a man of fifty-five years. The esophagus is much dilated and the terminal portion twists on the upper part in an attempt to find the hiatus in the fallen and flat diaphragm. The terminal portion of the esophagus is also very narrow.

If the esophagoscope cannot be manipulated through and beyond the stricture and into the stomach, the strictured lumen of the esophagus is dilated with flexible bougies until the mechanical dilator can be introduced, that is, up to 20 French. With the dilator in place the stricture is stretched. No rule can be given as to the safe amount of force to use with the dilator. It should be remembered in dealing with strictures if a stricture can be dilatated to the size of a 30 French bougie, that is stretched to a diameter of 10 mm., this amount of lumen is sufficient for normal swallowing. In the cases of slight gluing of the esophagus at the twist the passage of the large tube is sufficient.

Rarely will a case be encountered in which no instrument can be made to pass through the stricture. These naturally are trying. The temp-

tation is to use smaller and smaller instruments and more rigid instruments. Small metal probes or small olives in series on the end of a fine metal shaft are manufactured for this purpose. If used at all they should be used with the greatest care. They are liable to turn the emergency into a tragedy. Do not needle an apparently impassable stricture. Let the patient come out of ether and give him a silk thread to swallow. If this finds its way through the stricture, as it will in most cases, then the perforated olive with its flexible shaft should be tried threaded on the string. This failing there is nothing left to do if the patient is actually starving except to perform a gastrostomy, and later when the patient has been brought back to full nourishment to try the thread or the esophagoscope again.

**The String and Water Bag Method.**—Some surgeons use the string and water bag method for the treatment of cases of stricture of the lower end of the esophagus, and are very successful with it. In the past the writer used the water bag dilator somewhat but gave it up for dilatation through the esophagoscope by sight. As he understands the method, the patient first swallows a thread and then on this olives are passed until the stricture allows the water bag to be introduced. Then dilatation is continued by filling the bag with water until the water gauge registers a certain pressure. This method presupposes what might be called a soft and easily dilatable stricture like many which are encountered in the urethra. In these cases, as has been said, it is highly successful. The writer has not used this method enough to speak with authority about it. Plummer, of the Mayo Clinic, is its great exponent.

**Esophageal Tragedies.**—Every time an instrument is passed into the esophagus there is a tragedy in the offing. It is not necessary to actually perforate the esophagus to cause a tragedy, a slit in the mucous membrane, given the right bacteria in the neighborhood, is sufficient. A perforation of the esophagus is practically always fatal, a break in the mucous membrane may be. The safest way to pass a bougie is by sight and through a tube. Only after a stricture has been examined by sight is it justifiable to pass a bougie by touch. After the size of the lumen has been ascertained and as much about the character of the stricture as is possible to learn has been made out then I do not hesitate to pass a bougie by touch. Yet even when fortified by this knowledge I never feel entirely safe. It is a good rule to pass without ether a bougie a few sizes smaller than the one which would pass under an anesthetic. The esophagus is thrown into spasm by the bougie. The point of the bougie may pass but the belly of the instrument may burst the mucous membrane. It is heartbreaking to have this happen as it did in one case of the writer's after the patient had passed through an ether examination and dilation without the slightest sign of trouble.

When severe infection of the chest results after a slit in the mucous membrane or an actual perforation the patient at once or within forty-eight hours becomes gravely ill and dies within a week. The first signs are a stabbing pain from the sternum to the back on every breath, accompanied by a rising temperature. The chest fills with fluid, usually one side only, and there may be signs of peritonitis. At autopsy a large amount of thin greenish-yellow pus is found in the chest. The esophagus at the site of the perforation is gangrenous. The adjoining portion of the lung is in the same condition.

After the original dilatation under ether the bougie can be passed under

the fluoroscope. This will show whether or not the bougie has entered the stomach, but it will not tell what the bougie has done on the way down. Given a case of cardiospasm which dilated under ether only to 22 or 24 and with a marked bowing to the right of the lower end of the esophagus, it is better to pass the bougie through the esophagoscope introduced under a local anesthetic or if necessary to re-etherize.

Up to two years ago the writer felt that stricture rather than spasm is the more frequent cause of cardiospasm. Two things have happened in the past two years which have changed his views and modified his method of treatment. The English laryngologists have had the good fortune to accumulate a series of autopsy specimens from cases of cardiospasm, and have described their microscopic and histologic findings. The writer has introduced the diagnostic barium bougie and recorded his findings with it, and has developed a method of dilating the terminal portion of the esophagus under fluoroscopic control, in other words, dilatation by sight.

The autopsy specimens just referred to show that the terminal portion of the esophagus has a short tubular narrowing or a fibrous ring-like stricture. Above the narrowed portion the esophagus was much dilated and its walls in some instances were thickened, in others thinned. Ulcerations of the mucous membrane were not uncommon. The tubular narrowing apparently occurred between the crura. Stokes made the observation on a number of these specimens that the ganglion cells of Auerbach's plexus were diminished or wanting, especially in the terminal portion of the esophagus. Naturally this finding was eagerly seized upon to explain cardiospasm. It was held that as the result of infection Auerbach's plexus was destroyed and the motor nerve supply of the lower half of the esophagus abolished.

I have never been able to satisfy myself that there was spasm of the crura in my cases of cardiospasm. Yet in the autopsy specimens figured by Moore and Kelley the narrowing at the terminal portion of the esophagus lies between the crura. In a recent autopsy specimen of mine from a baby ten months old who had had difficulty in swallowing from birth, the narrowed portion of the esophagus was accurately confined to the space enclosed by the crura. In this case both crura seemed to my eye to be much thickened. A small esophagoscope was once passed on this infant with restoration of normal swallowing until its diet was changed from milk to milk and cereals. The crura, therefore, are now coming into the picture with me.

My present views as to the cause and nature of cardiospasm can be summarized as follows: The liver tunnel acts as a mold for the terminal portion of the esophagus. The cone of the diaphragm which lines the liver tunnel is filled with loose connective tissue. This surrounds the esophagus. Within the abdomen the lesser omentum is continued upward as a substantial layer of connective tissue over the peritoneal surfaces of the two crura and over the esophagus as it escapes from the crura. Infection of the double connective-tissue investment of the crura and of the esophagus can originate in the thorax or in the abdomen and be transferred to the esophagus. As the result of infection bands and adhesions may form. These can constrict the esophagus or distort it by traction. Both the lung-tips and the crura, especially the left lung tip and the left crus, play a part in making the deep notch which occurs on the left side of the esophagus when the diaphragm descends. In producing the momentary closure of the

esophagus which accompanies the descent of the diaphragm, I feel that the lung-tips are a greater cause than the crura. The typical finding with the esophagoscope in cases of cardiospasm, namely, a small round opening which is either central or a little to the left is located at the beginning of the pleated segment of the esophagus. In the resting condition the pleated segment is normally closed by vertical folds, and this closure is reinforced by the natural twist of the esophagus from right to left. The usual stricture in cardiospasm consists of a light gluing of the vertical folds. In long-standing cases a marked backward bend of the terminal portion of the esophagus occurs. An instance of this bend has been found by the writer even at birth. The bend acts as a trap. When the diaphragm becomes ptotic and nearly motionless, the cone of the diaphragm flattens and the lower end of the esophagus, lacking the support of the cone, bends and twists freely. With the destruction of the motor nerve cells (Auerbach's plexus) by infection, the muscles of the esophagus lose their power to contract, peristalsis is progressively lost and the walls of the esophagus after a preliminary period of hypertrophy become thin, and the whole of the esophagus with the exception of the terminal portion, gradually dilates.

**The Diagnostic Barium Bougie.**—In order to estimate the ability of an elastic tube like the esophagus to function as a carrier of fluid and food, it is necessary to know its maximum diameter in at least two directions, preferably the transverse and the anteroposterior. The ordinary elastic bougie will give this information, but only at one point, not over an extended area. The barium bougie, on the other hand, can be used to map out the esophagus from the stomach to the clavicle.

The barium bougie is an elaboration of an old idea, namely, the introduction into the esophagus of a rubber balloon filled with barium and recording by means of the Roentgen-ray plate the shape which it assumes. The end of the bougie has a blunt metal cap about 1 cm. wide, so that it will pass only a relatively large stricture. I have been pleasantly surprised to find how well the patients tolerate it. It has been passed successfully in 15 consecutive cases. The bougie is introduced by touch until it is beyond the cricoid and approximately at the arch of the aorta. From this point on its passage is guided by means of the fluoroscope.

Only in the first of my last series of 15 cases of cardiospasm have I passed the esophagoscope for treatment. In this case the first dilatation was done through the esophagoscope, but the subsequent dilatations were done with the Sippey bag. The barium bougie has given me a sureness of diagnosis which I had not had before. Dilatation by sight with the Sippey bag carries with it a sense of safety which I have never had with the elastic bougie or with the mechanical dilator used through the esophagoscope. I have not discarded my faithful friend of many years, the large oval esophagoscope. In my very next case I may not be able to pass the diagnostic barium bougie or the Sippey bag, and I shall, of course, go back to it. Where there is the slightest suspicion that the obstruction at the terminal portion of the esophagus might be due to cancer, the esophagoscope still holds first place.

Two years ago I began the use of the diagnostic bougie. It has given me a visualization of the lower end of the esophagus which I have never had before. From it I have gotten information which neither the Roentgen ray nor the esophagoscope has furnished, and it has made me doubt some of my

observations, made with the esophagoscope, at the lower end of the esophagoscope in years past. I have always been partial to large examining tubes. If the large tube will lead the observer astray, a small tube is a much greater sinner in this respect. The matter may be summed up as follows: It is difficult to be sure of what you see at the end of a 21-inch tube, large or small, and it is equally hard not to see what your mind has been made up to see. One observer sees spasm of the crura, another spasm of the cardiac sphincter, and another always sees stricture. I have been in the latter class.

In using the diagnostic bougie, the first thing was to establish the normal dimensions of the lower end of the esophagus in its various diameters. This was accomplished by passing the bag on "Bill," the Ward Tender of the Private Ward of the Infirmary. He was the only volunteer and will live long in my grateful memory.

At first the barium bag was used for diagnostic purposes only. It was soon found, however, that even the pressure exerted by the thin bag was sufficient to cause some dilatation. Then the Sippey bag, as it was stronger, with barium and cement lines like the diagnostic bougie was used both for purposes of diagnosis and dilatation. Owing to its silk cover, it is possible to exert a pressure of 10 to 15 pounds. So far I have not gone beyond 6 pounds.

For many years I have had a growing conviction that the obstruction in the terminal portion of the esophagus, in cases of cardiospasm, is a slight affair. I have repeatedly likened it to a soft stricture, for it readily gives way to the continued pressure of the large examining tube. In the majority of my cases I have found a small central opening at the lower end of the esophagus which would not open on ballooning but which did open after pressing the end of the esophagoscope against it for a minute or two. In some of these cases there seemed to be a small crescentic web as well, generally on the right.

**Findings with the Barium Bougie.**—In cases of cardiospasm the usual barium picture shows that the esophagus ends in an awl-like point. This is either on a level with the dome of the left half of the diaphragm or a little below it. The point is continued for an inch and a half as a thin ribbon of barium and then broadens out along the lesser curvature and the anterior wall of the stomach. The same awl-like point is seen through the fluoroscope, but at each inspiration as the lung-tips fill, half an inch or more of the point is amputated and the end of the esophagus becomes blunt and round. This happens regularly and is very marked on forced inspiration. The impression which one gets is that the esophagus is stric-tured to a small caliber. On passing the diagnostic barium bougie and inflating it, the picture changes. The apparent stricture disappears and a waist-like narrowing takes its place. The narrowing is usually greater on the left than on the right. In some cases the notch on the left of the esophagus is above the level of the diaphragm, in other cases below. When the notch on the left is large and above the level of the diaphragm I have held that it was due to the inflated left-lung tip, when below the dome of the diaphragm I have felt obliged to give the left crus the credit for producing it. The diameter of the waist-like narrowing is generally about 19 to 20 mm. With the Sippey bag with its barium lines under a pressure of 3 to 4 pounds on the second or third introduction the esophagus will be seen

to dilate to normal caliber 31 (mm.). The dilatation is followed by the disappearance of the difficulty in swallowing for weeks or months.

In my series of 15 recent cases of cardiospasm this waist-like narrowing was found in all except one. In this there was an annular stricture. With the exception of this stricture, and two of the waist-like narrowings, dilatation to normal was quickly accomplished. My present conception of this waist-like narrowing is that it is due to a light fibrosis of the connective tissue of the esophagus or of the connective tissue packed about the esophagus. I feel that the constant hammering of the lung-tips play a part in determining its form.

Even after dilatation to normal has been accomplished and the difficulty in swallowing has disappeared, when the patient returns, the barium picture is much the same as at first. There is usually fluid in the esophagus but at a lower level. There is the same awl-like point, although it is wider. The barium, however, flows into the stomach freely. It is evident that some of the old condition, which existed before the dilatation, has remained or

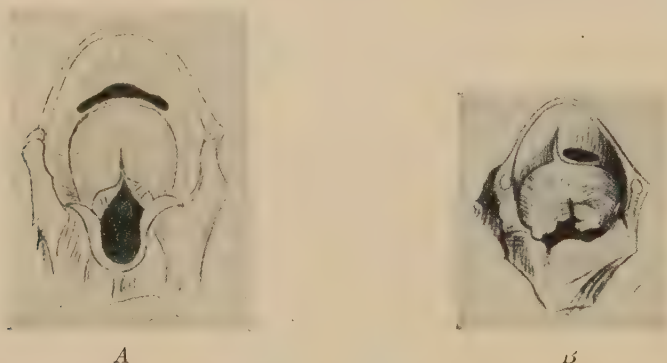


Fig. 598.—A, Specimen of a larynx and the upper end of the esophagus showing the size and shape of the opening at the beginning of the esophagus. Reduced one-half. B, Web of the esophagus. Specimen of a larynx and the upper end of the esophagus showing a single web springing from the upper rim of the posterior surface of the cricoid cartilage a little to the right of the median line. The esophageal opening is small and is found to the left of the web. The lumen of the right half of the beginning of the esophagus is obliterated. Reduced one-half.

returned. The waist-like narrowing is too large to account for all of the obstruction. I have come to the conclusion that the return of the closure or the remaining obstruction is best explained by a twist or a backward bend of the terminal portion of the esophagus.

It has long been known that in cases of cardiospasm the esophagus when filled with food or barium—generally to the level of the arch of the aorta—will bougie itself; another proof that the obstruction at the terminal portion is relatively slight. When these cases first present themselves for examination the esophagus is found to be full of fluid. At times the gas bubble of the stomach will bougie the esophagus from below and open it. It is a common history to get from a patient that when he eats he will feel the fluid or food stick at the lower part of the esophagus. He then does the natural thing, takes a drink of water, and at once gulps up gas, and this is followed by normal swallowing for the rest of the meal.

Recently I hit upon the use of a fizzing Seidlitz powder as a means of emptying the barium filled esophagus. This procedure has been of great help.

**Treatment of Cardiospasm.**—Mild cases of cardiospasm can be managed by dilatation, preferably dilatation by sight. Where this is unsuccessful or the case has advanced to the stage marked by extreme bend or twist, the condition must be dealt with by some form of surgery carried out through the lower throat or the upper abdomen. Incision into the musculature of the lower end of the esophagus, after the fashion of the treatment of pyloric obstruction at birth, has been tried successfully in this country in a few cases (Meyer). In England, Grey Turner has twice made a lateral anastomosis between the mobilized lower end of the esophagus and the fundus of the stomach. I feel that some such operation is the coming treatment for long-standing cases, especially those with twist and backward bend.

**Postericoid Webs and Strictures.**—Clinical and dissecting room findings show that postericoid webs are very common. They are found anywhere along the posterior surface of the cricoid cartilage. Those which occur at the top of the cartilage may be unilateral or bilateral. Unilateral



Fig. 599.—Webs of the esophagus. Specimen of a larynx and the upper end of the esophagus showing two webs, one on either side of the median line. They are symmetrically placed and each one springs from the upper rim of the posterior surface of the cricoid. The opening of the esophagus is small. Reduced one-half.



Fig. 600.—Drawings showing three views of a postericoid web. The upper drawing shows the web as it was first seen. The middle and lower drawings show the web as it was put on the stretch by strong ballooning. On the left the round knob is the remnant of a web which was broken by the passage of a bougie sixteen years before. The passage of the bougie relieved the patient for a few years. For three or four years before the patient was seen by the writer she was reduced to fluid diet. She complained of frequent attacks of strangling. The web on the right was bitten away. Bougies were passed a few times. The patient regained normal swallowing. Natural size.

webs are more common on the right. Diaphragm-like webs are found usually at the bottom of the cartilage. Akin to the web but an exaggeration of it is a gluing of one half of the mouth of the esophagus or asymmetry of the esophagus. This, like the unilateral web, is more common on the right. In some cases one half of the esophagus is glued to the posterior surface of the cricoid for the whole length of the cartilage.

**Cause of Webs.**—The fact that the mucous membrane on the back of the cricoid cartilage is very redundant and is normally thrown into numerous

folds must play a part in the formation of webs. A small examining tube held against the loose wall is liable to push a fold of the mucous membrane ahead of it and produce an artificial web. Webs are best discovered by using large tubes, as small tubes either pass them or do not put them on the stretch. Webs are produced by ulceration or abrasion of opposite areas of the esophageal membrane. The mucous membrane lesions may be caused by disease or trauma. Caustics also play a prominent part in their formation. Bilateral webs springing from the upper lateral edges of the cricoid cartilage are often so symmetrical they give the impression that they are a growth anomaly and not due to ulceration of the mucous membrane.

*Symptoms.*—A web springing from the upper lateral edge of the cricoid cartilage and connected with the posterior wall of the mouth of the esophagus turns the pyriform sinus of that side into a sizable pocket. Such a pocket will easily take the tip of the index-finger, and will hold a very considerable amount of fluid or food. These web-formed pockets are met with so commonly in the dissecting room that it is probable that most of them give no trouble during life. When they do cause symptoms in the living, and the writer has had many such cases, the patient feels that something sticks in the throat and he is subject to frequent attacks of spasm of the larynx during meals. Sometimes the patient will cough up small bits of food some hours after a meal. The coughing and strangling often so terrify the patient that he reduces his diet to liquids and soft solids. When the web occurs at the lower edge of the cricoid cartilage and for all intents and purposes is a thin annular stricture with a small central opening, this being the usual formation of webs in this location, the patient has more pronounced symptoms of obstruction there is the same coughing and strangling, these symptoms of course being due to food getting into the larynx. One of the writer's cases was completely ostracized from the table. Many cases of globus hystericus are really cases of postcricoid webs.

*Fluoroscopic and Roentgen-ray Findings.*—In webs the fluoroscope shows little or nothing, the process of swallowing is so rapid. If there is a stricture at the bottom of the cricoid with a small central lumen, both in the lateral and the front position, the stream of barium will be seen to be narrowed. If a film is taken of the larynx in the lateral position and a web is present, a shadow of the pocket made by it will appear. A web will not appear in the front position. The film, of course, will give the narrowing of the barium stream caused by a lower web in both lateral and front positions.

Unless the fluoroscopic examination is centered on the larynx or the film is taken of the larynx nothing will be found. It took the writer a number of years to find this out.

*Treatment.*—The treatment of a web is to expose it by a short full-sized oval esophagoscope and to remove it with a punch. Thin webs at the bottom of the cricoid with the characteristic central lumen are easily divulsed with bougies or by the mechanical dilator. The cutting of a web or the divulsion of a stricture is usually followed by a short after-treatment with bougies. The treatment of these webs and strictures gives immediate and most gratifying results.

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## RETROPHARYNGEAL DIVERTICULUM

## (Esophageal Pouch)

There are two types of esophageal pouches. The first or commoner type is due to a hernia of the esophageal mucous membrane through the lower part of the inferior constrictor muscle or through the weak triangle of the posterior esophageal wall opposite the cricoid cartilage. The second type is caused by the contraction of adherent scar tissue, and is usually found immediately below the bifurcation of the trachea. Turner has shown from autopsy records that practically all of these are of tubercular origin. Their formation is due to an adjacent tubercular gland which at some time suppurated and then healed. In the healing the contracting scar tissue being attached to the esophageal wall pulled a part of the wall with it. The pouches which are caused by the contraction of attached scar tissue are called fittingly traction pouches.

The two types of pouches just mentioned will be discussed separately, and the first or commoner type is discussed first.

## THE FIRST TYPE OF ESOPHAGEAL POUCH

**Location.**—Pharyngo-esophageal diverticula are the pulsion diverticula of Zenker. They are located on the posterior or posterolateral wall of the pharynx, just above its junction with the esophagus. Arrowsmith has

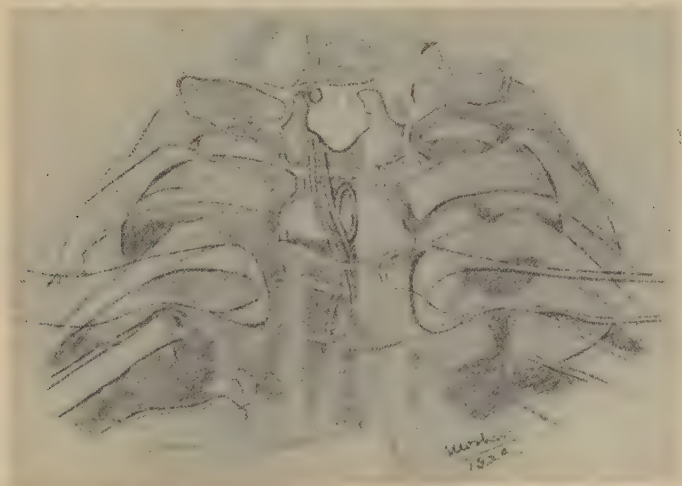


Fig. 601.—Retouched tracing from a Roentgen-ray film of a small esophageal pouch.

reported a double pouch. The body of the diverticulum occupies the prevertebral space behind and usually to the left of the esophagus between the layers of prevertebral and pretracheal fascia.

**Cause.**—As was said above, the cause of this type of pouch is a hernia of the esophageal mucous membrane through the lower part of the inferior constrictor or through the weak triangle of the posterior wall of the esophagus back of the cricoid cartilage, in other words, at the very beginning of the esophagus. The weak triangle has been described in the section on anatomy. It is weak because the esophageal wall lacks one of its two layers at this point, namely, the layer made by the longitudinal fibers.

Diverticula have never been found at birth. What starts the hernia, therefore, has been an unanswered question. A possible answer is as follows: It has been found that the mouth of the esophagus is frequently asymmetrical, due to the fact that one half of its lumen is obliterated. Since this was established at least one esophageal pouch of the beginning of the esophagus has been proved to be associated with the asymmetry of the esophageal mouth. If the mouth of the esophagus is only half its normal

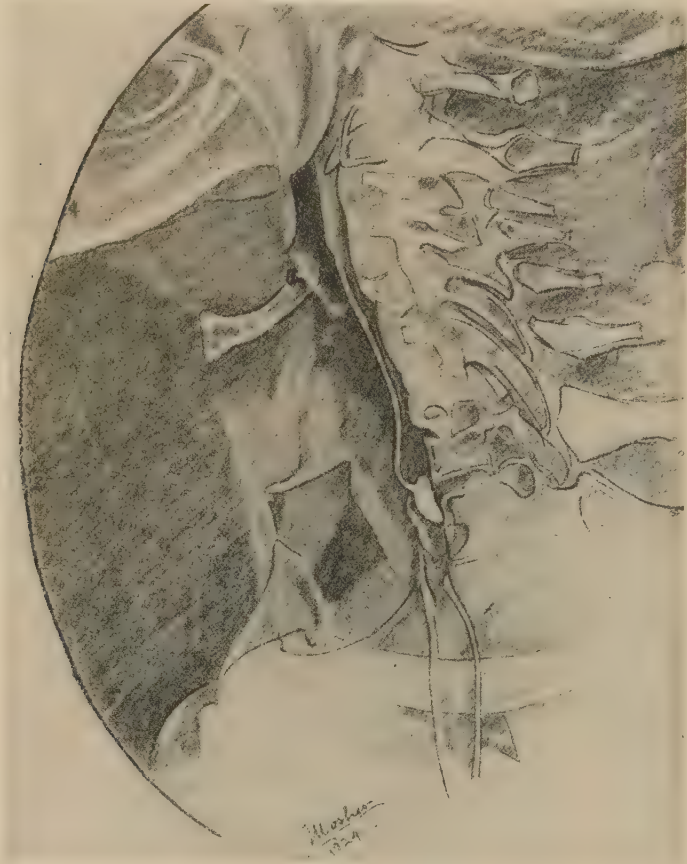


Fig. 602.—Retouched Roentgen-ray tracing. This film is the lateral view of the small pouch shown in Fig. 601. The upper limit of the pouch is at the bottom of the body of the seventh cervical vertebra. The opening of the esophagus is at the top of the pouch. Notice how the esophagus widens as soon as it escapes from between the pouch behind and the cricoid cartilage in front. This postcricoid narrowing of the esophagus is characteristic of pouch cases. Notice the laxity of the pharyngeal wall just above the pouch. Reduced one-half.

width more strain is thrown upon it in swallowing and this strain is off center. The cricoid cartilage acts as a cartilaginous stopper at the entrance of the esophagus. At times the upper half of the posterior surface of the cartilage wears an actual notch in the front face of the body of the fifth or sixth cervical vertebra and rests in it. This would increase the stopper-like action of the cartilage during the act of swallowing.

**Age.**—Patients who have diverticula are mostly of middle age, around forty, although diverticula are occasionally found in young adults and even

in the aged. The writer has knowledge of a museum specimen of a pouch in a boy who died at the age of fourteen.

**Symptoms.**—The chief symptom of an esophageal pouch is obstruction in swallowing. If the pouch is large or the mouth of the esophagus is not only narrowed but unyielding the patient lives on liquids and subsists in a half-starved condition. The patient lives on the overflow from the pouch. When he takes food he is never sure of retaining it. Consequently he is barred from the table. The food which is regurgitated shows rem-



Fig. 603.—Roentgen-ray tracing of an esophageal pouch. Notice the level at which the esophagus begins. At first glance it looks as if it began in the middle of the anterior wall of the pouch. After studying this point on wax casts and on wet specimens of the larynx and lower pharynx, it became clear that the opening of the esophagus was on a level with the top of the cricoid cartilage, and that the ascending, slightly curved oblique line above the opening of the esophagus was the outline of the pyriform sinus. The posterior rim of the mouth of the pouch is much higher than the level of the anterior rim; that is, it is roughly on a level with the top of the arytenoids. In this instance, owing to the higher level of the posterior rim of the mouth of the pouch, the pouch tends to stay wide open. Notice the difference in the anteroposterior width of the mouth of the pouch and the mouth of the esophagus. The esophagus is reduced to a narrow ribbon until it passes the bottom of the pouch. The esophagus is squeezed between the pouch and the cricoid cartilage. As soon as these two structures release it, it increases in size. As the mouth of the esophagus begins at the bottom of the cricoid cartilage and, as the level of the weak triangle is at this level also, the pouch in this instance is a hernia through the lower fibers of the inferior constrictor. Reduced one-half.

nants of articles eaten hours or days before. The patient cannot lie down to sleep because fluid escapes into his mouth or runs over into the larynx. At times air escapes from the pouch and gives a distressing gurgling sound in the neck. In one severe case the patient for two years had been compelled to take his food lying on his chest. He could not take even a glass of water at the table. The symptoms just given are those of a large and long-standing pouch. Small pouches often give only a moderate amount of trouble with swallowing accompanied from time to time by the return of undigested and putrid food.

**Types of Pouches.**—The Roentgen ray shows that there are two types of pouches of the upper end of the esophagus, the small pouch with a wide open mouth and the large pouch which is often pedunculated and in which the mouth is narrow. The difference in the width of the two types of pouch is only in the anteroposterior diameter of the mouth of the pouch. The transverse diameter takes the whole width of the transverse diameter of the lower pharynx in both types. The large pouch may, of course, be only a later stage of the small one. Large pouches gradually work their way down the neck and in time may reach the level of the clavicle or even invade the chest as far as the arch of the aorta.

**Fluoroscopic and Roentgen-ray Findings.**—Both the fluoroscope and the Roentgen-ray film give the size and the position of the pouch. The film should include the larynx, otherwise the level of the mouth of the pouch will not be recorded. The mouth of the pouch may be at either the top or the bottom of the cricoid cartilage. With the patient in the lateral position the fluoroscope shows that the pouch fills with barium and then overflows into the esophagus. The beginning of the esophagus is squeezed

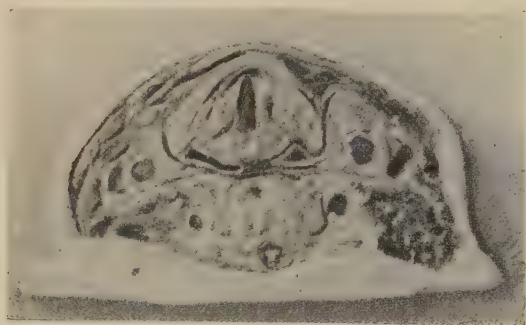


Fig. 604.—Cast from a frozen section—adult. The larynx is symmetrical. The right half of the mouth of the esophagus is obliterated. On the right there is a small triangular depression showing an attempt to start the mouth of the esophagus at this point. Reduced one-half.

between the pouch behind and the cricoid cartilage in front and the barium runs down in a narrow stream until the bottom of the cricoid cartilage is reached, at which point the esophagus at once becomes wider. When the patient swallows and the larynx ascends the pouch makes a similar upward excursion of about an inch. When the patient is examined the fluoroscope shows the position of the stream of barium in the esophagus as it comes to view below the bottom of the pouch. It may appear below the pouch in the midline or on one side. If it is on the side the mouth of the esophagus is asymmetrical.

**The Examination with the Esophagoscope.**—The writer uses a 9-inch full-size (20 by 10 mm.) oval esophagoscope, and examines the patient routinely under ether. The esophagoscope enters the wide-open mouth of the pouch without difficulty and is carried easily to the bottom of it. The pouch may be smooth throughout or there may be many trabeculae at the blind end. At times the pouch shows a low-grade inflammation. Often it is possible with the forceps to turn the smooth walled pouches inside out. The writer has done this a number of times and the procedure seemed an innocent one, but the last time he did this the patient on coming

out of ether complained of a stabbing pain in her right side and immediately developed an empyema which was considered probably embolic and due to the examination. The pouch therefore is easy to find and to explore. The opening of the esophagus, on the contrary, is often very difficult to see. In order to locate it the end of the esophagoscope must be tipped strongly upward. The ballooning attachment of the esophagoscope is invaluable at this point, especially if the mouth of the esophagus is strongly contracted. Inflation gives a dimple, sometimes even in the hard cases an actual lumen, at the point where the esophagus begins. The esophagoscope shows further whether the mouth of the esophagus is in the middle line or on one side. After the lumen of the esophagus is found its size and dilatability are tested with bougies. These two factors determine the subsequent treatment.

**Treatment.**—If the pouch is shown to be small by Roentgen ray, and the examination with the esophagoscope shows that the mouth of the esophagus dilates readily the patient can be made comfortable and even a clinical cure obtained by the periodical passage of bougies first by the physician himself and later by the patient. The writer has seen a few pouches remain stationary for ten years. One such patient gave up the



Fig. 605.—Cast showing the under surface of section No. 3. The esophagus is only half its normal width, the right half being obliterated. Reduced one-half.

passing of bougies after three or four years and is still a clinical cure. The bougie treatment was the first method employed in the treatment of pouches. It should not be used until an examination with the esophagoscope shows that it is justifiable. In the old days both suitable and unsuitable cases were treated in this way and all sorts of flexible finders were attached to the end of the bougies.

Some seven years ago the writer began slitting a portion of the common wall between the pouch and the esophagus. This was done with an appropriate scissor punch through the esophagoscope. The aim was to cut only a small part of the common wall, that is the first third. Experience showed that the procedure gave immediate relief of the difficulty in swallowing and the follow-up on the cases so operated, which was continued for some four or five years, proved that the clinical cure was permanent. The fluoroscopic examination of these operated patients showed that the remnant of the pouch emptied itself readily and the patients confirmed this by their statements that they had had no return of the trouble with swallowing. Six cases were successfully operated. The seventh developed a mediastinitis and died.

*The External Method.*—The usual operation for the cure of an esophageal pouch is resection through an incision in the side of the neck. This is suitable for all except the very small pouches or the large ones which have gravitated into the thorax. The operation has the same danger as slitting the common wall, namely, opening the posterior mediastinum and sub—is performed and suture of the esophagus is depended upon alone to prevent sequent infection from the contents of the esophagus. The one-stage operation has a high mortality—20 per cent.

In resecting a pouch the surgeon is greatly helped if an esophagoscope is introduced into the pouch and the pouch made to present in the side of

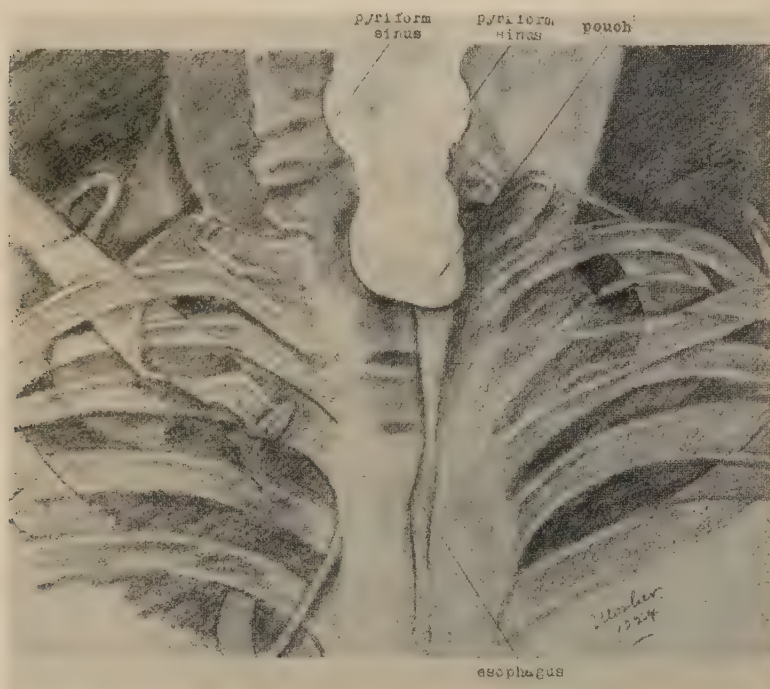


Fig. 606.—Roentgen-ray tracing of an esophageal pouch associated with asymmetry of the mouth of the esophagus. Notice that the esophagus runs down on the left side of the pouch. On examining the patient under ether it was found that the right half of the mouth of the esophagus was obliterated. Compare with Figs. 604, 605. Patient seen through the courtesy of Dr. C. E. Cooper, of Denver. Roentgen-ray plate reproduced through the courtesy of Dr. L. G. Crosby, of Denver. Reduced one-half.

the neck and under the surgeon's finger. This method was first introduced by Jackson. The writer considers the one-stage operation unjustifiable.

A few years ago Imperatori introduced the procedure of inverting the pouch, twisting it, and tying and cauterizing the pedicle. The operation relieved the difficulty in swallowing, but its safety has not yet been proved.

The surgical treatment of esophageal pouches began in 1830 by the establishment of a fistula from the pouch to the surface. In 1884 the first excision was done. Twelve years later invagination of the sac and suture of the esophageal walls were tried successfully. In 1917 Bevan elaborated this procedure and advocated operating under novocaine. "One-stage

operations, especially those of excision, had a high mortality because of leakage from the suture line, or about the ligature at the neck of the diverticulum, the esophagus being notably unreliable in its ability to heal tightly after suture" (Lahey). Leakage resulted in an infection of the posterior mediastinum. In 1909 Goldman introduced the two-stage operation. This operator freed the pouch, ligated the pedicle with silk, and fixed the sac to the surface of the wound. The sac sloughed off and the fistula which was left healed in two months. Murphy implanted the sac in the wound with its neck unligated. The second stage of his operation was taken two weeks later and consisted in resecting the sac in the granulating wound. Judd (1918) sutured the edges of the skin to the neck of the sac, closed the wound about it, and left the sac unopened upon the skin. Twelve days later, when healing had taken place in the wound and about the neck of the sac,

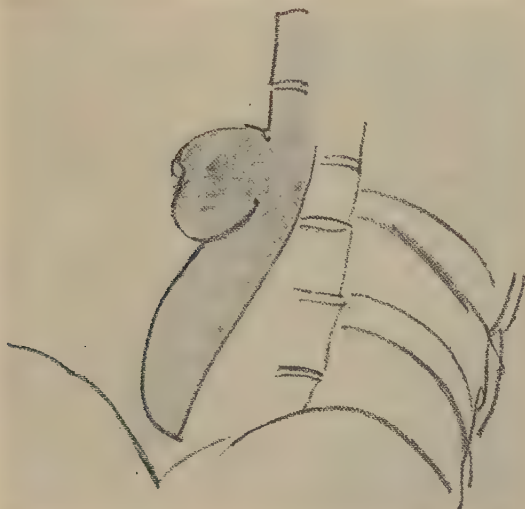


Fig. 607.—Tracing from a Roentgen-ray film of the esophagus of a man aged fifty years. The esophagus shows a pouch springing from the right side just below the bifurcation of the trachea. There was an annular stricture of the terminal portion of the esophagus. This was dilated under ether. The plate is put in at this point because it shows the cone of the diaphragm well.

the sac was cut away and its edges turned into the esophagus. Lahey reported a further modification of the operation in 1923. He twists the neck of the sac before implanting it in the skin wound. This prevents leakage when the sac is excised at the second operation. Closure of the resulting mucus-lined fistula connecting with the esophagus is accomplished by repeated cauterization of the fistula with crude carbolic acid. Care must be taken to prevent twisting of the esophagus itself into the neck of the sac and so narrowing the esophagus. Lahey<sup>1</sup> concludes his description of this operation as follows: "The use of the two-stage procedure, as has so often been the case in the recent progress of modern surgery, offers a method by which a hitherto extremely hazardous surgical procedure may be made very much more safe, eliminating as it does, when care is taken to prevent opening the sac during dissection, the danger of cellulitis of the neck and the almost universally fatal mediastinitis."

## THE SECOND TYPE OF ESOPHAGEAL POUCH

The second type of esophageal pouch is due to the contraction of adjoining scar tissue which is usually the product of a healed tuberculous gland. The majority of these pouches are found just below the bifurcation of the trachea. Those seen by the writer have been on the right side. Pouches of a similar nature are occasionally found at the lower end of the esophagus and just above the diaphragm. The writer has seen one pouch at this location. It was the size of a large orange and left the esophagus on the right. The patient came into the hospital starving and died after a forlorn gastrostomy. The pouches which are found in the region of the bifurcation of the trachea usually give no symptoms. Two such pouches in patients of the writer were associated with stricture of the terminal portion of the esophagus. One of these two cases was definitely tuberculous, the inference being that both pouch and stricture were of tuberculous origin.

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## CHRONIC ESOPHAGITIS, ACUTE ESOPHAGITIS. ULCERATION OF THE ESOPHAGUS. POSTCRICOIDAL ULCER OF THE ESOPHAGUS

**Chronic Esophagitis.**<sup>4</sup>—Chronic inflammation of the esophagus is more common than is generally supposed. The usual symptom is discomfort back of the sternum, and pain, dull, aching or sharp, extending through to the back. The esophagoscope is the only means of diagnosis. The most common cause is stenosis and retention of food. The esophagus normally empties itself promptly, and is very intolerant of retention. Lactic acid and other products of fermentation render the retained food additionally irritating. Treatment consists of dilatation of the subjacent stenosis and the administration of bismuth subnitrate, in small doses, dry on the tongue at frequent intervals.

**Acute Esophagitis.**—Acute inflammation of the esophagus occurs occasionally as the result of trauma, as by swallowed bones or other foreign bodies, but in most instances the inflammatory condition is the result of blind instrumentation, especially blind efforts with a bougie to push downward a foreign body that may or may not have been present. The prognosis in these cases is bad, and there is no treatment for instrumental perforations but rest of the esophagus. If the esophagitis is due to a foreign body, a prompt cure will follow esophagoscopy removal. Bismuth subnitrate dry on the tongue in small doses at frequent intervals is the best treatment for acute esophagitis without perforation.

**Acute Esophagitis from Lye Poisoning.**—The most frequent cause of acute esophagitis is the swallowing of corrosive acids or alkalis, especially household lye. These preparations are composed of sodium hydroxide in the hygroscopic form of soda ash. The cauterant action is almost instantaneous, as contact with a red hot iron would be. The poisonous nature of household lye is not realized because its container does not usually bear an adequate "scare" label.

Chemically neutralizing antidotes are vinegar or lemon juice with plenty

of water. Olive oil is also useful. These remedies, however, must be used immediately to be of any benefit. When seen by the physician the indications are to aspirate obstructive thick secretions and to see that the patient gets an abundance of water; swallowing is so painful that the patient, usually a child, will refuse water and will soon become dangerously dehydrated. Bismuth subnitrate, in small doses dry on the tongue, is the best remedy for the esophagitis. If later closure of the esophagus is imminent, the patient should be given a braided silk cord to swallow; this should be worn to keep the lumen patulous until treatment of the stenosis can be undertaken. The upper end of the string can be worn through the nose instead of the mouth to prevent its being bitten off. How soon the treatment can be undertaken will depend upon the condition of the esophagus as shown by esophagoscopy. The presence of ulceration or sloughing is no contraindication to the wearing of a string; but if the ulcer is located at the site of the stricture dilatation of the stricture will be fatal unless very slowly and carefully done with the esophagoscope under guidance of the eye.

**Ulceration of the Esophagus.**—Benign non-specific ulceration of the esophagus may result from infective trauma as with a tooth-brush bristle, a bone, or even hastily eaten bread crust or toast; the traumatizing agent may have passed on. Long sojourn of a foreign body is nearly always associated with ulceration at the site of lodgment. This or any other form of stenosis usually results in erosion or ulceration from stagnation of foods. Any of these may result in stricture. Lye burns are by far the most common cause of esophageal ulcer; this form of ulceration is usually deep, slow to heal, and if allowed to continue is usually followed by dense and extensive cicatricial stenosis. At the Bronchoscopic Clinic we have seen many cases of long ridge-like ulcers evidently due to trauma inflicted by blind passage of a bougie before admission, and such ulcers, if deep, heal slowly and may become chronic, especially in the lower end of the esophagus.

**Postcricoidal Ulcer of the Esophagus.**—This form of ulcer is located at the mouth of the esophagus. It is caused by pressure of the cricoid cartilage backward, pinching the esophageal and hypopharyngeal mucosa between two hard structures, the spine and the cricoid cartilage. The cricopharyngeal and inferior constrictor muscles do the pulling backward of the cricoid. This is a normal condition; it is only in cases of low vitality in wasting diseases such as typhoid fever that the pressure results in ulcer. It is called "decubitus ulcer" but the recumbent posture is not responsible. The weight of the cricoid cartilage is as nothing compared to the constant tonic muscular pull backward of the cricoid. Decubitus ulcer is often followed by cicatricial stricture. At the Bronchoscopic Clinic three brothers of one household were treated and cured of this posttyphoid type of stricture.

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### PEPTIC ULCER OF THE ESOPHAGUS

This disease is now known to be more common than was supposed in the pre-esophagoscopy days. It is located in the lower third of the thoracic esophagus. Notwithstanding the fact that it is above the hiatus esophagus it is flooded at times with gastric juice, which seems to be one of the chief etiologic perpetuating factors, as well as the chief cause of the pain which extends through to the back. Diseased tonsils may be a cause. Peptic ulcer leaves dense cicatrices which usually result in stricture. The esophagoscopy appearances are shown in Plate XIX. Sodium bicarbonate will relieve the pain. Esophagoscopy applications of silver nitrate twice weekly will cure the ulcer in some cases. Diseased tonsils or any other focus of infection should be removed. Bismuth subnitrate given dry on the tongue after food or drink is a useful adjunct. Rough foods should be avoided, and the diet should be carefully planned by a gastro-enterologist.

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### TUBERCULOUS ULCER OF THE ESOPHAGUS

Tuberculous disease of the esophagus may be encountered by the esophagoscopist in four forms: (1) compression stenosis; (2) periesophageal cicatrices affecting the lumen but not involving the wall; (3) erosion and superficial ulceration starting in the mucosa, which is rare; (4) through ulceration of a broken down tuberculous mediastinal gland; this is not so rare.

The superficial type usually heals if the general condition improves; and little if any scar is left. The type associated with glandular invasion usually leaves dense cicatrices, which may cause a diverticulum of the type usually spoken of as "traction," though the mechanism here is not always that of traction. The esophagoscopy treatment is directed to the prevention of secondary stenosis. General treatment is, of course, very important.

CHEVALIER JACKSON.

#### REFERENCE

1. Jackson, Chevalier: *Bronchoscopy and Esophagoscopy*, 2d ed., W. B. Saunders Company, Philadelphia, 1927. (Contains illustration, in color, of tuberculous ulcer of esophagus.)



## PLATE XIX

### ESOPHAGOSCOPIC APPEARANCES OF PEPTIC ULCER OF THE ESOPHAGUS.

1. Peptic ulcer in the acute stage. The lesion is flat; the edges are somewhat granular. There is a quite red zone surrounding the ulcer, about 1 or 2 millimeters back from the edge. Diagnostic esophagoscopy was done because of retrosternal pain. There was no dysphagia.

2. The same patient as in illustration No. 1, one year and two months later. The ulcer has deepened somewhat, and is bleeding freely. Patient had no dysphagia, and only slight odynphagia, but felt severe pain immediately after dinner.

3. Ulcer of the abdominal esophagus and stomach. The symptoms were of gastric ulcer, and with characteristic pain. Diagnostic esophagoscopy was done because the pain persisted after the gastric ulcer had apparently healed.

4. Girdling peptic ulcer covered with a membranous, yellowish exudate which could be peeled up at its edge, showing a flat bleeding surface. Pain came on after the evening meal every day but could be relieved by taking sodium carbonate.

5. The same patient as in No. 4, as seen three years later. The annular cicatrix is the result of ulceration which has healed. The ulcerative stage had probably lasted for more than a year and a half.

6. At the left of the field is seen a scar with a cicatricial roll at its upper margin that is almost keloid in form. At the lower margin is a small fungation that had the esophagosopic appearances of cancer. This fungation was removed esophagoscopically, and was found, on histologic examination, to be squamous-celled epithelioma. The history justified the inference that the scar had resulted from a peptic ulcer of the esophagus of many months duration, healed a few years prior to the patient's coming under our observation.

7. The triangular area at the right represents a peptic ulcer in the esophagus of a man previously supposed to have cancer. The ulcerated area was covered with a yellowish exudate. A specimen esophagoscopically removed from the edge of the ulcer showed gastric cells, though the ulcer was 6 millimeters above the diaphragm (evidently an island of gastric mucosa).

8. The same patient as in the preceding illustration, as seen two years later. The ulcer has begun to heal, the triangular zone has diminished in size, and is covered with small bright red granulations.

9. Four months later. The granulating area has still further diminished in size.

10. The ulcer is seen to be completely cicatrized. The scar is white, showing fine red streaks where small vessels are located. This was the appearance nearly five years after the ulcer was first seen esophagoscopically.

PLATE XIX





## SYPHILIS OF THE ESOPHAGUS

The esophagus is one of the less common locations for a syphilitic lesion, but such a lesion here is not so rare as was supposed in the pre-esophagoscopy days. Mucous plaques are rare, but ulcer from breaking down of a gumma is not rare. The esophagoscope is invaluable for diagnosis and the prevention of stenosis. If the diagnosis is late, esophagoscopy treatment of cicatricial stenosis will be of utmost importance.

The *diagnosis of ulcer of the esophagus* can be made in only one way, namely, by the esophagoscope. This instrument is, in fact, an esophageal speculum. The Roentgen-ray examination (which should always precede esophagoscopy) may or may not show the ulcer; it will show cicatricial narrowing of the lumen if any such post-ulcerative lesion exists. The esophagoscopic appearances to the experienced eye are often sufficient to distinguish between the different types of ulcer, but the corroboration of the systemic tests and in some cases biopsy to exclude malignancy are important adjuncts. In many cases at the Bronchoscopic Clinic the patient has come in the agony of despair from a previously made, erroneous, inferential diagnosis of cancer. These patients we have been able to send away happy after the cure of benign ulceration. These cases are important demonstrations of the fact that a diagnosis of esophageal disease ought not to be made without looking at the esophagus, and doing a biopsy when indicated.

The non-specific treatment of ulcer of the esophagus consists in restriction of the diet to liquids, not only to avoid the mechanical irritation of solids but to obviate the irritation always set up by retention of any food in the esophagus. If there is stenosis, its removal is fundamental to the cure of the ulcer. Argentic nitrate and other medicaments may be accurately applied through the esophagoscope. Bismuth subnitrate dry on the tongue in small doses at frequent intervals is the best local application. When swallowed alone (*i. e.*, without food or drink) it adheres to the ulcerated surface. In some instances gastrostomy may be necessary for stenosis; it has the additional advantage of putting the esophagus at rest. Post-ulcerative cicatricial stricture calls for esophagoscopy treatment (*q. v.*).

CHEVALIER JACKSON.

## REFERENCE

1. Jackson, Chevalier: *Peroral Endoscopy and Laryngeal Surgery*, Text-book, 1914. (This book is out of print, but is in most libraries. The French translation entitled *Endoscopie, Bronchoscopie, Esophagoscopie*, is still obtainable from the publisher, Gaston Doin, Paris.)

## ANGIONEUROTIC EDEMA OF THE ESOPHAGUS; URTICARIA OF THE ESOPHAGUS; SERUM DISEASE OF THE ESOPHAGUS; HERPES OF THE ESOPHAGUS.

These uncommon diseases are usually, but not always, associated with similar manifestations elsewhere in the same patient. They have certain affinities that may prevent differential diagnosis as among them, but there is no difficulty with the esophagoscope in differentiating them from other diseases of the esophagus. Manifestations elsewhere should settle any doubt. The esophagoscopic appearances are those of the same disease

occurring on other mucosal surfaces. Herpes is distinguished from the others by the occurrence of blisters, which rupture leaving a superficial erosion. Treatment is general rather than local, and is in many cases unsatisfactory. Recurrences have appeared in more than half the cases at the Bronchoscopic Clinic. Each attack, however, is of too short duration for the dysphagia to produce serious results. The patient may be for a time reduced to liquids because of difficulty in swallowing solids.

CHEVALIER JACKSON.

#### REFERENCE

1. Jackson, Chevalier: Angioneurotic Edema of the Esophagus; Urticaria of the Esophagus; Serum Disease of the Esophagus; Herpes of the Esophagus, Proceedings of the American Bronchoscopic Society, 1928.

### CICATRICAL STENOSIS OF THE ESOPHAGUS

**Definition.**—Narrowing of the esophageal lumen by scar tissue. It is also called fibrous stricture and non-malignant stricture.

**Etiology and Prophylaxis.**—Scars in the esophagus are usually the result of ulceration and retarded healing. The most common cause of ulceration is the escharotic action of sodium hydroxide. This powerful corrosive poison enters the household under the guise of lye, washing powders, cleansers, etc. Owing to the activities of the Committee on Lye Legislation of the Section of Laryngology of the American Medical Association, a Federal Law has been enacted providing for proper warning labels. Many states have followed with similar laws. It still remains to educate the laity to the danger of leaving these preparations within reach of children. Many of the preparations resemble sugar. Sometimes it is only the moistened dregs in the can that are swallowed. Household ammonia, "salts of tartar" (potassium carbonate), "washing soda" (sodium carbonate), corrosive sublimate, mineral acids, very hot liquids, trauma, syphilitic and tuberculous ulcerations, operations on the neck, scarlet fever, and diphtheria have been the causes in some cases that have come to the Bronchoscopic Clinic. Quite a number of cases have resulted from postericoidal ulceration of the hypopharyngeal wall in typhoid fever; in some of them the ulcer extended into the upper end of the esophagus. Three cases of this kind, in three brothers, happened in the same household. Peptic ulcer has been the cause in many of our cases of ulceration of the lower third of the esophagus. Foreign bodies do not cause stricture unless allowed to remain until ulceration has resulted. Blind attempts to push a foreign body down, if not fatal, are usually followed by cicatricial stenosis. Other forms of trauma such as stab wounds, and wounds from penetrating projectiles, are causes. Radium burns have been responsible for a few cases. Most cases of lye stricture are in children and are the result of accidents. A few cases occur in adults as a result of accident or attempted suicide.

**Pathology.**—Promptly healed trauma in the esophagus does not often result in stricture. But healing is rarely prompt in the esophagus; usually there is a long period of ulceration and frustrated repair, which builds up

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## PLATE XX

ESOPHAGOSCOPIC VIEWS FROM OIL-COLOR DRAWINGS FROM LIFE, BY CHEVALIER JACKSON.

1. Direct view of the larynx and laryngopharynx in the dorsally recumbent patient, the epiglottis and hyoid bone being lifted with the direct laryngoscope or the esophageal speculum. The spasmodically adducted vocal cords are partially hidden by the overhang of the spasmodically prominent ventricular bands. Posterior to this the aryepiglottic folds ending posteriorly in the arytenoid eminences are seen in apposition. The esophagoscope should be passed to the right of the median line into the right pyriform sinus, represented here by the right arm of the dark crescent. 2, The right pyriform sinus in the dorsally recumbent patient, the eminence at the upper left border, corresponds to the edge of the cricoid cartilage. 3, The cricopharyngeal constriction of the esophagus in the dorsally recumbent patient, the cricoid cartilage being lifted forward with the esophageal speculum. The lower (posterior) half of the lumen is closed by the fold corresponding to the orbicular fibers of the cricopharyngus which advances spasmodically from the posterior wall. (Compare 10.) This view is not obtained with an esophagoscope. 4, Passing through the right pyriform sinus with the esophagoscope; dorsally recumbent patient. The walls seem in tight apposition, and, at the edges of the slit-like lumen, bulge toward the observer. The direction of the axis of the slit varies, and in some instances it is like a rosette, depending on the degree of spasm. 5, Cervical esophagus. The lumen is not so patent during inspiration as lower down; and it closes completely during expiration. 6, Thoracic esophagus; dorsally recumbent patient. The ridge crossing above the lumen corresponds to the left bronchus. It is seldom so prominent as in this patient, but can always be found if searched for. 7, The normal esophagus at the hiatus. This is often mistaken for the cardia by esophagoscopists. It is more truly a sphincter than the cardia itself. In the author's opinion there is no truly sphincteric action at the cardia. It is the failure of this hiatal sphincter to open as in the normal deglutitory cycle that produces the syndrome called "cardiospasm" (*q. v.*). 8, View in the stomach with the open-tube gastroscope. The form of the folds varies continually. 9, Sarcoma of the posterior wall of the upper third of the esophagus in a woman of thirty-one years. Seen through the esophageal speculum, patient sitting. The lumen of the mouth of the esophagus, much encroached upon by the sarcomatous infiltration, is seen at the lower part of the circle. 10, Coin (half-dollar) wedged in the upper third of the esophagus of a boy aged fourteen years. Seen through the esophageal speculum, recumbent patient. Forceps are retracting the posterior lip of the esophageal "mouth" preparatory to removal. 11, Fungating squamous-celled epithelioma in a man of seventy-four years. Fungations are not always present, and are often pale and edematous. 12, Cicatricial stenosis of the esophagus due to the swallowing of lye in a boy of four years. Below the upper stricture is seen a second stricture. An ulcer surrounded by an inflammatory areola and the granulation tissue together illustrates the etiology of cicatricial tissue. The fan-shaped scar is really almost linear, but it is viewed in perspective. Patient was cured by esophagoscopic dilatation. 13, Angioma of the esophagus in a man of forty years. The patient had hemorrhoids and varicose veins of the legs. 14, Luetic ulcer of the esophagus 26 cm. from the upper teeth in a woman of thirty-eight years. Two scars from healed ulcerations are seen in perspective on the anterior wall. Branching vessels are seen in the livid areola of the ulcers. 15, Tuberculosis of the esophagus in a man of thirty-four years. 16, Leukoplakia of the esophagus near the hiatus in a man aged fifty-six years.

PLATE XX



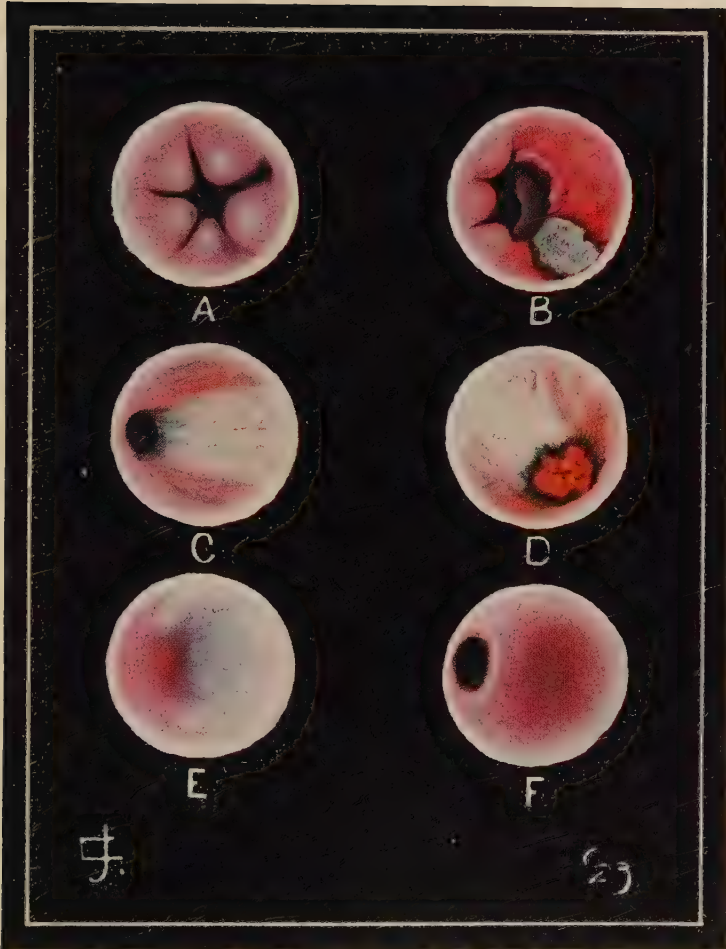
ESOPHAGOSCOPIC VIEWS. NORMAL.



ESOPHAGOSCOPIC VIEWS. ABNORMAL.



PLATE XXI



A, Shows an esophagoscopic view of the normal thoracic esophagus; the folds are soft, elastic, and readily displaced with the tube-mouth; the mucosa is pink and velvety. B, Sloughing, ulcerative esophagitis due to lye burn. C, Excentric stricture and scar following a lye burn. Permanent cure is obtained by dilatation of the segment of normal wall at the left by many months of careful dilatation. Attempts at rapid dilatation will split this normal wall, not the fibrous cicatrix. D, Orifice of strictured lumen obstructed by granulations, granulation atresia. E, Total, cicatricial atresia. F, Illustrates why patients, as Trousseau said, "sooner or later die of the bougie." The orifice of the strictured lumen is higher than the floor of the suprastrictural dilatation. (Photoprocess reproduction of chalk-talk sketch by Chevalier Jackson.)



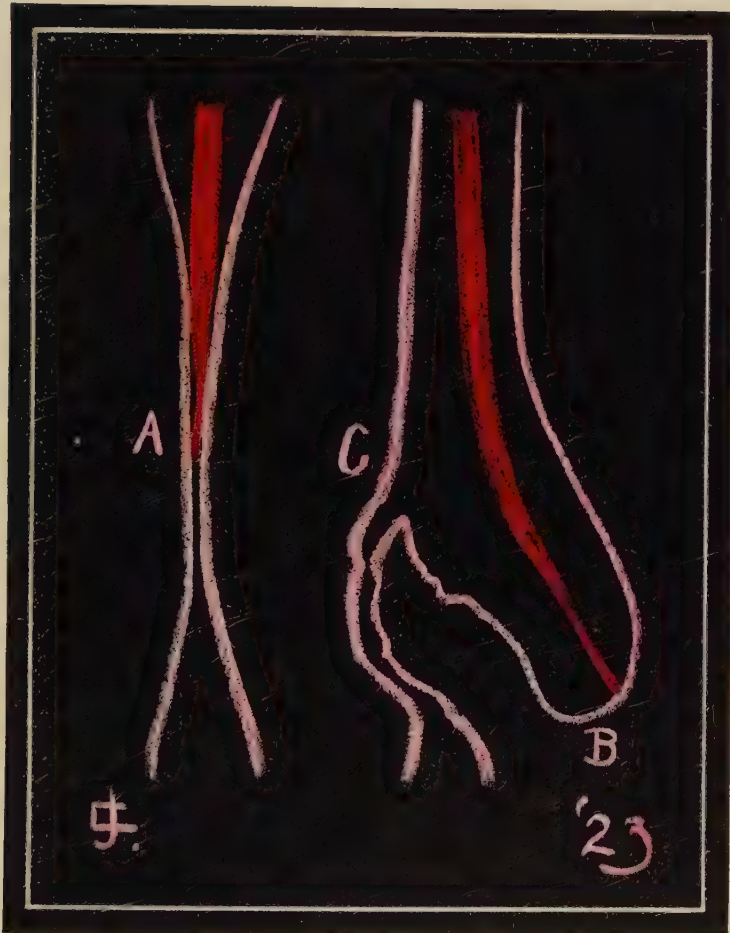


Illustration of the dangers of blind bouginage. The operator thinks his bougie is engaged in a stricture funnel-shaped lumen which will be dilated when he pushes upon the bougie. In reality, the tip of his bougie is not in the orifice of the stricture (C) but in a pocket (B), where a slight push will send it through with the mediastinum. (Photoprocess reproduction of a chalk-talk sketch by Chevalier Jackson.)



a fibrosis, the contraction of which later results in stricture. The density and extent of the fibrosis are usually in proportion to the duration and depth of the ulceration. Superficial erosions and chronic esophagitis may in time build up enough fibrous tissue to result in contraction and stricture. These may be secondary to stasis from any cause.<sup>1</sup> Even congenital narrowings (Adam Brown-Kelly) may thus be still further narrowed. Lye and other corrosives destroy tissue, which is replaced in the prolonged healing processes by fibrous tissue. The ulcer in typhoid fever may be the result of infective embolus, specific infection or pressure necrosis as that caused by the pressure of the cricoid against the spine under the pull of the cricopharyngeus. Although it is called decubitus ulcer, posture has probably nothing to do with it. *Peptic ulcer* is perpetuated if not caused by reflux of gastric juice through the *diaphragmatic pinchcock*.<sup>2</sup> It is a long, slow, stubborn form of ulceration resulting in some cases in large and dense accumulations of fibrous tissue.<sup>1</sup> Strictures due to lye burns are usually multiple. They may be annular; but they are perhaps more often eccentric, in which case only a part of the wall of the stricture is cicatricial, the other part being made up of more or less normal esophageal wall.

**Symptoms.**—The symptoms are those common to all obstructive diseases of the esophagus. Dysphagia, regurgitation, distress after eating, and loss of weight vary with the degree of the stenosis. The intermittency of the symptoms is sometimes confusing, for the lodgment of relatively large particles of food often simulates a spasmodic stenosis, and in fact there is often an element of spasm which holds the foreign body in the strictured area until it relaxes. Static esophagitis results in a swelling of the esophageal walls and a narrowing of the lumen, so that swallowing is more or less troublesome until the esophagitis subsides.

**Diagnosis.**—The diagnosis can be readily made by esophagoscopy preceded by Roentgen-ray examination. All other methods are inferential, dangerous, and obsolete.

**Esophagoscopic Appearances.**—The appearances vary according to the stage. Shortly after a burn, as by lye, the stenosis may be seen to be made up of edematous sloughs and ulcerated or bleeding areas, rather than cicatrices. Later the color of the cicatricial area is usually paler than the normal mucosa. The scars may be very white and elevated, or they may be flush with the normal mucosa, or even depressed. Occasionally the cicatrix is annular, but more often it is eccentric and involves only a part of the circumference of the wall. If the amount of scar tissue is small, the lumen maintains its mobility, opens and closes during respiration, cough, and vomituration. Between two strictures there is often a pouch containing food remnants. It is rarely possible to see the lumen of the second stricture, because it is usually eccentric to the first. Stagnation of food results in superjacent dilatation and esophagitis. Erosions and ulcerations which follow the stagnation esophagitis increase the cicatricial stenosis (Plates XX, XXI).

**Prognosis.**—All patients with a pervious esophagus, no matter how small the lumen of the stricture, are curable. If the methods herein described are used there is no mortality from treatment. All rapid and all blind methods are exceedingly dangerous. If there is an absolute atresia of only a small extent, say less than 3 mm., there is a chance of perforating this occlusion, but the procedure cannot be said to be free from risk. Once the

perforation is successfully made, the later treatment by the retrograde bougie is free from risk. To treat the esophagus without regular esophagoscopic inspection is to court disaster.

**Treatment.**—The first step in the treatment of these patients is to determine the exact conditions present in the particular case. If ulceration is present, it may be well to administer bismuth subnitrate, dry on the tongue, for a period before commencing treatment. If the stricture is of pinhole size and multiple, a silk thread should be swallowed as hereinafter mentioned, to prevent total atresia. If there is no danger of atresia and the strictures are only one or two in number esophagoscopic bouginage is indicated. The earlier the treatment is begun the better; that is, after the acute stage of the burn is over, say two or three weeks.

*Blind bouginage* is too dangerous to be justifiable. Splitting the esophagus, making a false passage, and other traumata are the inevitable and inevitably fatal results of the blindly passed bougie. Trousseau, in the pre-esophagoscopic days, said in effect that sooner or later all patients with esophageal stenosis died of the bougie.

*Gastrostomy.*—Patients with impending death from dehydration should be treated as acidotic and in most of them gastrostomy should be done without delay. Of this operation the author has said: "As with tracheotomy, we always preach doing gastrostomy early, but nearly always do it late." When necessary, gastrostomy has the added advantage that the retrograde bougie can be used. This method will cure every case in the shortest possible time consistent with perfect safety. In hundreds of patients we have never seen a death due to its use.

*Esophagoscopic bouginage* with the silk-woven steel-shank endoscopic bougies has proved, in our hands, the safest and most successful method of treatment when no gastrostomy has been done. With the esophagoscope the strictured lumen is centered in the esophagoscopic field, and three successively increasing sizes of bougies are used under direct vision. Larger and larger bougies are used at the successive treatments, which are given at intervals of four to seven days. No anesthesia, general or local, is used for esophagoscopic bouginage. The tightness of the grasping of the bougie by the stricture on withdrawal determines the limitation of sizes to be used. When the upper stricture has been dilated, the lower ones in the series are taken seriatim. If concentric, two or more closely situated strictures may be simultaneously dilated. For the use of bougies of the larger sizes the special esophagoscopes with both the light-carrier canal and the drainage canal outside the lumen of the tube are needed. These are known as "full-lumen" esophagoscopes. Functional cure is obtained with a relatively small lumen at the point of stenosis. A lumen of 7 mm. will allow the passage of any well masticated food. It is unwise and unsafe to attempt to restore the lumen to its normal anatomical size. In cicatricial stricture cases it is advisable to examine the esophagus at monthly periods for a time after a functional cure has been obtained, in order that tendency to recurrence may be early detected.

*Retrograde Dilatation with Tucker Bougies.*—This is the method of choice in all cases that do not readily yield to esophagoscopic bouginage. A gastrostomy for the purpose is justifiable. Adults swallow the silk thread readily, washing it down with water; so that they need not wear it between treatments. With children, however, the string should be worn "endless."

A 2-yard length of twisted silk is wound on a folded paper bobbin and pinned to the waist. The free end is put back through the nose, and more slack given as needed. In the course of a few days the bight of the string stretched across the stomach, from the cardia to the pylorus, is fished out with a pillar retractor. The string is attached to the Tucker bougie and a fresh string is pulled in to replace the one pulled out. Sizes are increased only at long intervals; never until a preceding size comes up loosely. We have never had a death because of this careful method of procedure. If the child is under two years, or is unable to swallow the string because of the degree of stenosis, the silk is placed by retrograde esophagoscopy or as in total atresia. Soft twisted silk is best for the first swallowing of a string in difficult cases. Once down, this can be used to pull through the thicker, stronger, Pearsall's braided silk or linen.

*Impending Atresia.*—In obliteration of the lumen of a stricture, which usually occurs from union of interlocking granulations, no time is to be lost. It is easy and safe to maintain a lumen; but total atresia requires the utmost skill to create an opening through, and the procedure is not free from risk. A silk thread will be our sheet anchor in impending atresia. A gastrostomy should be done. A cure by the Tucker retrograde bougie is then only a matter of careful work. Postponing gastrostomy is a common mistake. Apart from the risks of delay, the sooner it is done the less fibrous tissue there will be to combat.

*Total Atresia.*—This was formerly considered as condemning the patient to lifelong dependence upon gastrostomic feeding and a pocket spitecup for drooling. The development of the method of combined peroral and retrograde esophagoscopy (*q. v.*) has enabled us to cure all of these cases in which the obliterated portion is not too lengthy. So far we have lost but one patient, but the operation cannot be considered free from risk. It should not be attempted without a double plane fluoroscope and the aid of an expert fluoroscopist.

In cases of absolute atresia the saliva does not reach the stomach. No one realizes the quantity of normal salivary drainage, nor its importance in nutritive processes. Oral insalivation is of little consequence compared to esophagogastric drainage. Gastrostomized children with absolute atresia of the esophagus do not thrive unless they regurgitate the salivary accumulations into the funnel of the gastroscopic feeding tube. This has been abundantly proved by observations at the Bronchoscopic Clinic. My attention was first called to this clinical fact by Miss Frances Grove, who had charge of some of these children.

CHEVALIER JACKSON and CHEVALIER L. JACKSON.

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## CARCINOMA AND SARCOMA OF THE ESOPHAGUS

**Definition and Synonyms.**—"Cancer" has come into common use as a word with which to designate any form of malignant growth. Strictly, however, it refers to carcinoma as distinguished from sarcoma and endothelioma. A malignant growth is one whose cells not only proliferate but also infiltrate normal tissue cells.

**Etiology.**—Beyond age and irritation little is yet proved as to the etiology of carcinoma and sarcoma anywhere in the body, and this is equally true of the esophagus. The irritations peculiar to the esophagus are the swallowing of irritating foods, the stagnation and fermentation of foods, and the regurgitation of acid contents of the stomach. The most common form of irritant food is that taken excessively hot. The statistics of A. Logan Turner,<sup>1</sup> J. S. Fraser,<sup>2</sup> W. T. Gardiner,<sup>3</sup> and G. Ewart Martin<sup>4</sup> have shown a preponderance of location of esophageal cancers in the hypopharynx and upper end of the esophagus in women who drink excessively hot tea. In view of the preponderance in the statistics elsewhere of esophageal cancer located in the lower third, and in males, the inference as to the drinking of hot tea seems well founded. A parallel inference, and one that lends support, has been drawn by Carmody in regard to the eating of excessively hot rice by men in China. There the high esophageal cancers preponderate in men, who eat first, while the rice is hottest. Another factor in irritation of the esophageal mucosa is stagnation. The esophagus normally empties itself upward or downward, and is intolerant of the continued presence of anything. Foods quickly ferment and become excessively irritating. Hence, any obstructive disease becomes a possible cause of cancer, chronic esophagitis, erosion, and ulceration being intermediate steps. At the Bronchoscopic Clinic we have had cases of cancer of the esophagus that occurred after *peptic ulcer*, *pre-ventriculosis* (so-called "cardio-spasm"), diverticulum, and cicatricial strictures. The predilection of cancer everywhere for sites of irritation is paralleled in the esophagus by the greater frequency at the points of physiologic narrowing; obviously these points are more exposed to irritation. Many cases of cancer at the lower end of the esophagus are extensions of gastric carcinoma. Hence, the etiology of gastric cancer is to be considered.

**Symptomatology.**—The chief symptom is difficulty in swallowing, but it may be absent or not noted by the patient during the first year or more in the development of cancer of the esophagus. A slight, vague sense of something not being just right in the swallowing function may be present for a long time prior to actual difficulty in getting food down. Suddenly some day the patient finds that food has lodged. After a time, possibly with the aid of swallowed water, it goes down and the patient may notice no more trouble for many days, or even months. His general health is good, his weight is normal, he has no aches or pains. The patient and sometimes the medical attendant is misled into supposing the transient difficulty in swallowing to have been "a little spasm." Whereas, it was not spasm, but a lumen narrowed by cancer, causing ill-masticated food to lodge. If a stomach-tube is passed it will usually go through into the stomach. If an esophagoscope is passed at this time, a fungating, ulcerating, bleeding lesion is usually found, and biopsy confirms the esophagosopic diagnosis of cancer. Unfortunately, not one patient in hundreds comes for esopha-

gосcopy at this stage. Soon the lodging of food becomes frequent; later, no solids will go down; still later even liquids cannot be swallowed. Then the patient has the typical hopelessly late symptoms of anemia, emaciation, and cachexia; pain may still be absent.

**Diagnosis.**—The symptoms and the history of the case are not only useless for diagnosis, but are often actually misleading. For instance, one patient may insist he has never had any trouble in swallowing until the day before; another patient may insist he has had trouble in swallowing at times for twenty years; yet in each instance the patient may have cancer. The accumulation of invaluable scientific data requires that the full list of diagnostic steps enumerated in a previous chapter be gone through an duly recorded; but it cannot be too strongly emphasized that there are only two methods of examination worthy of a moment's consideration for the diagnosis or the exclusion of cancer of the larynx, and no examination should be considered complete unless both of them have been used. They are: (1) Esophagoscopy, and (2) Roentgen-ray examination. Though the esophagoscopy is mentioned first it should not be done until after the Roentgen-ray examination has excluded aneurysm and has yielded such other information as it can give as to foreign body or disease. It is especially important as showing not only the presence of periesophageal cancer, but the lateral extent of any growth. The reader desiring information on the Roentgen-ray signs of cancer is referred to the writings of Manges, Pancoast, Pfahler, Hirsch, and other authorities on this subject. The esophagoscopic appearances of endo-esophageal cancer are typical and will rarely be mistaken for anything else by those who are thoroughly familiar with the normal esophagoscopic appearances. The taking of a specimen for biopsy involves no risk if fungations are present. It is not wise to bite through a normal mucosal surface in the effort to reach infiltration beneath the mucosa. It is better to postpone the biopsy in such cases until a month or two later, when fungations probably will be present.

The *esophageal bougie* as a diagnostic means is always negative early, inconclusive later, inferential at best, and often fatal. A diagnosis by the classical symptoms of pain, emaciation, and cachexia is hopelessly late, and even these symptoms may be present in the absence of cancer. Pain, a prominent symptom of cancer elsewhere, is always late in cancer of the thoracic esophagus and may be totally absent to the end.

By esophagoscopy, endo-esophageal cancer can be diagnosticated not only early, but with the absolute certainty essential in order to get the consent of a comparatively well man to an operation he may not survive. Endo-esophageal cancer can be diagnosticated just as early, just as quickly, and just as certainly as cancer of the cervix if an opportunity for esophagoscopy is afforded early.

**Esophagoscopic Appearances.**—Endo-esophageal malignant growths develop at a very early stage characteristic and unmistakable appearances. These have been fully described, and are well known to esophagoscopists. Anyone who has seen many cases of cancer in the pharynx and who is also accustomed to monocular vision through an endoscopic tube will rarely, if ever, fail to recognize ulcerative, fungating cancer of the esophagus, such as is illustrated in Plate XXIV. Some delay in esophagoscopic diagnosis arises in the case of periesophageal growths producing a compression stenosis of the esophageal lumen. In these cases the presence of a hard

stenosing mass palpable with the tube mouth, outside the wall, and obliterating the soft resilient unfolding of the normal esophageal wall (Plate XXIV) is diagnostic to the experienced esophagoscopist. When these appearances are present the esophagoscopist should always consult with the roentgenologist, who is able in practically every such case to outline the exterior border of a periesophageal growth. The internist will exclude pulmonary tuberculosis and suppurative disease that might produce a secondary compressive adenitis, and lues will be excluded by the usual means.

**Prognosis.**—The mortality of cancer of the esophagus has been practically 100 per cent. A few skilful surgeons have obtained a few cures in advanced cases by exsection; and undoubtedly they would obtain many cures if diagnosis were made early; in other words, if an opportunity for esophagoscopy occurred early. Under palliative treatment the patient may survive as long as five years from the time of probable incipience of the disease. Early esophagoscopy would improve the prognosis.

**Prophylaxis.**—Prevention of incidence of cancer in the present state of our knowledge consists in avoidance of irritation. As applied to the esophagus this is to be done by the early diagnosis and cure of all curable diseases of the esophagus, the avoidance of irritating and excessively hot foods and drinks. Hot tea<sup>1</sup> and coffee are probably the most commonly used of these

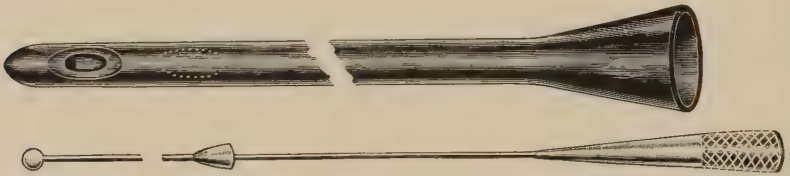


Fig. 608.—Soft-rubber intubation tube for intubation of esophageal cancer in patients who refuse permission for gastrostomy.

irritants. So many of the cancers encountered at the lower end of the esophagus in the esophagoscopy examinations in the Bronchoscopic Clinic were the upper edge of extensions from malignant growths in the stomach that the prevention of cancer of the esophagus is closely allied to prevention of gastric cancer. In 2 cases there was good reason to believe that cancer of the esophagus was secondary to peptic ulcer of the esophagus. Prophylaxis of mortality from esophageal cancer considered apart from incidence, rests fundamentally, here as elsewhere, on early diagnosis. This can be made easily and positively by esophagoscopy preceded by Roentgen-ray examination, and in no other way. So long as the patient is allowed to drift on to certain death under erroneous inferential diagnoses of "spasm" and "globus hystericus," just so long will it be useless to try to lessen the mortality of cancer of the esophagus.

**Treatment.**—There is good reason to believe endo-esophageal cancer is curable, surgically, at some levels at least, if a sufficiently early diagnosis be made. The fundamental reason for the thousands of agonizing deaths from cancer of the esophagus is false education. The mind of the medical student and the medical profession is so saturated with the idea that cancer of the esophagus is to be detected by obstruction to the passage of a bougie, in a patient with dysphagia, pain, cachexia, and emaciation, that the surgeon never gets a chance to cure esophageal cancer while it is still a local process.



Illustration of the misleading and fatal results of blind bouginage for diagnosis. (Painted from a postmortem specimen by Chevalier Jackson.) The patient had been admitted moribund with mediastinal hemorrhage and emphysema. The trachea is opened through the "rings" to the right of the membranous posterior wall; the larynx is laid open from behind; the esophagus is split down the right wall as far as the diaphragm. The perforation evidently had been made by a bougie, shortly before admission, causing the fatal mediastinal hemorrhage shown by the huge blood-clot. The edges of the perforation showed no histologic evidence of malignancy. The bougie had evidently gone through normal esophageal wall, where the lumen was not pathologically stenosed, above the cricopharyngeus, and far above the cancer, which is seen in the esophagus at the level of the diaphragm surrounded by a mass of lymph-nodes under the pleura. The danger of perforation by a blindly passed bougie through cancerous tissue is frequently mentioned, but the greater danger of perforation of the perfectly normal wall is not so generally realized. It is also noteworthy that this patient was treated for a supposed "cardio-spasm" at three different clinics before reaching the place where an erroneous diagnosis of cancer of the cervical esophagus was made because the bougie was arrested in the neck and came back bloody! No esophagoscopy was done in this case because the patient was dying when admitted.





## PLATE XXIV

### ENDOSCOPIC VIEW OF CARCINOMA AND SARCOMA OF THE ESOPHAGUS

These views were sketched by Chevalier Jackson from memory shortly after the respective esophagoscopies, hence represent a view of the interior of the esophagus at a certain level in the particular cases, the patient being in the dorsal position in each instance. No anesthetic, general or local, was used, a fact which must be borne in mind when considering color. The sketches serve to illustrate the modern method of diagnosis of esophageal disease by looking at the lesion. They also show how accurately a specimen, when desirable, can be taken from any selected part of the lesion.

1. Normal esophageal folds as seen at the moment the esophagoscope enters the thoracic esophagus. The delicate pink, velvety surfaces, the soft folds flattening without resistance at the approach of the tube-mouth and yielding to the slightest manipulation of the tubal lip; the resilient respiratory recession and advance; the pulsatory movement of one wall at certain levels; all these are, esophagoscopically, so characteristic of the normal esophagus, to the accustomed observer, that any abnormality in color, form, or movement is instantly apparent. The sketch shows the image during the momentary pause at the end of the expiratory phase.

2. Whitish, nodular form of carcinoma on the posterior wall of the upper end of the esophagus in a woman, aged fifty-eight years, who for over a year and a half was thought to have had "spasm of the esophageal muscles," "cardiospasm," "neurasthenia," "major hysteria," finally "mental derangement," because of increasing "globus hystericus" and, later, "refusal" (!!) to swallow food, for which she was ultimately placed in a psychopathic institution. She was fed with a stomach-tube, the passage of which was thought to rule out organic esophageal disease. After having consulted specialists in various parts of the world she came under the observation of an internist who at once decided the esophagus should be looked at with the esophagoscope. It required thirty seconds to find the lesion, make an esophagoscopic diagnosis of cancer, and take a specimen, which Dr. B. L. Crawford found to be a squamous-celled epithelioma. The symptomatic stimulation of a neurosis by cancer of the esophagus is very frequently observed by esophagoscopists.

3. Cancer of the thoracic esophagus in a man, aged twenty-six years, sent in with a diagnosis of "cardiospasm." Form more often than color is the esophagoscopic criterion of malignancy. The anterior (upper) fold is not very different in color from the normal mucosa, but it is granular, almost nodular, with fungations which at a later stage are often quite exuberant. The creases between the folds are filled with oozing blood.

4. Cancerous infiltration of the thoracic esophageal wall in a man, aged forty-five years, supposed to have spasmodic stenosis in the middle-third of the esophagus. Here again the color of the mucosa was not far from normal; but the hardness, the rigidity, the absence of the normal characteristics of movement incidental to respiration, pulsation, and manipulation described above (1) were deemed so characteristic of malignant infiltration that an esophagoscopic diagnosis of cancer was made. As it was deemed inadvisable to take a specimen at this stage, it was requested that the patient be sent back in six weeks. (See the legend to the following illustration, 5.) An esophagoscopic diagnosis of malignancy was made, however, on the conditions here described, and an immediate gastrostomy was advised, but was postponed.

5. Same patient as in the preceding illustration (4). The anterior rounded ridge of infiltration has ulcerated; a center slough, discolored with bismuth sulphide, is ready to come away. The edges are beginning to fungate, which renders the taking of a specimen perfectly safe. This was done, and Dr. B. L. Crawford reported upon the specimen, squamous-celled epithelioma. The relatively rapid progress of the ulceration was probably due to the constant presence of stagnant, fermenting, irritating food.

6. On the right wall is seen an old scar. This was probably due to the swallowing of lye, twenty years before, of which there was a very clear history. The bleeding fungations at the distal edge of the scar looked so suspicious of malignancy that they were nipped off and sent to the laboratory. Dr. Ernest W. Willetts reported upon the specimen as undoubtedly squamous-celled epithelioma.

7. Boy, aged sixteen years. Lymphosarcoma involving the anterior wall of the esophagus at the level of the crossing of the left bronchus. This patient had no difficulty in swallowing. He was supposed to have asthma, causing him to "wheeze and to cough until he vomited." He was referred for bronchoscopy because of the asthmatic symptoms. The bronchoscopic findings shown in the following illustration (8) led to the esophagoscopy. Air-bubbles were seen at the depressed necrotic area, and a hissing sound was heard through the esophagoscope when the patient voluntarily coughed at request. Part at least of the dark grayish color of the exudate is due to sulphided bismuth.

8. View down the left main bronchus of the patient referred to in the preceding illustration (7). The bleeding granular mass on the posterior wall of the bronchus evidently was part of the growth visible esophagoscopically (7). What appears like a grayish slough was partially sulphided, swallowed bismuth that came through from the esophagus. Blood was oozing among the fungations.

PLATE XXIV



1



2



3



4



5



6



7



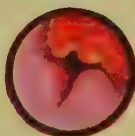
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9



10



11



12

Chevalier Jackson, 1924



9. Woman, aged forty-two years. Difficulty in swallowing diagnosticated cancerous on three previous occasions because of arrest of the bougie, the tip of which came back bloody, and of the history of progressive dysphagia and emaciation. The yellow, globular mass protruding in the ragged, irregular opening was scooped out with the mechanical spoon and found to be cheesy debris and thick pus, with a few giant-cells and elements of glandular structure. No tubercle bacilli were found, but Dr. Ernest W. Willets deemed the process suppurating tuberculosis of the mediastinal glands rupturing into the esophagus. There was no clinical or ray evidence of active pulmonary tuberculosis. Dr. Russell H. Boggs stated that there were slight indications of healed apical lesions. The patient made a good recovery, with normal swallowing, and was above normal weight four years later.

10. Man, aged forty-seven years. Gastrostomy had been done before admission. This illustration shows a mixed lesion, luetic and cancerous. The large, smooth, rounded mass on the left wall was not very hard and was movable with the tube-mouth. Because of the 4+ Wassermann reaction it was deemed best not to take a specimen. The patient was put upon energetic antiluetic treatment, which resulted in the total disappearance of the dysphagia.

11. The same patient as in the preceding illustration (10); esophagosopic appearances after two months of energetic antiluetic treatment (KI and Hg prot.). The nodular mass, which was probably a gumma, is seen to have disappeared; but the granular mass has increased in extent and is fungating exuberantly and oozing blood freely. Three endoscopists at the Bronchoscopic Clinic were unanimously of the opinion that the fungating lesion was cancerous. This diagnosis was doubted by the attending physician because of the total disappearance of dysphagia under antiluetic treatment, the gastrostomy tube having been entirely abandoned. A specimen was therefore taken and was reported by Dr. Baxter L. Crawford as squamous-celled epithelioma. The progress of this case confirmed this diagnosis.

12. Peptic ulcer of the esophagus in a man, aged forty-seven years, suffering from dysphagia, odynphagia, severe nocturnal substernal pain extending through to the back. These symptoms, with obstruction to the passage of a bougie which came back bloody had led to the diagnosis of cancer of the esophagus. The patient was emaciated apparently from pain, sleepless nights, and worry in anticipation of a death by cancer, rather than from lack of nourishment. The border of the ulcer was not hard and a small esophagoscope would pass it, going on through the hiatal esophagus into the stomach, which was about 8 cm. below the lower border of the lesion. A specimen removed from the edge of the ulcer showed no evidence of malignancy; only inflammation and ulceration. Histologic elements of gastric mucosa and glandular structure led to the diagnosis of peptic ulcer of the esophagus, starting in an esophageal island of gastric mucosa.



When every patient mentioning the slightest abnormal sensation in the cervical, retrosternal, or epigastric region is considered not necessarily neurotic but possibly cancerous an early diagnosis will be made by the roentgenologist and the esophagoscopist. It seems probable that radon seeds, accurately placed with the esophagoscope in an early endo-esophageal cancerous nodule, as advocated by Pancoast, will cure some cases.



Fig. 608a.—Pancoast-Jackson esophagogoscopic radon seed planter.

**Palliative Treatment.**—The most important palliative measure is very early gastrostomy to stop the mechanical irritation of passing and lodging food, and the chemical irritation of fermenting and stagnant food and secretions saturated with oral infections. If an early gastrostomy is done and the esophagus put at rest with only water by mouth, saliva will go through for a long time. If the patient is allowed to go far in food and water starvation he cannot be brought back even after gastrostomy. Intubation will postpone the necessity for gastrostomy but will rarely obviate it. If used the best tube is that shown in Fig. 608. Stretching the cancerous stenosis has been advocated by some, condemned by others. It is condemned for all other regions by all surgeons. Radium has been disappointing in our hands; but as shown by the excellent work of Sargon<sup>13</sup> an improved technic may demonstrate better results in the future.

*Deep Roentgen-ray therapy* has been the most efficient palliative measure in our experience, when combined with early gastrostomy and proper diet. Unless carefully watched patients will drift into a diet of meat broths, milk, and eggs. They should have an abundance of fruit juices and vegetable soups, properly strained so that no particle will lodge in the esophagus and cause irritation. After gastrostomy the same rule applies to tube feeding.

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## BENIGN TUMORS OF THE ESOPHAGUS AND HYPOPHARYNX

**Varieties.**—The case records of the Bronchoscopic Clinics of Pittsburgh and Philadelphia show histories of the following benign tumors in which the esophageal wall was involved:

*Granuloma, edematous polypi, papilloma, keloid fibroma, adenoma, lipoma, myoma, myxoma, angioma, hematoma, varix, lymphangioma, and cystic growths.*

Periesophageal tumors, by compression, often give rise to esophageal symptoms. (See under Compression Stenosis of the Esophagus.)

**Etiology.**—The cause of benign tumors is discussed in works on pathology.<sup>4</sup> Varicosities are usually due to obstruction to the venous return, and those in the lower third may be due in part to excessive hiatal pinchcock constriction; they are often associated with disease of the liver. In the hypopharynx the cricopharyngeal pinchcock may be a factor in the obstruction to venous return.

**Symptoms.**—If the tumor is not large enough to cause obstruction there may be no symptoms. Larger growths will cause difficulty in swallowing, regurgitation and, rarely, nausea. Laryngotracheal and pulmonary symptoms may result from compression, or may be due to inflammatory lesions secondary to overflow and inspiration of infected secretions that normally drain away harmlessly and unnoticed, through the esophagus. Coughing and choking on taking food occurs in the latter class of cases. A long-pediced tumor in the hypopharynx may get over into the larynx and cause cough, dyspnea, and even asphyxia.

**Diagnosis.**—Esophagoscopy is the only method of diagnosis. Histological diagnosis is invaluable; it may be done on the entire tumor in the case of small growths, or on an esophagoscopically removed specimen in case of large growths. The esophagoscopic appearances show absence of infiltration, and the experienced eye will see at once the absence of invasion of normal structures. Benign growths are usually covered by an epithelial surface; erosion occurs only after irritation. Vascular tumors are evidenced by their red, or more often crimson or purplish color.

**Treatment.**—The growths should be removed, entire if possible; piecemeal if necessary. The biting forceps are used through the laryngoscope for hypopharyngeal growths; through the esophagoscope in the case of growths farther down. The sole exception is varicosity. If the condition is one of varix, general treatment directed to the liver is indicated. Angiomata do not bleed dangerously if the coagulation time is within normal limits. There is no indication for radical removal of basic normal tissues, and such a procedure would involve great danger, because of the normally thin wall of the esophagus and hypopharynx. Especial care is necessary to avoid the tearing loose of firmly attached fibromata, lest trauma be inflicted on the delicate esophageal wall.

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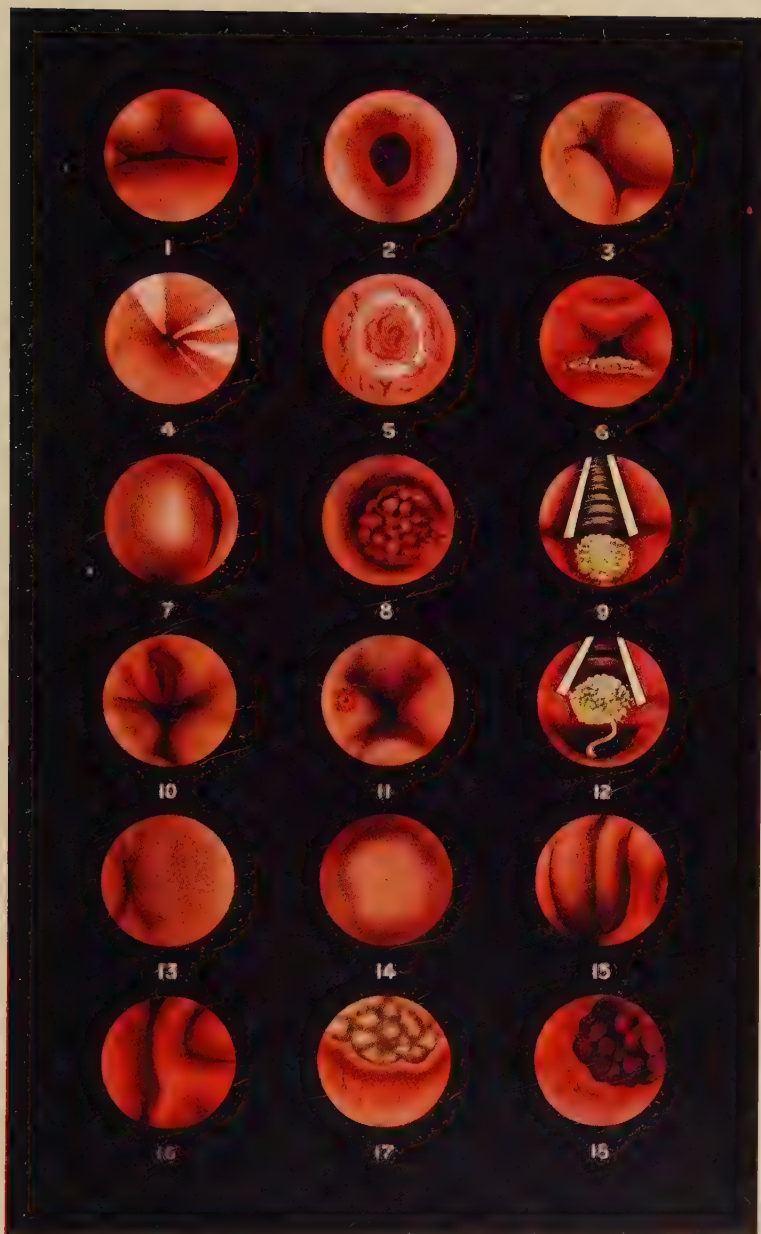
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## PLATE XXV

1. Introitus esophageus. Normal. Dark line must not be understood as a gaping. Collapsed shut. Man of thirty-six.
2. Intra-thoracic esophagus. Unusual view, but normal. More usual appearance shown in Fig. 1, Plate IV.
3. Esophagus at hiatus diaphragmatis normal. Note axis of lumen. Man of sixty.
4. Cicatricial esophageal stenosis. Pin-hole lumen. White scars. Recurrence of stenosis following ulceration during typhoid fever. Primary lesion, burned by swallowing lye in childhood fourteen years previously. Mr. H., aged twenty-one years.
5. Ibid. Bottom of diverticulum. Mucosa chronically inflamed.
6. Tubercular ulceration posterior esophageal wall, simulating decubitus ulcer often seen in typhoid fever. Tubercular lesion in this location is somewhat rare, though still more rarely is it diagnosed. Incidentally this figure shows the introitus esophageus when the cricoid cartilage is lifted by the laryngeal speculum. Compare Fig. 1, above.
7. Carcinoma of the thoracic esophageal wall (left) covered with normal mucosa. Lumen pushed to the right and almost obliterated. Man aged sixty years.
8. Carcinoma, endo-esophageal. Woman of forty-one years, referred for chronic nasal sinus disease. Esophageal symptoms slight and attributed to globus hystericus.
- 9 and 12. Fibroma papillare, attached by long slender fibrous peduncle. Disappeared into the esophagus at times after swallowing. Fig. 12 shows the attachment within the esophagus when the cricoid cartilage is moved forward (instrument not shown). Removed through tubular speculum. Man aged thirty-six years.
10. View in thoracic esophagus showing wounds (above) made by blind groping with a coin extractor which did not extract. Boy of fourteen years.
11. Wound in esophageal wall made by a pin which was afterward found higher up. Woman of twenty-three years.
13. Normal. "Kink" of the esophagus at the hiatus, probably more a preventive of regurgitation than the cardia.
14. Peri-esophageal carcinoma overlaid with normal mucosa, lumen deviated so far to right as to be out of view. Diagnosis upon hardness of mass, and age of the patient. Man of sixty years.
15. Stomach ulcer (on left side of right fold in the view), bed showing dark after secretions had been wiped away. Other folds normal. Woman aged twenty-six years.
16. Stomach. Normal. Branched fold. Dark crimson color. Examined one hour after drinking milk. Man aged thirty-two years.
17. Stomach. Carcinoma. Zone of hyperemia. Man of forty-six years.
18. Stomach. Same patient. Mulberry-like nodule at another portion of growth.

PLATE XXV





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## ANATOMY OF THE TRACHEOBRONCHIAL TREE

Extensive consideration of the anatomy of the tracheobronchial tree will be found in anatomical works. Special considerations of value to the endoscopist have been separately published.<sup>3</sup> What is necessary for the bronchoscopist is the mental conception of what surrounds the trachea at each level and the anatomical landmarks as seen endoscopically. A con-

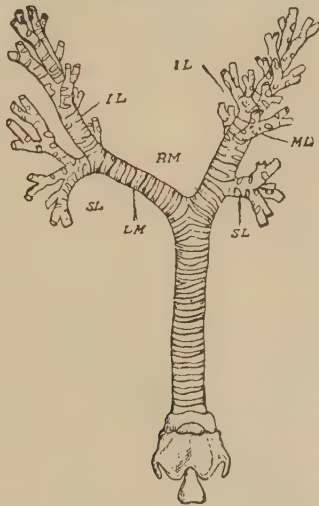


Fig. 609.—Tracheobronchial tree: *LM*, Left main bronchus; *SL*, superior-lobe bronchus; *ML*, middle-lobe bronchus; *IL*, inferior-lobe bronchus. It is essential to remember that the tree is extremely elastic, that it elongates and the lumen enlarges on inspiration, converse movements occurring on expiration. The tree is dinged and deflected by transmitted pulsatory movements and the thoracic contents normal or abnormal, as the case may be, 1, 2, 3.

ception of these two things can be acquired only on the cadaver. The endoscopic landmarks after being identified seriatim on the way downward with the bronchoscope dozens of times on the cadaver are promptly identified in the living, so that the bronchoscopist always knows where his tube-mouth is in the tracheobronchial tree. This is absolutely essential and it can be acquired in no other way. In order to get a conception of what is outside the trachea or bronchi at any of the different levels it is necessary to pass the bronchoscope on a cadaver of which the entire front wall of the thoracic cage has been removed. The cadaver should be limp. For ready reference the illustrations, Figs. 609, 610, will be useful.

The movements of the tracheobronchial tree are of the utmost importance not only in foreign-body work but also because their absence or alteration are valuable diagnostic points; for instance, in adenopathy at the bifurca-



Fig. 610.—Bronchoscopic Left Bronchus views: *S*, Superior-lobe bronchus; *I*, inferior-lobe bronchus; *M*, middle-lobe bronchus; *SL*, Superior-lobe bronchus; 1, 2, 3.

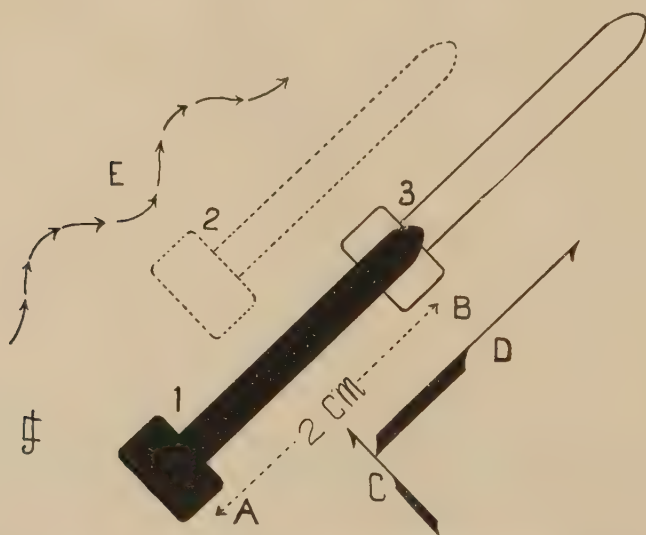


Fig. 611.—Schematic illustration of the normal respiratory and pulsatory movements of the bronchi as measured in the fluoroscope in the case of the child with an iron casting in its bronchus (Fig. 000). The foreign body was fixed in the right bronchus. There was an excursion of the bronchus longitudinally (elongation and shortening) of about 2 cm. between maximum inspiration and maximum expiration (*A* to *B* as shown by arrow *D*). During this longitudinal excursion there were from 2 to 4 lateral deflections in the direction of the dart *C* amounting to about 1 cm., due to transmitted cardiac movements. This caused the elongating and shortening excursions to be wavy as shown schematically at *E*. This elongating and shortening causes points of foreign bodies to bury and the entire foreign body to work ratchet-like more deeply into the lung.<sup>4</sup> (From Jour. Am. Med. Assn., January 27, 1917.)

tion and in cancer anywhere in the chest. The normal movements of the tracheobronchial tree consist of elongation and shortening (Fig. 611) as well as of expansion and contraction (Plate XXVI).<sup>4</sup>

CHEVALIER JACKSON and CHEVALIER L. JACKSON.

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## FOREIGN BODIES IN THE AIR AND FOOD PASSAGES

The enormous increase in the importance of this subject within the last twenty years is due chiefly to three factors: (1) The discovery that foreign body, far from being an uncommon cause of pulmonary symptoms, is really the first diagnostic possibility to be excluded in every case of acute or chronic disease of the chest; (2) the development of Roentgen-ray diagnosis; and (3) the development of endoscopic methods to the point where 98 per cent. of the patients can be cured by the removal of the foreign body through the mouth.<sup>1, 2, 3, 4, 5, 6</sup>

**Definition.**—Any solid or semisolid substance that is lodged in the air or food passages may, broadly speaking, be called a foreign body. The term has been extended to include not merely an intruder from without, but also substances that, originating within the body, take on the qualities of a foreign substance—teeth, bone, cartilage, sloughs, casts of membrane, broncholiths, calcified glands, etc. A mouthful of meat, if it passes into the stomach, is food; if it lodges in the esophagus it becomes a foreign body. A classification of the 2125 foreign bodies removed at the Bronchoscopic Clinic shows one or more examples of nearly every substance with which human beings come in contact. The animal, vegetal, and mineral kingdoms are each represented by a large number of specimens (Fig. 636).<sup>6</sup>

**Etiology.**—Statistics of the Bronchoscopic Clinic show that carelessness is the chief cause of foreign body accidents. A penetrating projectile may be inspired (Moure, Garel Sargnon<sup>25</sup>). We have had one such case. All the etiologic factors have been analyzed.<sup>19</sup>

**Symptomatology and Diagnosis of Foreign Body in the Air or Food Passages.**—Whether or not he expects to do bronchoscopy and esophagoscopy for foreign body, it is essential that the laryngologist have for reference a complete tabulation of the essentials of symptomatology and diagnosis. The following is a summary of the essentials culled from a personal experience of forty years and from the records of the Bronchoscopic Clinic covering to date 2125 cases of foreign body in the air and food passages:

**Initial Symptoms.**—The most important initial symptoms are choking, gagging, coughing, and wheezing, often followed by a symptomless interval. A foreign body may be in the larynx, trachea, bronchi, nasal chambers, nasopharynx, fauces, tonsil, pharynx, hypopharynx, esophagus, stomach, intestinal canal, or may have been passed by bowel, coughed out or spat out, with or without knowledge of the patient. Initial choking, etc., may have escaped notice or may have been forgotten.

*Symptoms of Laryngeal Foreign Body.*—One or more of the following laryngeal symptoms may be present: Hoarseness, croupy cough, aphonia, odynphagia, hemoptysis, wheezing, dyspnea, cyanosis, apnea, subjective sensation of foreign body. Croupiness usually means subglottic swelling. Obstructive foreign body may be quickly fatal by laryngeal impaction on inspiration, or on abortive beehee expulsion. Lodgment of a non-obstructive foreign body may be followed by a symptomless interval. Direct laryngoscopy for diagnosis is indicated in every child supposed to



Fig. 612.—Roentgenogram of a child aged two years sent to the Bronchoscopic Clinic for decannulation after a supposed laryngeal diphtheria. The symptoms of diphtheria were due to a safety-pin that had been in the larynx for eight months. Prompt recovery and decannulation followed removal of the safety-pin.

have laryngeal diphtheria without faucial membrane (Fig. 612). (No anesthetic, general or local, is required.) In the presence of laryngeal symptoms the following possibilities should be considered:

1. A foreign body in the larynx.
2. A foreign body loose or fixed in the trachea.
3. Digital efforts at removal.
4. Instrumentation.
5. Overflow of food into the larynx from esophageal obstruction due to foreign body.

6. Esophagotracheal fistula from ulceration set up by a foreign body in the esophagus, followed by leakage of food into the air-passages.

7. Laryngeal symptoms may persist from the trauma of a foreign body that has passed on into the deeper air or food passages, or that has been coughed or spat out.

8. Laryngeal symptoms (hoarseness, croupiness, etc.) may be due to digital or instrumental efforts at removal of a foreign body that never was present.

9. Laryngeal symptoms may be due to acute or chronic laryngitis, diphtheria, pertussis, infective laryngotracheitis, and many other diseases.

10. Deductive decisions are dangerous.

11. If the Roentgen ray is negative, laryngoscopy (direct in children, indirect in adults) without anesthesia, general or local, is the only way to make a laryngeal diagnosis.

12. Before doing a diagnostic laryngoscopy, preparations should be made for taking a swab-specimen and for bronchoscopy and esophagoscopy.

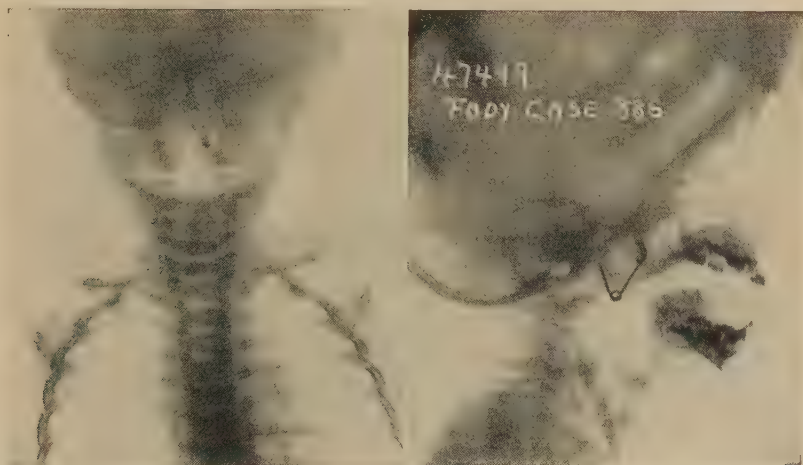


Fig. 613.—Roentgenogram showing a safety-pin in the nasopharynx of a baby aged nine months. It was invisible on oral examination. The mother said the baby "swallowed" a safety-pin. The foreign body would have been overlooked had it not been our rule to have ray examination include the entire region from the nasopharynx to the tuberosities of the ischia.

*Symptoms of Tracheal Foreign Body.*—(1) "Audible slap," (2) "palpatory thud," and (3) "asthmatoïd wheeze" (Jackson<sup>22</sup>) are pathognomonic. The "tracheal flutter" has been observed by McCrae. Cough, hoarseness, dyspnea, and cyanosis are often present. Diagnosis is by Roentgen ray, auscultation, palpation, bronchoscopy. The "asthmatoïd wheeze" is heard with the ear or stethoscope bell (McCrae) at the patient's open mouth, not at the chest wall.<sup>22</sup> Listen long for the "audible slap," also heard at the open mouth. It is the name given by the author to the peculiar sound caused by the sudden arrest of the foreign body by the subglottic narrowing, during nature's expiratory or bechic attempt at expulsion of the intruder. The impact felt by the examiner's thumb on the trachea constitutes the sign that the author has called the "palpatory thud." History of initial choking, gagging, and wheezing is important if elicited, but is valueless negatively.

*Symptoms of Bronchial Foreign Body.*—Initial symptoms are coughing, choking, asthmatoïd wheeze, etc., noted above. There may be a history of these or of tooth extraction. At once, or after a symptomless interval, cough, blood-streaked sputum, metallic taste, or special odor of foreign body may be noted. Non-obstructive metallic foreign bodies afford few symptoms and few signs for weeks or months. Obstructive foreign bodies cause atelectasis, drowned lung, and eventually pulmonary abscess. Lobar pneumonia is an exceedingly rare sequel. Vegetal organic foreign bodies such as peanut kernels, beans, watermelon seeds, and the like cause at once violent laryngotracheobronchitis (vegetal bronchitis) with toxemia, cough, and irregular fever (Fig. 620), the gravity and severity being inversely to the age of the child.<sup>8</sup> Bones and metallic bodies after months or years produce pathological changes which cause chills, fever, sweats emaciation, clubbed fingers, incurved nails, cough, foul expectoration,



Fig. 614.—Roentgenogram showing safety-pin in the pelvis. At the right is a roentgenogram of the same patient showing how the foreign body may be overlooked when only the chest is included in the ray examination. In both this patient and the one illustrated in Fig. 615 the foreign body would have been overlooked were it not for the rule to have all the region from the nasopharynx to the tuberosities of the ischia subjected to ray examination in every case.

hemoptysis, in fact, all the symptoms of chronic pulmonary abscess or bronchiectasis, and many signs which may suggest pulmonary tuberculosis. The apices, however, are normal, and Koch's bacilli are absent from the sputum.

It must always be borne in mind that a foreign body lodged in the esophagus may present many of the signs of bronchial foreign body. This is caused in two ways:

1. A foreign body in the esophagus may, by the obstruction to swallowing, cause solid foods, liquids, and saliva to overflow into the larynx, whence they are aspirated into the lungs.

2. A foreign body lodged in the esophagus may in time ulcerate its way into the trachea, producing a fistula through which food, liquids, and saliva leak directly into the lower air-passages. The symptoms of pulmonary disease are violent in either form, and if the foreign body is not removed, death quickly follows.

**Diagnosis of Foreign Body in the Tracheobronchial Tree.**—*The Roentgen Ray.*—This is the most valuable diagnostic means, but careful notation of physical signs by an expert is, in all cases, essential. Expert ray work will show all metallic foreign bodies and many of less density, such as natural



Fig. 615.—(Case No. Fbdy. 1173.) Another illustration of the wisdom of the rule to have the ray examination, in all cases of suspected foreign body, include all structures from the nasopharynx to the tuberosities of the ischia. After esophagoscopic removal of the "burr" of bent safety-pins from the hypopharynx and cervical esophagus the child would have been discharged were it not for our knowledge of the presence of the needle in the intestines. This saved the child's life. The needle perforated and was removed by laparotomy. Possibility of multiple foreign bodies requires the complete ray examination mentioned above.<sup>1</sup>

teeth, bones, shells, buttons. Artificial teeth do not throw a shadow, but vulcanite plates do. If the ray is negative, a diagnostic bronchoscopy should be done in all cases of unexplained obstruction. The ray examination should in all cases include the entire anatomy from the nasopharynx to the tuberosities of the ischia (Figs. 613-615). The differ-

ential diagnosis as to location in the air or the food passages is illustrated in Figs. 616 and 617.

Peanut kernels, maize, watermelon seeds, and other vegetal substances in the bronchi produce obstructive emphysema of the invaded



Fig. 616.—Roentgenograms of a safety-pin lodged in the larynx. The sagittal position of the plane of the expanded pin is diagnostic of location in the larynx. This plane is maintained if the pin passes spontaneously down the trachea.<sup>1</sup>



Fig. 617.—Roentgenograms show safety-pin in the esophagus of a child aged six months. The expansion of the pin being in the coronal plane is diagnostic of location in the esophagus. This plane is necessary for the pin to pass between the cricoid and the spine and the same plane is maintained in the thoracic esophagus if the foreign body should pass downward. Note in Fig. 633 that four safety-pins all following each other downward lodged in the thoracic esophagus in the same way, *i. e.*, expanded in the coronal plane.<sup>1</sup>

side.<sup>8</sup> Fluoroscopy shows the diaphragm flattened, depressed, and of less excursion on the invaded side; at the end of expiration the heart and mediastinal wall move over toward the uninvaded side, and the invaded lung becomes less dense than the uninvaded lung, from trapping of the air

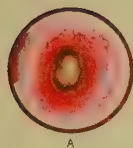


## PLATE XXVI

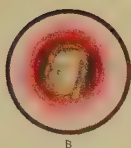
### ENDOSCOPIC VIEWS ILLUSTRATING MECHANICAL PROBLEMS ENCOUNTERED IN CASES OF FOREIGN BODIES IN THE LUNGS.

A, Foreign body (a bone) impacted in a bronchus so tightly that no forceps-spaces existed. Before admission prolonged fruitless efforts under general anesthesia had been made to grasp the foreign body without realization of the impossibility of doing so in the absence of forceps-spaces. The inflammatory areola shows where the mucosa had been punched with the opened forceps. B, Same patient as in A after I had created lateral forceps-spaces by withdrawing the foreign body to a higher level with a hook. Forceps were then readily applied and the foreign body was easily removed. C, Mucosal trauma inflicted by the attempt to force forceps jaws onto a foreign body sagittally where no forceps-spaces existed, ignoring good lateral forceps-spaces. D, The trauma, indicated by the inflammation, the swollen dividing-spur and the patch of exudate on the mucosa of the left-hand orifice, was inflicted before admission by the faulty attempt to grasp the foreign body seen in the right hand orifice. My predecessor in the case stated that he had grasped the foreign body and had pulled as hard as he dared. As the foreign body was free to move it is certain the traction was being made upon forceps that included tissue as well as foreign body. E, Endoscopic view in the lower-lobe bronchus showing a tack that, before admission, had been injudiciously pulled upon without first disengaging the point. Release of the point by the author's outward rotation method after pushing the tack downward resulted in a prompt and safe removal. F, Annular edematous (not fibrous) stenosis from the trauma inflicted before admission, in jamming the foreign body (a screw) down in a bronchus, in a faulty effort to grasp the screw-head in the absence of forceps-spaces. On admission only a tiny portion of the screw-head was visible and the situation of the slot or fillister indicated a slight tilting of the screw. The problem in this case was solved by withdrawing the screw to a new position above the edematous area with the closed side-curved forceps used as a hook. The same forceps were then used in the usual way to grasp and remove the foreign body. G, A peanut kernel in the bronchus of a child. During expiration, as here shown, no forceps-spaces existed because of the collapse of the bronchial walls during this phase of the respiratory cycle. H, Immediately upon the beginning of inspiration the bronchial walls recede from the peanut kernel, creating large forceps-spaces between it and the bronchial walls. In the case here illustrated the spaces are located anteriorly and posteriorly. It is early during this inspiratory stage of respiration that the forceps must be placed. The here-shown phenomenon of the opening and closing of the forceps-spaces in respiration is the mechanism by which air is trapped in the invaded lung or lobe, producing the obstructive emphysema that is diagnostic of the presence of a peanut kernel in the lung. In this case Dr. Willis F. Manges had made a diagnosis of non-opaque foreign body, probably peanut kernel, in the right bronchus, in the absence of a history.

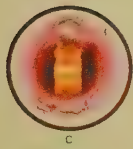
PLATE XXVI



A



B



C



D



E



F



G



H

♀



on expiration by the valve-like effect of obliteration of the "forceps-spaces" that during inspiration afford air ingress between the foreign body and the swollen bronchial wall.<sup>8</sup> This *partial* obstruction causes *obstructive* emphysema (Fig. 618), which must be distinguished from *compensatory* emphysema in which the ballooning is in the unobstructed lung, because its fellow is wholly out of function through *complete* "corking" of the main bronchus of the invaded side. It cannot be too strongly emphasized that, to get a film like that illustrated in Fig. 618, *exposure must be made at the end of expiration*. The phenomenon of obstructive emphysema, first discovered by Iglauer, has been so thoroughly studied by Manges<sup>16</sup> that he has been able in over 100 cases to tell me with absolute accuracy that a peanut kernel or a grain of maize or other non-opaque vegetal substance was



Fig. 618.—Obstructive emphysema caused by non-radiopaque foreign body, a peanut kernel in right main bronchus. Note (a) depression of right diaphragm, (b) displacement of heart and mediastinum to left, (c) greater transparency of the invaded side. *Exposure made at end of expiration*. At inspiration the heart and mediastinum moved back to their proper position. This sidewise movement with respiration is characteristic of obstructive emphysema; it does not occur in obstructive atelectasis.<sup>1,8,16</sup> Compare B, C, Fig. 619. Ray-plate made by Willis F. Manges.

present in the lung at a certain definite location with reference to the bronchial tree. Also, in a number of cases of incomplete removal, he has been able to tell me that a fragment remained behind, and on this information I have done a second bronchoscopy and found the remaining fragment in the location specified. *Obstructive atelectasis*, on the other hand, resulting from the collapse of a lung after "corking" by the swelling of a bean, for instance (Fig. 619), is equally diagnostic.<sup>8</sup> The mediastinal structures move toward the obstructed side and stay there; that is, they do not move sidewise with respiration as in obstructive emphysema. Both obstructive emphysema and obstructive atelectasis are most often seen in cases of vegetal foreign bodies (*q. v.*). The fluoroscope shows this sidewise motion and is used for diagnosis, but it is not used during the bronchoscopy in these cases.

**Physical Signs.**—Secretions, normal and pathological, may shift from one location to another; the foreign body may change position, admitting more, less, or no air; or it may shift to a new location in the same lung or even in the other lung.<sup>11</sup> A recently inspired pin may produce no signs at all. The signs of diagnostic importance are chiefly those of partial or complete bronchial obstruction, though a non-obstructive foreign body, a pin for instance, may cause limited expansion (McCrae) or, rarely, a peculiar râle or a peculiar auscultatory sound. The most nearly characteristic physical signs are:

1. Limited expansion.
2. Decreased vocal fremitus.
3. Impaired percussion note.
4. Diminished intensity of breath-sounds distal to the foreign body.

Complete obstruction of a bronchus followed by "drowned lung" adds absence of vocal resonance and vocal fremitus, thus often leading to an erroneous diagnosis of empyema. Varying grades of tympany are obtained over areas of obstructive or compensatory emphysema. With complete obstruction there may be tympany from collapsed lung for a time. In cases of complete obstruction, râles are usually most intense on the uninvaded side. In partial obstruction they are most often found on the invaded side, distal to the foreign body, especially posteriorly, and are often most intense at the site corresponding to that of the foreign body. A foreign body at the bifurcation of the trachea may give signs in both lungs. Early in a foreign body case, diminished expansion of one side, with dullness, may suggest pneumonia in the affected side; but the decreased vocal fremitus and the diminished breath-sounds with absence of or decreased vocal resonance, and absence of typical tubular breathing should soon exclude this diagnosis. Bronchial obstruction in pneumonia is exceedingly rare. As stated by McCrae:

"There is no one description of physical signs which covers all cases. If the student will remember that complete obstruction of a bronchus leads to a shutting off of this area, there should be little difficulty in understanding the signs present. The diagnosis of empyema may be made, but the outline of the area of dullness, the fact that there is no shifting dullness, and the greater resistance which is present in empyema nearly always clear up any difficulty promptly. The absence of the frequent change in the voice-sounds, so significant in an early small empyema, is of value. A large empyema should give no difficulty. If difficulty remains the use of the needle should be sufficient. In thickened pleura, vocal fremitus is not entirely absent, and the breath-sounds can usually be heard, even if diminished. In case of partial obstruction of a bronchus, it is evident that air will still be present, hence the dullness may be only slight. The presence of air and secretion will probably result in the breath-sounds being somewhat harsh, and will cause a great variety of râles, principally coarse, and many of them bubbling. Difficulty may be caused by signs in the other lung or in a lobe other than the one affected by the foreign body. If it is remembered that these signs are likely to be only on auscultation, and to consist largely in the presence of râles, while the signs in the area supplied by the affected bronchus will include those on inspection, palpation, and percussion, there should be little difficulty." For a complete presentation

of diagnosis and all clinical features of foreign bodies in the air-passages see article by Thomas McCrae.<sup>5</sup>

**Symptoms of Esophageal Foreign Body.**—After the initial choking and gagging, or without these, there may be a subjective sense of a foreign body, constant or, more often, only on swallowing. Odynphagia and dysphagia or aphagia may or may not be present. Hematemesis and fever may occur from the foreign body or from rough instrumentation. Symptoms referable to the air-passages may be present due to: (1) Overflow of secretions on attempts to swallow through the obstructed esophagus; (2) erosion of the foreign body through from the esophagus into the trachea; (3) compression of the trachea by a large foreign body in the esophagus;

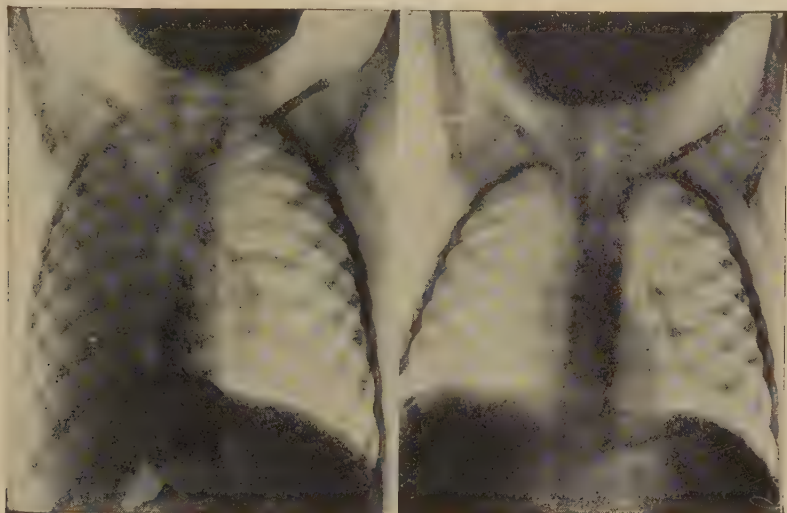


Fig. 619.—The roentgenogram at the left shows a typical obstructive atelectasis from a bean inspired two days previously. The bean is in the opaque lung. The heart and mediastinal structures have moved into the right chest as a result of the negative pressure resulting from absorption of the imprisoned air. The roentgenogram at the right shows restoration of lung, heart, and mediastinum to normal one day after removal of a bean from the right lung. But all the time the bean was “corking” the right bronchus the heart and mediastinal structures remained in the left chest; they *did not* move sidewise with respiration. This is characteristic of obstructive *atelectasis* and distinguishes it from obstructive *emphysema*. (Compare Fig. 618.)<sup>1, 8, 16</sup>

or (4) trauma inflicted on the larynx during attempts at removal, digital or instrumental, the foreign body still being present or not.

**Diagnosis** is by the Roentgen ray, first without, then, if necessary, with a capsule filled with an opaque mixture. Flat objects, like coins, always lie with their greatest diameter in the coronal plane of the body when in the esophagus; in the sagittal plane when in the trachea. Lateral, antero-posterior, and sometimes also quartering roentgenograms are necessary.

**Arachidic and Other Forms of Vegetal Bronchitis.**—A violent inflammatory condition is set up in the lungs of children by vegetal foreign bodies. From an experience of over 400 cases at the Chevalier Jackson Clinics the following conclusions have been reached<sup>8</sup>:

1. Vegetal bronchitis is a peculiar and serious type of septic bronchitis due to the inspiration of vegetal foreign bodies into the lungs, especially of children (Fig. 620).

2. The disease is much more frequent than is supposed, because most of the cases are overlooked. About 200 cases have been observed at the Bronchoscopic Clinic. Many other cases have been published in the literature, though the true significance is not noted in the older literature.

3. The chief etiological factor in vegetal bronchitis is the inspiration into the lower air-passages of peanut kernels, nut kernels, beans, water-

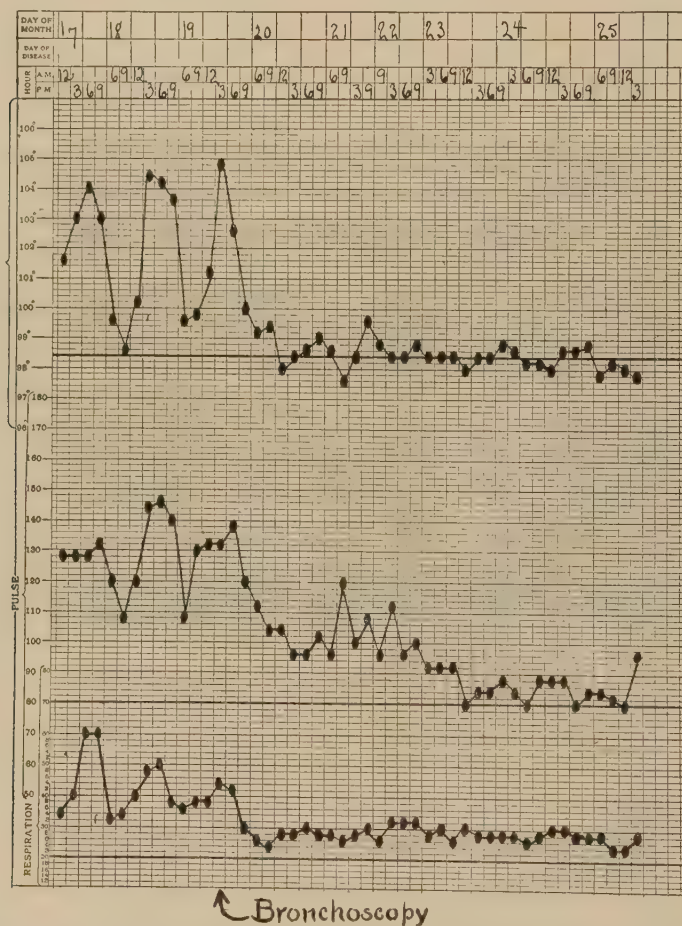


Fig. 620.—(Case No. Fbdy. 2008.) Clinical chart of a child aged nine years showing temperature, pulse, and respiration records typical of vegetal bronchitis. The prompt fall to normal after the peroral bronchoscopic removal of the causative peanut kernel is typical of cases in which the vegetal foreign body has not been in the lung more than a few days. The return to normal is not a crisis; there was no pneumonia present. The condition was a vegetal bronchitis and the fall was due to the removal of the vegetal foreign body.

melon seeds, maize (corn), apple, orange, or other fruit seeds or pulp. Foliage, stems, wood fiber, or any vegetal substance may cause it. Age is the most important general factor; it is peculiar to children, and severity is usually inversely as the age. Carelessness is usually one factor in the etiology.<sup>19</sup>

**Prognosis of Foreign Body in the Lung.**—If allowed to remain, only about 2 or 3 per cent. of the patients recover, by coughing up of the foreign

body. Bronchoscopic removal can be accomplished in almost 100 per cent. of the cases and about 98 per cent. of the patients so treated recover perfect health.<sup>23</sup> Pins even below the level of the dome of the diaphragm can be bronchoscopically removed.

**Prognosis of Foreign Body in the Esophagus.**—If the foreign body remains, almost all cases result fatally. Almost 100 per cent. of foreign bodies in the esophagus can be removed through the mouth esophago-



Fig. 621.—From a photograph of dental foreign bodies removed bronchoscopically and esophagoscopically at the Chevalier Jackson Clinics of Philadelphia.<sup>24</sup>

scopically. In 800 cases at the Bronchoscopic Clinic it has never yet been necessary to resort to external esophagotomy. Many enormous and jagged dentures (Fig. 621), bones, and other objects have been removed by peroral esophagoscopy.<sup>24, 25</sup>

**Prognosis of Foreign Body in the Stomach.**—Any radiopaque foreign body that has gone down the natural passages into the stomach can be

removed through the mouth. The advisability of so removing it is elsewhere herein considered.<sup>1</sup>

**Prophylaxis of Foreign Body Accident.**—In view of the statistical fact, established by the records of over 2000 cases at the Bronchoscopic Clinic,<sup>19</sup>

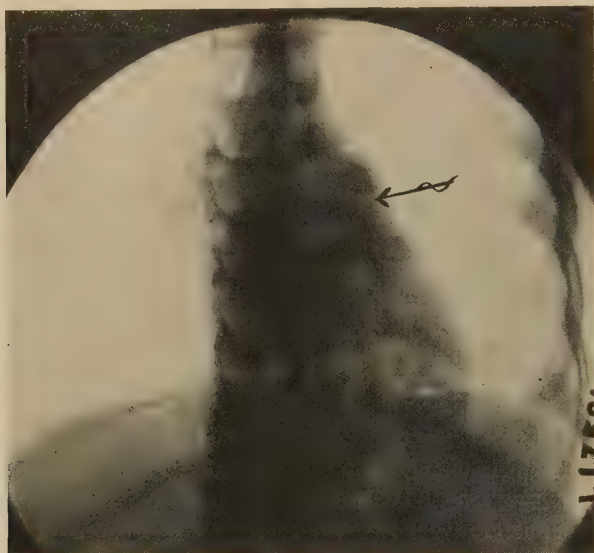


Fig. 622.—(Case No. Fbdy. 2116.) Roentgenogram of a child aged three years showing a tooth in the left main bronchus, dislodged by the mouth-gag and aspirated during tonsillectomy.

that nearly 90 per cent. of the cases of foreign body in the air and food passages have been the result of avoidable accidents, it seems the duty of every physician to teach prophylaxis to mothers, nurses, cooks, and waitresses.<sup>14</sup>

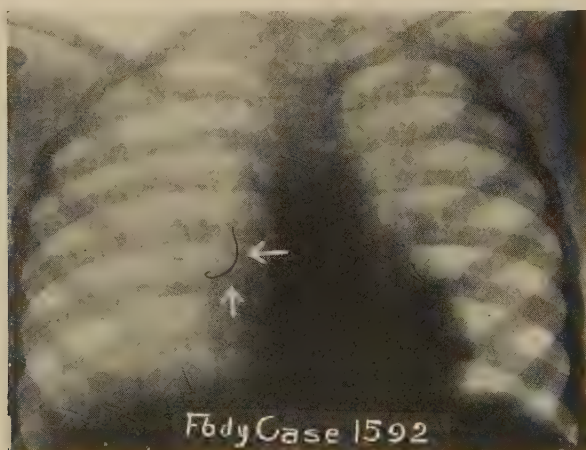


Fig. 623.—Tonsil snare wire inspired during tonsillectomy. Early diagnosis and prompt bronchoscopic removal forestalled chronic pulmonary suppuration. The shadow of the wire was retouched for reproduction. (Film by E. P. Pendergrass.)

The endoscopic removal at the Bronchoscopic Clinic of over 100 dental and surgical objects as foreign bodies (Fig. 621) is a fact that calls

for the study of prophylaxis of foreign body accidents by all who work around the nose, throat, and mouth.<sup>26</sup> *Teeth* were lost downward not only



Fig. 624.—Portion of tonsil forceps inspired into the lung during tonsillectomy. Prompt recovery followed the bronchoscopic removal of the piece of tonsil forceps. This case illustrates the value of early diagnosis and prompt bronchoscopic removal of the foreign body in the prophylaxis of chronic lung suppuration. (Film by Willis F. Manges.)

during exodontia, but in many instances during tonsillectomy (Fig. 622). George Fetterolf has made it a rule in his Nose and Throat Clinic at the

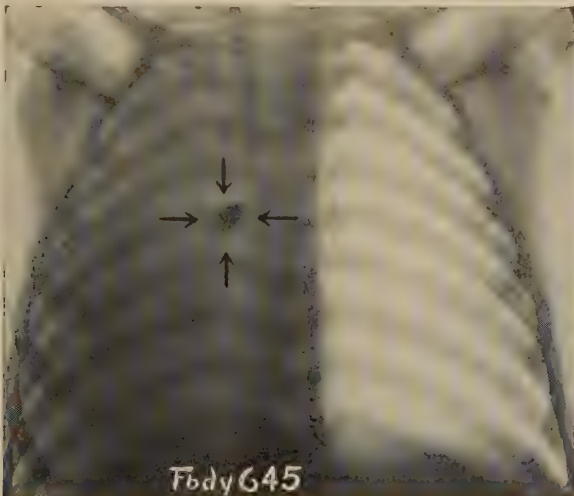


Fig. 625.—Arrows point to part of a mouth-gag inspired during tonsillectomy. Atelectasis with beginning suppuration of the left lung was arrested by bronchoscopic removal. This case illustrates the value of early diagnosis in the prophylaxis of chronic lung suppuration. (Film by David R. Bowen.)

University of Pennsylvania Hospital that any very loose deciduous teeth, ready to come away, should be removed before tonsillectomy. *Artificial dentures* were lost downward because of either one of two things: (1)

They were ill-fitting from breakage or alveolar changes, or (2) the patient was unconscious from epilepsy, shock, delirium, intoxication, or natural

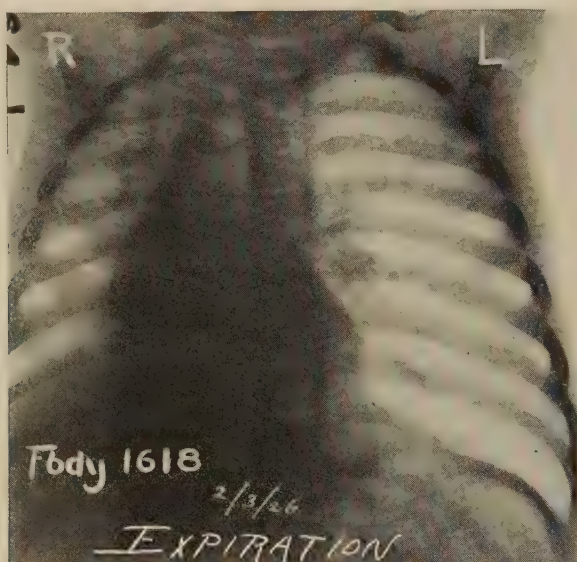


Fig. 626.—Atelectasis and infectious disease of the right lower lobe secondary to inspiration of a tonsil swab. After bronchoscopic removal of the swab many bronchoscopic aspirations were required before the lung fully recovered from the suppurative process. (Film by Willis F. Manges.)

sleep. The prophylaxis is obvious. *Portions of tonsil instruments as foreign bodies* removed from the lung at the Bronchoscopic Clinic (Figs. 623–625)

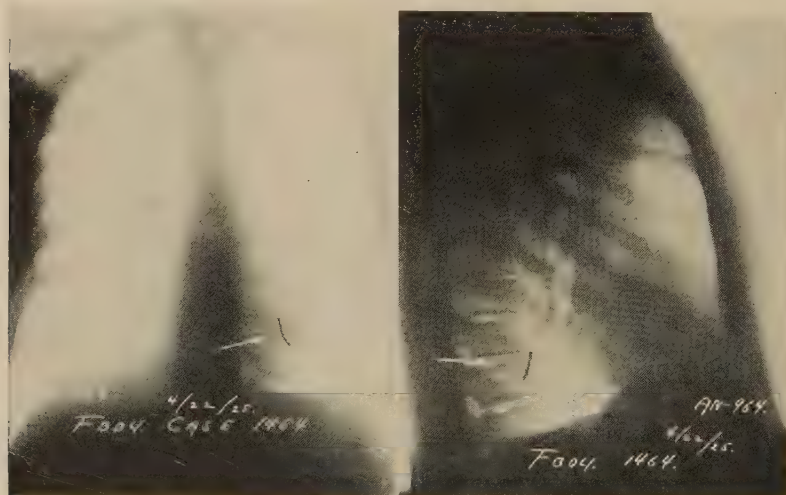


Fig. 627.—Roentgenogram of a man aged forty-five years showing the shadow of a piece of nasal rasp broken off during an intranasal operation. The foreign body was removed from a posterior branch deep in the lower lobe.

suggests the need of the utmost care in the inspection and use of these instruments before operation, and also the need of buying only from

responsible makers, who have a reputation to lose. As suggested by George Fetterolf snare wires should not be used over again. *Swabs as foreign bodies* (Fig. 626) in the lung suggest that if left in the tonsillar fossa a swab should be tethered, and that an applicator as now made of wood is so liable to break that a metal one should be used for all purposes in the nose or throat. I have taken up with my esteemed colleague, Professor Okada of the Imperial University of Tokyo the making of practically unbreakable

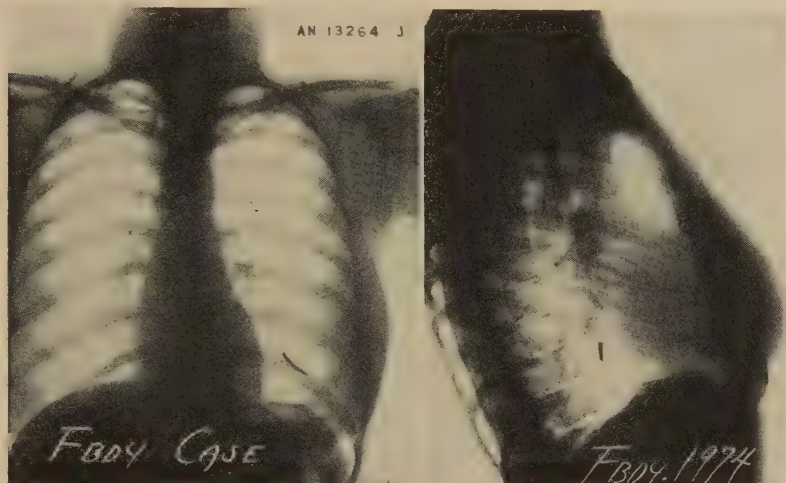


Fig. 628.—Roentgenogram of a woman aged twenty-three years showing the shadow of a fragment of nasal rasp broken off during intranasal operation.

applicators of bamboo. The pieces of *nasal rasps as foreign bodies in the lung* (Figs. 627–629) indicate that it is a dangerous error to temper a nasal rasp “glass-hard” throughout its diameter. Only the teeth need be hard and, being for use on bone, they need not be as hard as a hack-saw or file for metal. The temper should be drawn to the point where the instrument will bend before it will break. Though we do not do these operations,

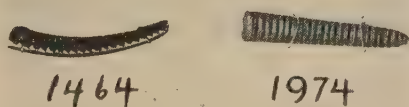


Fig. 629.—From photographs of the two pieces of the nasal rasp removed bronchoscopically from the respective patients shown in Figs. 627 and 628.

we suggested to Mr. Charles J. Pilling that he make rasps of this kind as a prophylactic measure.

**Treatment of Foreign Bodies in the Air and Food Passages.**—Endoscopic methods have superseded all others in these cases because of the fact that in about 98 per cent. of the cases the foreign body can be removed with a risk of less than 2 per cent. mortality, in well trained hands.

During the endoscopic procedure it is important to have the roent-

genographic film, suitably illuminated and placed in *inverted* position in the shadow box (Figs. 630, 631).<sup>1</sup>

**Treatment of Foreign Body in the Larynx.**—The removal of a foreign body from the larynx by direct laryngoscopy is such a simple and easy procedure that no other method is worthy of a moment's consideration. As shown by the statistical tables of the Bronchoscopic Clinic only a few instruments are necessary for picking out the intruder. The laryngoscope (Fig. 556) and the laryngeal forceps (Fig. 562) are used. Occasionally a foreign body may slide on downward into the trachea, for which reason the bronchoscope should be in the hands of the instrument nurse lighted and

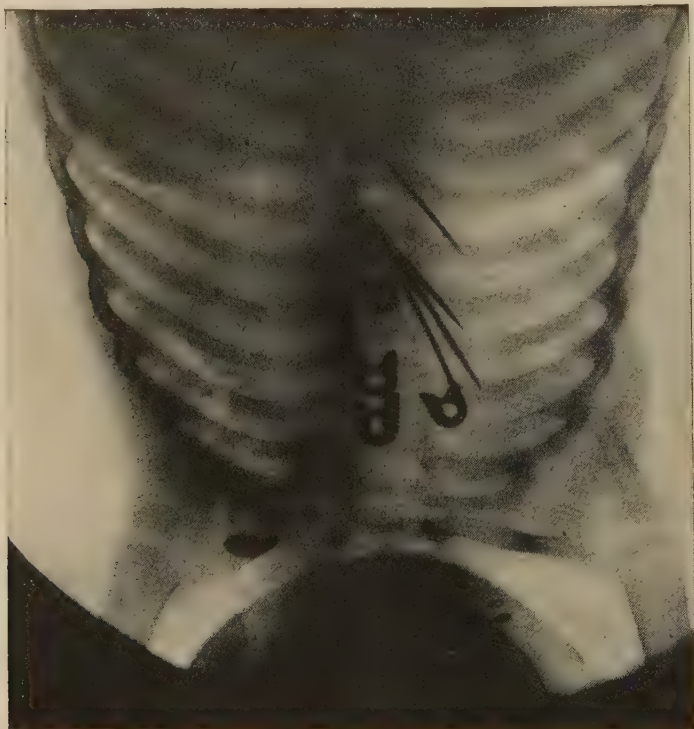


Fig. 630.—This illustration shows the advantage of turning the roentgenogram upside down for esophagoscopy and bronchoscopy in the recumbent position. This contributes to a proper conception at bronchoscopy of where the unseen parts of a foreign body are in relation to the visible parts. Comparison with Fig. 633 will show the great advantage of this plan. We have here the three points to our right, one to the left, in true relationship to our instruments and all manipulations of instruments and foreign bodies.<sup>1</sup>

ready for immediate insertion through the laryngoscope, and the bronchoscopic forceps and sponge carriers should be on the sterile table.

**Direct Laryngoscopy for Foreign Bodies Overhanging the Larynx.**—In case of a foreign body in the larynx or overhanging it the exceptional position shown in Fig. 631 is necessary. It will be noted that though the chest is inclined relatively to the horizon and that the head is lower than the chest, yet the anatomical relations are the same as in the Boyce position.

**Treatment of Foreign Body in the Tracheobronchial Tree.**—No attempt at removal of a foreign body from the tracheobronchial tree by any method other than bronchoscopy is worthy of a moment's consideration. As

stated by Keen the bronchoscope has entirely revolutionized this department of surgery. The same is true of foreign bodies in the lung that have reached the parenchyma of the lung, either spontaneously as in cases of slender bodies like common pins, or by suppurative processes. It should be stated, however, that bronchoscopy, especially for foreign bodies, and particularly in babies, is a highly technical procedure requiring an ample instrumentarium, a well-trained organization, and a well-taught endoscopist. After removal no treatment is usually required in recent cases, but the patient should be kept in the hospital for a few days for observation. In cases of vegetal foreign bodies, such as nut kernels, a longer period of observation may be advisable. A fragment of nut remaining may require a second bronchoscopy. In cases of prolonged sojourn of a foreign body with extensive secondary pathological processes, rest in bed outdoors is usually

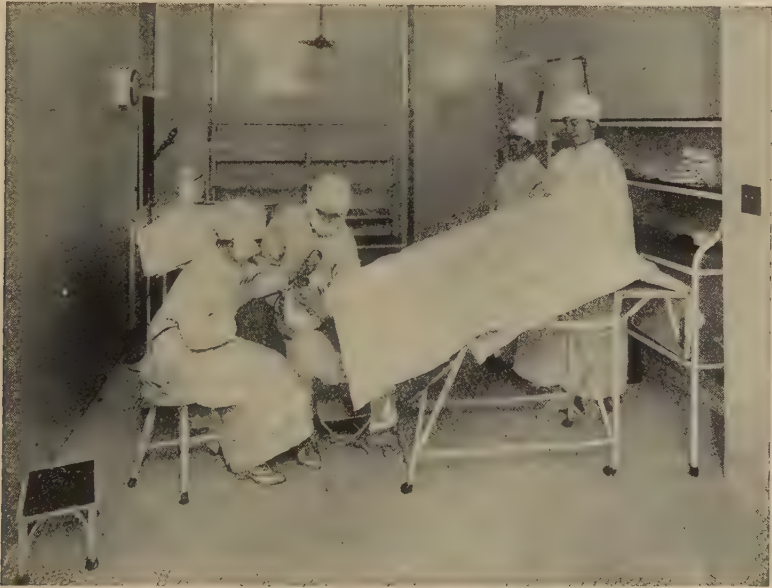


Fig. 631.—The position for the removal of foreign bodies from the larynx or from any of the upper air or food passages. If dislodged, the intruder will not be aided by gravity to reach a deeper lodgment. Anatomically the parts are in the same relation to each other as in the position previously described.

all that is required. Even bronchiectasis following foreign body will get well under this régime. An occasional patient may require redilatation of a stricture or repeated bronchoscopic aspirations.

**Treatment of Foreign Body in the Hypopharynx.**—Foreign body in the hypopharynx should be removed under guidance of the eye looking through the laryngoscope or the esophageal speculum or the esophagoscope. The laryngoscope and the esophageal speculum each have the advantage of the co-ordinate use of both hands (left holding laryngoscope, right using forceps) in the disimpaction and removal of foreign bodies in this region. An expert has the same dexterity of bimanual co-ordinate manipulation of these instruments that most persons have in the use of knife and fork. The always present probability of the foreign body passing on downward has led us at the Bronchoscopic Clinic, in the case of large impacted foreign

bodies, to prefer the esophagoscope, which enables the endoscopist to follow the foreign body without delay. The use of a self-retaining mechanical appliance to force open the hypopharynx is almost certain to result in an empty hypopharynx by the time the operator has succeeded in getting his apparatus fixed in his patient, even in the case of large impacted foreign bodies. In the latter class of cases the problem will have been converted from an easy into a difficult one because of the difficulty of removal of very large foreign bodies from lower levels. However, an experienced endoscopist can remove through the mouth any foreign body that has gone down the natural passages and has lodged at any point not beyond the pylorus.



Fig. 632.—Roentgenogram showing metallic radium capsules each containing 50 mg. of radium. They are contained in a rubber finger-cot. Dr. J. T. Farrell reported that the foreign bodies were in the stomach. They were removed through the mouth by gastroscopy.

**Treatment of Foreign Body in the Esophagus.**—Esophagoscopy removal is the only method worthy of a moment's consideration if there is an experienced esophagoscopist within a distance permitting of transportation of the patient; and in view of the prolonged sojourn of the foreign body in many cases it can rarely be said that immediate removal is so urgently required as to justify blind attempts at removal or the always dangerous external esophagotomy. It cannot be too strongly urged, however, that training in the use of the esophagoscope is essential for safety of the patient. The old "blind" instruments such as the bougie, the bristle probang, the

sponge probang, Bond's forceps, the Kirmisson hook, and the Graefe basket are entirely discarded as obsolete and dangerous.

**Treatment of Foreign Body in the Stomach.**—Peroral gastroscopic removal is indicated in any case of foreign body that will probably not pass out through the pylorus of the particular patient, or that would endanger the patient's health or life if allowed to pass into the intestines. The foreign body should be watched by daily observations with the fluoroscope. The question often arises as to whether or not a foreign body will pass out of

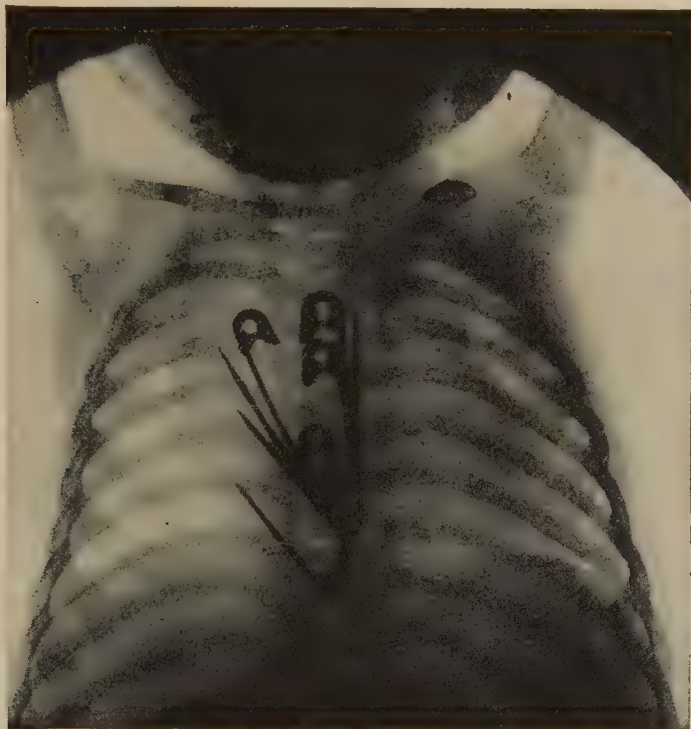


Fig. 633.—(Fbdy. 1071.) Four large, stiff, interlocked safety-pins impacted in the esophagus of a child aged nine months. In addition to the interlocking and impaction from one month's sojourn the pins were bound together with an entangled mass of wool. The problem was rendered still more difficult by the enormous size of the pins and the small size of the passages in a nine-month-old baby. The two lower pins were disentangled off downward; the upper two were removed through the mouth by the point-sheathed method with Tucker forceps, plus the assistance of the Arrowsmith closer with one of the divergent keeper branches. A 6-mm. esophagoscope was used. The only way to get a true conception of such a problem is to compare Fig. 630 with this illustration. Another case of multiple safety-pins is shown in Fig. 615.

the stomach. This depends on the size of the pylorus; if it is normal, any foreign body that has spontaneously reached the stomach will pass out eventually; but a long time may elapse before it does so. My observation of these cases convinces me that under normal conditions a foreign body tries many times at the pyloric vestibule before it is allowed to pass through the doorway. Often it will present in a faulty position and will be refused passage. I have seen safety-pins, hairpins, staples, and the like try to enter points in advance; invariably they have returned to the stomach,

and usually they have eventually presented themselves properly as, for instance, by the round end of a staple or hairpin, and been admitted,



Fig. 634.—(Fbdy. 1740.) Cap off tooth-paste tube removed by gastroscopy through the mouth. It had been in the stomach for two years. As demonstrated by Dr. L. T. Le Wald the cap was unable to escape because of pyloric stenosis.

subsequently passing safely. Coins often remain a few weeks and then pass onward. Nothing should be done to increase peristalsis; regular mixed

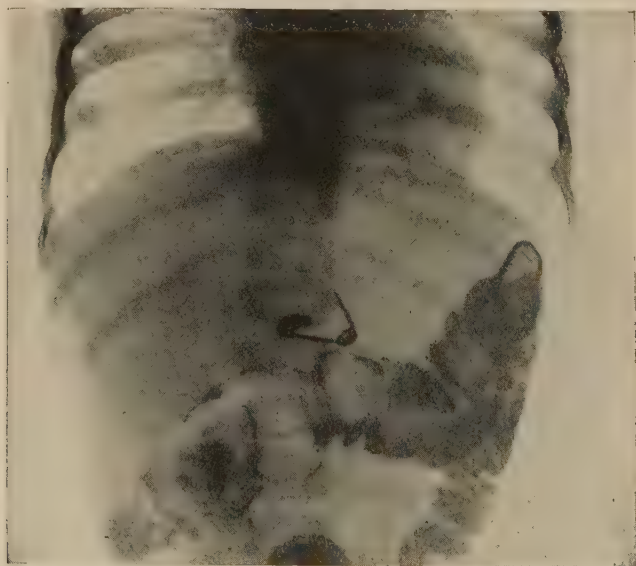


Fig. 635.—(Fbdy. 1183.) Roentgenogram showing a safety-pin jammed in the pylorus of an infant aged eleven months. The keeper branch of the pin was in the duodenum, the pointed branch hooked over the pyloric ring with the point against the stomach wall. The pin had been in this position a number of days. It was removed through the mouth by *peroral pyloroscopy* without anesthesia, general or local. The barium shadow shows the colon. (Film by Dr. Willis F. Manges.)

diet should be given. Perhaps it would be best to do a *peroral gastroscopy* for removal without waiting longer than a few weeks in the case of coins.

External surgery is not called for except perhaps in some cases of multiple foreign bodies, such as are seen occasionally in insane patients.

*Peroral Gastroscopy for Foreign Body in the Stomach.*—Any foreign body that has gone down the natural passages into the stomach can be removed by peroral gastroscopy; but such removal is not always necessary. If the passage through the intestine would be dangerous, as in the case

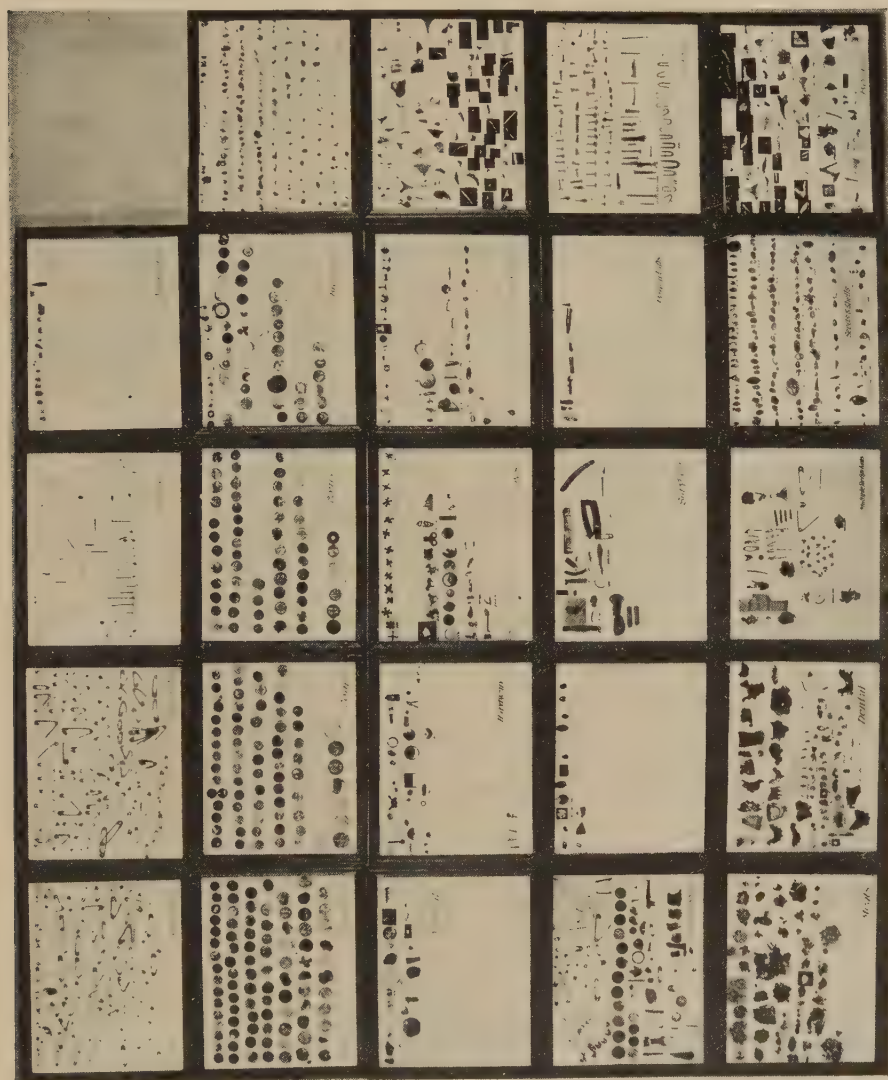


Fig. 636.—Photograph of specimens of foreign bodies removed by peroral endoscopic methods from the air and food passages at the Chevalier Jackson Bronchoscopic Clinics. The specimens are in the Mutter Museum of the College of Physicians of Philadelphia.<sup>24</sup>

illustrated in Fig. 615, removal is urgently called for. Safety-pins are potentially dangerous though many have passed through and out of the intestines safely. The exact size in relation to the size of the patient is important in determining the chances of safe passage. The presence or absence of stenosis of the pylorus is important in decision, as in the case illustrated in Fig. 634.

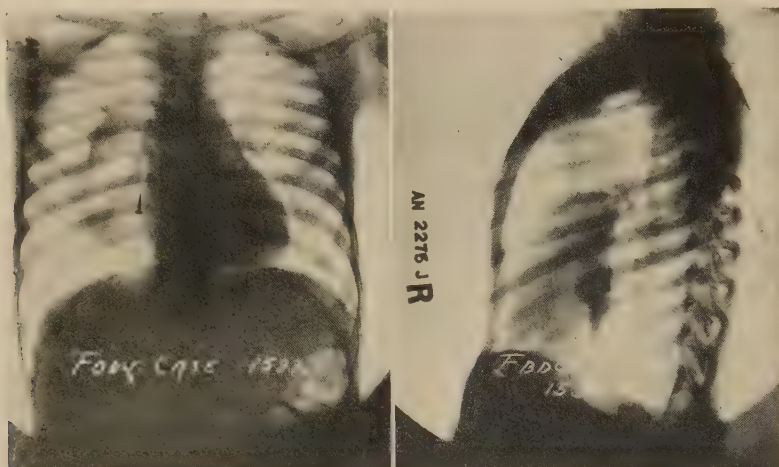


Fig. 637.—Roentgenograms, anteroposterior and lateral, showing, pneumothorax due to injudicious traction on a tack resulting in pneumothorax from perforation. When admitted the child was in serious condition from pus pressure in the pleural cavity. After peroral bronchoscopic removal of the tack with point properly sheathed in the distal end of the bronchoscope the lung gradually expanded and in two weeks the child was discharged perfectly well.

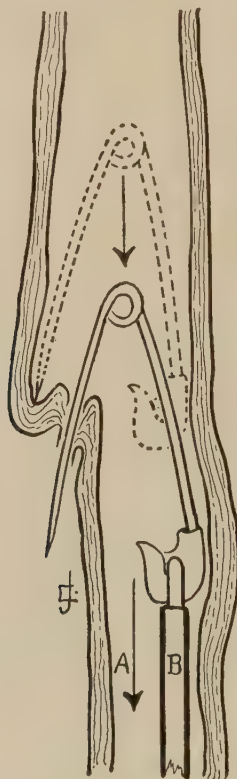


Fig. 638.—Schematic drawing of what will happen if the author's dictum, "advancing points perforate, trailing points do not," is ignored. Injudicious traction by the forceps *B*, in the direction of the dart, *A*, has drawn the pin upward from the position shown dotted and has drawn the advancing point through the esophageal wall. (From *Bronchoscopy and Esophagoscopy*, by Chevalier Jackson, 2d ed., W. B. Saunders Co., 1927.)

*Peroral Pyloroscopy for Foreign Body.*—The pylorus is accessible to the peroral gastroscope with the aid of an abdominal manipulator. We have usually been able to bring the pylorus in which a foreign body was impacted over to the mouth of the esophagogastroscope in the stomach, and the foreign body has been removed by grasping the forceps inserted through the gastroscope (Fig. 635).

**Foreign Bodies in the Intestines.**—After a foreign body has passed the pylorus its progress through the intestines should be watched, and the patient not regarded as out of danger until the foreign body has been evacuated. Large foreign bodies sometimes cause intestinal obstruction, pointed foreign bodies seldom perforate by direct puncture, but the point may ulcerate through. Thus a warning is given: any foreign body that remains in one place in the intestines for as long as four or five days should be removed by external operation (Fig. 615). The roentgenologist is the

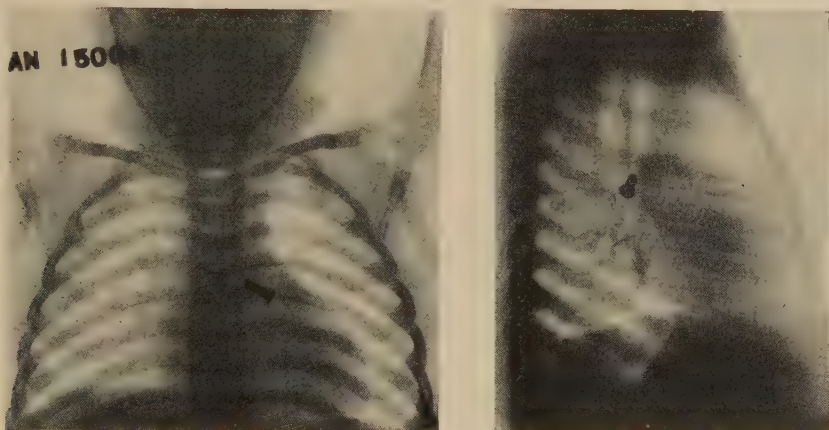


Fig. 639.—(Case No. Fbdy. 2030.) Roentgenograms, anteroposterior and lateral, of a boy aged three years showing a metal mouth-piece from a toy balloon in the left bronchus. Removed bronchoscopically through the mouth in one minute and forty-five seconds, without anesthesia general or local. One jaw of the mosquito rotation forceps was inserted through the 4-mm. bronchoscope, under guidance of the eye into the central canal of the mouth-piece, the other jaw was outside. This placement of forceps is best for all hollow foreign bodies.

safest guide in these cases. Under no circumstances should peristalsis be increased by laxatives. Bulky foods are not needed, and the change of diet may cause intestinal indigestion with increased peristalsis. Normal intestinal contents and normal peristalsis are the safest conditions.

**Mechanical Problems of Bronchoscopic and Esophagoscopic Removal of Foreign Bodies from the Air and Food Passages.**—The introduction of the bronchoscope or esophagoscope is easily learned by anyone who will devote the time to it under a competent teacher. In foreign body cases, however, there is in addition, in most cases, a mechanical problem involved in the seizure, version, disentanglement or disimpaction of a foreign body. This problem varies with the many kinds of foreign bodies (Fig. 636), it may be easily solved, or may present great difficulty unless it was known beforehand, and has been properly worked out on the manikin-board. To grasp a foreign body and pull upon it, in many cases will not only not succeed in removing the foreign body but will make removal a more difficult matter,

and may kill the patient. Many deaths have resulted from failure to realize the danger of pulling upon a foreign body without sheathing the the point in the tube mouth (Figs. 637, 638). This is particularly true of pins, needles, safety-pins, tacks, staples, or any pointed, sharp, rough, curved, or bent foreign bodies. Many hours of practice with a duplicate of the foreign body in a rubber tube and in the living and moving bronchi of a dog under the instruction of a competent master are essential not only to removal, but to the avoidance of disaster. Figure 639 shows neat careful delicate work, by sight, through the smallest bronchoscope (4 mm. diameter). The extreme difficulties encountered and the fact that these difficulties may be successfully dealt with by careful working upon the problems were demonstrated in the case illustrated in Fig. 633.

The small esophagoscope required in babies (only nine months old in this case) enormously increases the difficulties. The extent to which the solution of the mechanical problems of safe bronchoscopic removal of foreign bodies has been worked out will be realized from the fact that there are 16 methods of peroral bronchoscopic removal of safety-pins.<sup>12</sup>

CHEVALIER JACKSON and CHEVALIER L. JACKSON.

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## BACTERIOLOGIC FINDINGS IN CASES OF TRACHEOBRONCHIAL FOREIGN BODIES

Secretions obtained bronchoscopically from the trachea and bronchi in foreign body cases contain a variety of micro-organisms. The more important of these are: *Streptococcus hemolyticus*, *Streptococcus viridans*, *Streptococcus non-hemolyticus*, *Diplococcus pneumoniae*, *Micrococcus catarrhalis*, *Staphylococcus aureus*, *Staphylococcus albus*, *Micrococcus tetragenus*, *Bacillus influenzae*, diphtheroid bacilli, *Bacillus mucosus capsulatus*, *Micrococcus pharyngis siccus* and yeasts. Some of these contribute to the infection, while others, such as the common yeasts, are purely saprophytic.

In studies made at the Chevalier Jackson Bronchoscopic Clinic it has been shown that, as a rule, a variety of bacteria are present in a single case. Usually two, three, or four organisms can be isolated, and occasionally as many as six or seven may be present. Pure cultures of bacteria are of infrequent occurrence.

In a report<sup>1</sup> based on 100 cases of foreign bodies in the tracheobronchial tree, streptococci were present more often than any other organism. *Streptococcus viridans* occurred more frequently than *Streptococcus hemolyticus*. The incidence of the latter was proportionately higher in cases of vegetal and bony foreign bodies than in metallic foreign body cases.

Pneumococci and staphylococci, both the *aureus* and *albus*, were often isolated. However, they were usually associated with other kinds of bacteria. This statement also holds true for such micro-organisms as *Micrococcus catarrhalis*, *Bacillus influenzae*, the diphtheroid bacilli, and *Micrococcus tetragenus*.

Bacilli of the *lucosus capsulatus* group and *Micrococcus pharyngis siccus* were comparatively rare and did not occur in pure culture. On two occasions *Bacillus coli communis* was isolated, and in 1 case in which there was an accompanying putrid bronchitis, *Bacillus fusiformis* and spirochetes were recovered along with other organisms.

The type of foreign body, according to McCrae<sup>2</sup> and Jackson,<sup>3</sup> influences the severity of reaction on the part of the tracheobronchial mucosa. Vege-

tal foreign bodies cause more reaction than metallic ones; this is particularly true in children. Bacteriological findings in these cases do not warrant the conclusion that the bacteria are of primary importance in these reactions, since on many occasions identical types of organisms have been isolated from metallic and vegetal foreign body cases.

The age of the patient and the length of sojourn of the foreign body in the tracheobronchial tree do not seem to bear direct relationship to the type of bacteria present. This is illustrated by the following 2 cases. In the first instance, the patient was a child, aged two years, who had a peanut in his bronchus for five days. A diphtheroid bacillus, *Streptococcus hemolyticus*, and *Staphylococcus aureus* were isolated from the tracheobronchial secretions. The child was actually ill. The second case, a woman aged twenty-nine years, had a nail in her bronchus for eighteen years. These organisms were obtained from cultures: *Staphylococcus albus*, *Streptococcus hemolyticus*, and *Micrococcus catarrhalis*.

Individual immunity to certain bacteria and the virulence of the organisms present unquestionably play an important part in the accompanying infections. These factors, however, are difficult to measure, and to conclude that the differences in the varying degrees of reaction are due entirely to the bacteria present would be assuming too much without adequate proof.

The question of drainage and of the ability on the part of the patient to rid himself of secretions loaded with bacteria is an important consideration. This is best exemplified by the following cases. A child aged four years aspirated a peanut which was removed two days later. The left main bronchus was completely occluded and the child was very ill. Cultures from the bronchial secretions contained the *Pneumococcus*, *Micrococcus tetragenus*, *Bacillus influenzae*, and *Micrococcus catarrhalis*. In the second case a child aged three years aspirated a peanut and it lodged in the trachea. It was removed after three days sojourn in this location, and these organisms were isolated from the secretions: *Streptococcus hemolyticus* and *Micrococcus catarrhalis*. This child had a very mild reaction, although a small amount of the pure culture of the *Streptococcus hemolyticus* obtained from the patient and injected into a white mouse killed it within a few hours.

In estimating the relative importance of the bacteria isolated from any given case it is important to consider all of the other factors bearing on the case such as the type of the foreign body, the age of the patient, the length of sojourn of the object in the tracheobronchial tree, and the degree of obstruction to drainage. To attach too much significance to the isolation of any one organism is apt to lead one astray. It seems likely that, other things being equal, any one or any combination of pathogens which may be present can give rise to any grade of reaction, from the very mildest to one which will rapidly prove fatal.

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## BRONCHOSCOPY FOR DISEASE

**Indications for Bronchoscopy in Disease Other Than That Due to Foreign Body.**—The indications are so many that they will be considered under separate headings. The most important thing is to refrain from doing a bronchoscopy when it is contraindicated. If this be done, bronchoscopy may be considered indicated in almost any case of pulmonary disease. It is a harmless procedure and may contribute largely to the diagnosis in many unexpected ways. At the Bronchoscopic Clinic we rely largely on the broad viewpoint of the internist for guidance. We have, therefore, requested Dr. Elmer H. Funk to present the matter as viewed by the internist.

**Contraindications to Bronchoscopy in Other Than Foreign Body Cases** (By Elmer H. Funk).—For a skilled bronchoscopy without a general anesthetic the contraindications are few. Fortunately general anesthesia is quite unnecessary.

As a *diagnostic method* in pulmonary disease bronchoscopy is contraindicated in the following five classes of cases: (1) Any patient, until after a careful history has been elicited and a complete physical examination, a Roentgen-ray examination, and proper laboratory studies have been made. (2) In the presence of aortic aneurysm. (3) If there has been a recent hemorrhage. This latter, however, must be considered individually, since in some patients hemorrhage was stopped by the direct treatment through the bronchoscope. (4) In the presence of active tuberculosis. Here again the study of the particular case may develop special indications for the performance of bronchoscopy. Diagnostic bronchoscopy may be advisable to exclude neoplasm or other lesions. (5) In moribund patients. Bronchoscopists have been asked to help hopelessly ill patients.

As a *therapeutic procedure*, in other than foreign body cases, the contraindications to bronchoscopy are better understood if one recalls that the ideal indication is to facilitate drainage of localized lung suppuration when such a lesion is near to and communicating with the tracheobronchial tree. Localized lung abscess and localized bronchiectasis (unassociated with active tuberculosis) come within this category. Recent cases do better than long-standing cases for obvious reasons; though this constitutes no contraindication. The contraindications to bronchoscopy in lung suppuration may be summarized as follows:

1. Purulent pneumonia or that suppurative lung lesion like purulent pneumonia which precedes the formation of an abscess (we are not speaking of foreign body cases).

2. Very extensive multiple abscess formation.

3. Extensive bronchiectasis, that is, involving the whole lung or greater part of both lungs.

4. In localized abscess near the periphery of the lung, which is unlikely to be drained satisfactorily through the tracheobronchial tubular system, and which can readily be approached by an external surgical operation.

5. Bronchoscopy may be considered as contraindicated in the localized bronchiectasis associated with the marked distortion of structures associated with extensive fibrosis; if it be done with the expectation of absolute cure. If it be done with the hope of giving relief to the annoying, offensive, and fetid expectoration and of improving the general condition by lessening the toxemia (which results when the bronchiectatic area is kept clean),

then there is no contraindication in bronchiectasis, even of long standing. Such cases frequently approach a clinical recovery under bronchoscopic treatment.

6. Bronchoscopy is contraindicated in the presence of marked cardiac weakness. This condition, involving especially the right heart, is not uncommon in long-standing cases of pulmonary fibrosis and bronchiectasis.

7. Bronchoscopy is contraindicated in case of a serious complication, obvious or suspected, in a patient whose pulmonary lesion would otherwise indicate its use. Several examples from experience may be cited: (a) A patient with chronic abscess who before bronchoscopy had developed pneumonia in the opposite side. (b) A patient with localized bronchiectasis in the right lower lobe had had headache with fever for several weeks; under observation it became evident that he was developing a brain abscess,



Fig. 640.—From a photomicrograph showing carcinomatous histologic structure in a bronchoscopically removed specimen, confirming a bronchoscopic diagnosis of cancer of the lung in a woman, aged thirty-eight, supposed to be tuberculous. Histologic diagnosis by Dr. Baxter L. Crawford.

from which he eventually died. (c) Two patients with associated pleural lesions, one an empyema and the other a pyopneumothorax. (d) A patient whose symptoms and signs suggested a perforative empyema. The indications for bronchoscopy outnumber the contraindications; the procedure will be more widely used when all medical men realize its safety in skilled hands. A good working rule is this: When in doubt, if there are no clear contraindications, give the patient the benefit of a bronchoscopic examination. In a large number of carefully selected cases of pulmonary disease I have never seen an untoward effect, either as a result of the procedure *per se*, the insufflation of bismuth, the introduction of medicaments, the removal of tissue for biopsy, the aspiration of secretions, etc. This personal experience of an internist attests the value of skilled bronchoscopy in suitably selected cases.

**Bronchoscopy for Diagnosis.**—The recognition of diseased conditions

rests fundamentally on an eye and perceptive faculties trained on the normal, so that any departure therefrom is quickly recognized. The appearances of many conditions are characteristic. In other cases removal of specimens of secretions or tissue is essential for diagnosis. Bronchoscopy is the only means by which sputum can be obtained in children too young to expectorate, and the only way uncontaminated specimens can be obtained from the bronchi in patients of any age. In some cases bronchoscopic pneumonography will give invaluable diagnostic information.

**Bronchoscopic Diagnosis of Cancer of the Lung.**—Here we have to deal with a mild, slowly metastasizing, relatively benign disease.<sup>13</sup> Only an early diagnosis is required to enable the surgeon to obtain a good percentage of cures. The only way to make the diagnosis early is by bronchoscopy and histologic confirmation (Fig. 640); this is easily and safely obtained by the endoscopic removal of a specimen with the longer form of the forceps (K10, Fig. 556). Metastases cannot travel far in eighteen hours, which is all that is required for histological examination. Except for 1 case of hemorrhage, no complication has followed the taking of a specimen

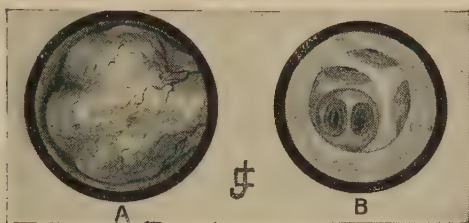


Fig. 641.—Endobronchial obstructing endothelial tumor in a man, aged thirty-five, who complained of coughing, wheezing, and “a sensation as of a ball-valve shutting off the breath, sometimes on inspiration and at other times on expiration.” B, Bronchoscopic view down right main bronchus. A, Tumor presenting itself in its self-made bronchial enlargement, when the bronchoscopic tube mouth reached the location at which the view B should appear. The tumor was removed with forceps through a bronchoscope passed through the mouth. The patient was free from symptoms at the end of three weeks and remained perfectly well twelve years later.<sup>23</sup>

in over 400 cases. It is a deplorable fact that cancer of the lung is usually treated for a considerable time, in some instances for several years, under an erroneous diagnosis.<sup>1</sup>

A number of cures of endobronchial malignant growths have followed bronchoscopic removal by different bronchoscopists (Fig. 641). The number of cases in which this should be attempted will be relatively few. Success is possible only in the earliest stages of the disease; but the cases of cure are of the utmost importance as clinical evidence of the relatively mild degree of malignancy and slowly metastasizing character of endobronchial carcinoma and endothelioma. The bronchoscope will contribute to cure more often as a means of early diagnosis than as a means of removal. In many cases at the Bronchoscopic Clinic we have been much gratified to see malignant growths of the lung arrested and the patient remain in good health for as long as four years after deep Roentgen-ray treatment. These apparent cures have been so remarkable that even we ourselves would have questioned the diagnosis had we not had the histologic evidence afforded by a bronchoscopically removed specimen.

**Bronchoscopic Treatment of Disease.**—Endobronchial medication is useful in chronic inflammatory conditions, and vaccines prepared from uncontaminated, bronchoscopically removed specimens have been useful adjuncts to medical treatment of suppurative conditions; but the outstanding feature of bronchoscopic treatment is the fact that purulent stagnation can be successfully combated by bronchoscopic aspiration in a certain proportion of cases. In most cases of inspired foreign body no treatment other than bronchoscopic removal is indicated.

**Bronchial Obstruction.**—Observations in thousands of cases at the Bronchoscopic Clinic have proved beyond all question that the fundamental etiologic factor in all suppurative diseases of the lung is bronchial or

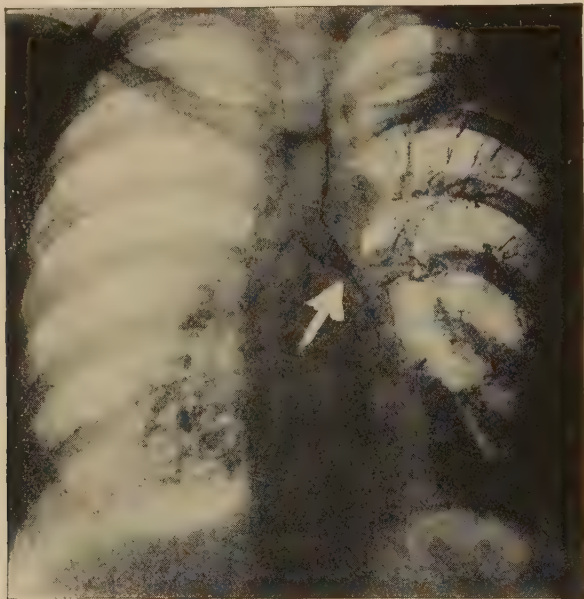


Fig. 642.—Bronchoscopic pneumonogram (by Dr. Gabriel Tucker) of a woman aged fifty-one showing cicatricial stenosis of a bronchus secondary to a suppurating tuberculous mediastinal adenopathy. The suppurative tuberculous process had healed four years previously under the care of Dr. Lawrence Flick. Patient remained well until acute influenzal infection six months prior to admission. Impaired drainage below the stricture favored residual suppuration (non-tuberculous). Favorable results followed the peroral bronchoscopic dilatation of the stricture and aspiration of stagnating pus by Dr. Gabriel Tucker. Patient of Dr. Isabelle Balfe and Dr. Max Goepf. Film made by Dr. H. K. Pancoast.

bronchiolar obstruction. So long as free ventilation and drainage are maintained the *normal defensive powers of the lung* will annihilate almost any infection.<sup>5</sup> Any form of obstruction to the ventilation and normal upward drainage by ciliary and beehee action will certainly end in chronic suppuration. The diagnosis as to the nature of the obstruction can be made by bronchoscopy through the mouth, without anesthesia, general or local, in a few minutes, and usually with great accuracy. When a bronchus is obstructed by a foreign body, a stricture (Fig. 642) or web (Fig. 646), bronchoscopy is the only method of treatment worthy of a moment's consideration. It is the method of first choice in cases of obstruction due to thick pus (Fig. 643), granulation tissue, membrane or stricture (Fig.



## PLATE XXVII

### BRONCHOSCOPIC VIEWS OF NEOPLASTIC DISEASES

Photoprocess reproductions of oil-color drawings from life by Chevalier Jackson.

1. Bronchoscopic view as seen in a recumbent normal person, when the tube is a few centimeters proximal to the bifurcation of the trachea. The carina is seen as normally thin, sharp, and whitish, and it is located a little to the left of the median line. The left bronchial orifice appears somewhat as a crescent; the right bronchus, which is anatomically the continuation of the trachea, is seen extending into the depths of the right lung. Anteriorly a small portion of the middle-lobe bronchus is visible. In addition to normal form and color, normal movements, beautiful, wavy, flexible, rhythmic respiratory, and more abrupt pulsatory movements are noted in the normal. It is of the utmost importance that these normal appearances be studied.

2. Broad carina in a man, aged forty-eight, indicating infiltration of the glands beneath the bifurcation (compare with 1). The glands might have been inflammatory, tuberculous or malignant, but the accompanying fixation, rigidity and other evidences of infiltration warranted a diagnosis of cancerous metastases, which was confirmed by the bronchoscopic finding of a malignant lesion in the right bronchus.

3. Deformity and displacement of the carina backward to the right by a large cancerous infiltration, not yet ulcerative, which occludes the left bronchial orifice.

4. Cancerous nodules occluding the right main bronchus in a man aged forty-eight. The erroneous diagnosis of asthma had been made prior to the patient's coming under the care of Dr. E. H. Funk, who suspected cancer and requested a diagnostic bronchoscopy.

5. Fungating carcinoma of the trachea with a scabbard-shaped lumen due to metastatic infiltration. Histological examination of a bronchoscopically removed specimen showed the growth to be a carcinoma. The patient, a woman, aged forty-eight, had been treated for more than a year for asthma because of paroxysmal coughing and wheezing.

6. Cancerous nodules in the right bronchus of a man aged forty-two. The bronchoscopic diagnosis was confirmed by the histologic report of adenocarcinoma made on a bronchoscopically removed specimen.

7. Bronchoscopic view of an endobronchial carcinoma diagnosed bronchoscopically, and verified histologically by an examination of a specimen removed through the bronchoscope. Exsanguinating hemorrhages ceased after bronchoscopic insufflation of bismuth subcarbonate. Deep Roentgen-ray therapy arrested progress of the growth. The patient is in good general condition after four years. Prior to admission the patient was erroneously supposed to have tuberculosis.

8. Bronchoscopic appearances of an endobronchial carcinoma in a woman, aged thirty-eight. Histologic examination of a bronchoscopically removed specimen verified the bronchoscopic diagnosis. The surgeon deemed the growth and metastases inoperable. Deep Roentgen-ray therapy arrested the progress of the growth, which has remained stationary for four years.

9. Fibropapilloma with a long slender pedicle attached to the wall of the right bronchus below the level of the right upper-lobe bronchus, discovered at diagnostic bronchoscopy in a woman aged forty-four. The symptom of paroxysmal coughing, wheezing, and bubbling mucopurulent secretion led to an erroneous diagnosis of asthma. The bronchoscopic diagnosis was confirmed by biopsy. Cure of the symptoms followed peroral bronchoscopic removal. The patient was free from recurrence two years later.

10. The fungations seen at the right side of the lower-lobe bronchial orifice might be either exuberant granulations or malignant fungations. The bronchoscopic diagnosis of cancer was based on the thickened infiltrated spur between the lower and middle-lobe bronchial orifices. The latter is uppermost in the illustration. The diagnosis of cancer was verified by histologic examination of a specimen removed at diagnostic bronchoscopy.

PLATE XXVII



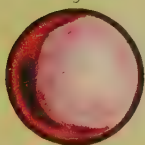
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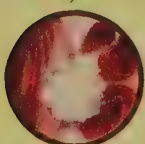
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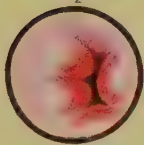


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Chevalier Jackson



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642). Except in cases of aneurysmal compression and malignant involvement, bronchoscopic dilatation or removal is practicable, and usually it is indicated. Even in cancer (*q. v.*) remarkable results have been accomplished. We believe that the toxemia of pulmonary tuberculosis is due to mechanical obstructive conditions relievable by bronchoscopic methods.

**Cicatricial stenosis of a bronchus**, whether due to traumatic, inflammatory, tuberculous or syphilitic disease, is quite amenable to bronchoscopic dilatation. Bronchial stenosis is an exceedingly rare sequel of foreign body even after years of lodgment and suppuration; almost all cases recover after bronchoscopic removal of the foreign body and fresh air treatment. About 1 per cent. require dilatation and aspiration for a time.

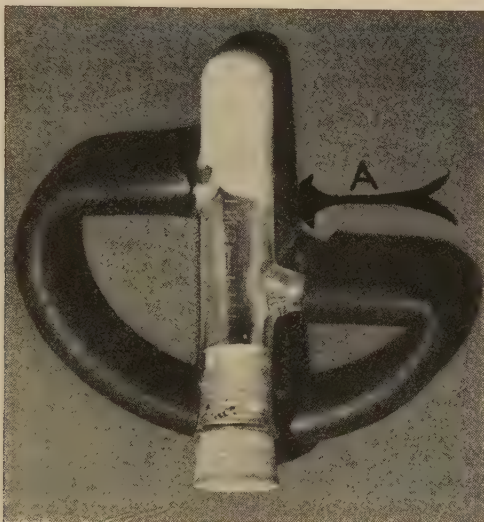


Fig. 643.—Illustration of a fundamental factor in suppurative diseases of the lung. The collecting tube of the bronchoscopic aspirator has been inverted, but the pus is so thick that it will not run down. The patient had been coughing for hours, but could not get this thicker part of the pus up. Thick, gummy, tenacious pus like this clogs the cilia to the point of total inefficiency. Stagnation of pus is the largest factor annihilating the defensive power of the lung. Bronchoscopic aspiration of the obstructive pus and secretions restores the defensive power of the lung. This kind of pus will almost always result in progressively increasing pathological changes in the lung unless removed bronchoscopically. Dr. John A. Kolmer found that this consistency was due to the presence of large amounts of fibrinogen probably of hematogenous origin.<sup>22</sup>

**Compression Stenosis of the Trachea and Bronchi.**—Compression of the trachea is most commonly caused by goiter (substernal or cervical), aneurysm, malignant neoplasm, and in children, by enlarged thymus. Less frequently, enlarged mediastinal tuberculous, leukemic, luetic or Hodgkin's glands compress the airway. The left bronchus may be stenosed by pressure from a hypertrophied cardiac auricle. Compression stenosis of the trachea associated with pulmonary emphysema accounts for the dyspnea during attacks of coughing.

The endoscopic picture of compression stenosis is that of an elliptical or scabbard-shaped lumen when the bronchus is at rest or during inspiration. Concentric funnel-like compression stenosis, while rare, may be produced by annular growths. As a rule, however, it is obvious endoscopically that the bronchial wall is dinged in and not involved in the process as it is

in malignant and post-inflammatory conditions. Any kind of bronchial stenosis leads to suppuration from stagnation of secretions.

**Treatment of Compression Stenosis of the Trachea.**—If the thymus be at fault, rapid amelioration of symptoms follows Roentgen-ray or radium therapy. Tracheotomy and the insertion of the long cane-shaped cannula (Fig. 540) past the compressed area is required in the cases caused by conditions less amenable to treatment than thymic enlargement. Permanent cure depends upon the removability of the compressive mass. Should the bronchi be so compressed by a benign condition as to prevent escape of secretions from the subjacent air passages, bronchial intubation tubes may be inserted, and, if necessary, worn constantly. They should be removed weekly for cleansing and oftener if obstructed.

**Bronchoscopic Aspiration.**—This can be carried out by bronchoscopy through the mouth in a few minutes without any anesthetic, general or local. A sedative may be used or not, as preferred. The aspirating bronchoscope has an aspirating canal in the wall of the tube; but more often, in children, especially, the independent aspirating tube is preferred. Often both are used. The independent aspirating tube (V2, Fig. 556) can be inserted into small bronchi and fistulae, even at the periphery of the lung if necessary. It can be inserted through the laryngoscope without using a bronchoscope if deemed desirable. Bronchoscopic aspiration is of great aid in the treatment of all suppurative diseases of the lung. It is the most useful adjunct to medical treatment in asthma, chronic bronchitis, bronchiectasis, pulmonary abscess, and pneumonia, lobar as well as bronchopneumonic. We have seen the pneumonic crisis promptly precipitated by bronchoscopic aspiration as first suggested by J. E. Sweet of Cornell University.<sup>18</sup> In our cases cocaine was sprayed in preliminary to aspiration.

**Abolition of the Cough Reflex.**—Quite a number of patients coming to the Bronchoscopic Clinic have manifested total absence of the cough reflex, permitting bronchoscopy without exciting any cough, notwithstanding no anesthetic, general or local, was used.<sup>17, 19, 20</sup> Though the first cases of this kind I saw were reported seventeen years ago, no satisfactory explanation has been offered. *Drowning of the patient* in his own secretions is inevitable if bronchoscopic aspiration be not promptly resorted to. In some cases of absence of the cough reflex the secretions are tough and *atelectasis* of a lung occurs from absorption of air below the obstruction. This is the condition in the so-called *postoperative* massive collapse.<sup>14</sup> In other cases the secretions are dry and require removal with the bronchoscopic forceps.<sup>17</sup> The abolition of the cough reflex has occurred in foreign body cases, in arachidic and other forms of vegetal bronchitis, and in acute purulent bronchitis, bronchopneumonia, diphtheria, and the exanthemata.

**Stagnation.**—The natural physiological drainage of the lung is by cough and ciliary action. Our investigations at the Bronchoscopic Clinic indicate that when an acute infective process has been followed by a subacute condition, and this in turn is in process of drifting into chronic suppuration, there is stagnation of pus and secretions in the affected portion of the lung. When in such a case, after the patient has coughed and coughed until he can get no more sputum up, we go down with a bronchoscope and find foul, discolored pus, we feel justified in saying there is stagnation. In spite of cough, the pus has remained long enough for the saprophytes to decompose it, and for the chromogenic bacteria to discolor it. When we

relieve this stagnation by bronchoscopic aspiration, the odor disappears and usually a prompt cure follows. We feel that in these subacute cases, especially, bronchoscopic aspiration will be a large factor in the prevention of chronic bronchitis, bronchiectasis, and chronic abscess.

**Chronic Pulmonary Abscess.**—Clinically, and to a certain extent pathologically, sharply defined classifications of non-tuberculous chronic suppurative diseases of the lung are unsatisfactory because they leave such a large group of cases in the mixed or undefined class. The main points in relation to bronchoscopy are three, namely:

1. The endobronchial conditions in relation to drainage of the suppurative foci can be studied through the mouth, without general anesthesia, by a relatively minor procedure.<sup>13</sup>

2. Suppurative areas, large or small, can be efficiently emptied through the mouth in a few minutes, without general anesthesia; and this emptying can be repeated as often as necessary.

3. By taking the load off the drowned cilia for a time, these and the other factors in the spontaneous mechanism of drainage are enabled to recover, the efficiency of spontaneous drainage is re-established, and the defensive power of the lung is restored.

In the cases usually classified as chronic pulmonary abscess there is a group in which a large portion of the lung is a mass of fibrous tissue with devious fistulæ leading to one or a number of pockets. The presence of this condition can be determined in the following way:

1. Clearing away the granulations, débris, pus, and secretions from the fistulæ by bronchoscopic means, especially the vertebrated "around the corner" aspirating tubes, supplemented so far as necessary by the bronchoscopic forceps and dilators.

2. The fistulæ are injected with *lipiodol* under guidance of the eye.

This kind of *pneumonography* cannot be done in any way other than bronchoscopically, because the *lipiodol* cannot enter a fistula until the obstruction is cleared from its lumen. A false impression, or at best an uncertain result, would be obtained by pneumonograms made otherwise than bronchoscopically. With this pneumonogram as a basis, a consultation is held by the internist, the surgeon, the roentgenologist, and the bronchoscopist to determine the best plan of procedure for the patient's best interests. The technic of pneumonography is given in another section of this book. If it is found that any or all of the abscesses require external drainage, the surgeon has accurate mapping in two planes to guide him. If the case seems to be one for bronchoscopic aspiration, the fistulæ can be kept open bronchoscopically; but under no circumstances is the case regarded as one for any kind of treatment independent of the internist. He is the balance-wheel that equalizes the separate impulses of those who specialize. The broad viewpoint of the internist is especially necessary to the bronchoscopist, who is apt to see things narrowly through a small tube, metaphorically as well as actually. Bronchoscopic aspiration and bronchoscopic clearing of obstructed drainage channels in or leading to the tracheobronchial tree often reverses an adverse trend in the patient's condition and starts him toward recovery; in other words, while general medical care has held the patient's condition stationary, the supplementary bronchoscopic drainage has been the turning factor. In any case, rest is essential. Unless watched and controlled, the patient will take advantage

of the improvement in his general condition obtained by bronchoscopic drainage to increase his activities, instead of building up a reserve to increase his resistance. Rest in bed outdoors sixteen to twenty hours should be insisted upon. Diet should be carefully regulated. Free elimination should be regularly, not intermittently, maintained. All sedatives should be forbidden, not only because of their effect in depressing nutrition, but because they restrain cough. "The cough reflex is the watchdog of the lungs" (Chevalier Jackson). Antituberculous drug the watchdog to sleep.<sup>20</sup>

**Pulmonary Abscess Due to Foreign Body.**—The chief cause of pulmonary abscess in foreign body cases is the overlooking of the foreign body by the general physician. If the foreign body is removed within a few days, abscess practically never occurs. Foreign bodies in the lung are overlooked not so much because of inability to make the diagnosis as because foreign body as a diagnostic possibility is rarely considered.<sup>5, 6</sup>

**Bronchiectasis,** if dependent on foreign body, will almost always get well without treatment after the removal of the foreign body. When bronchiectasis is due to other causes, such as disease of the nasal accessory sinuses, nearly all cases can be cured, if seen very early, by a series of bronchoscopic aspirations. In cases of long standing, bronchoscopic aspirations once or twice a week will cause the odor to disappear by preventing stagnation. After a time the normal expulsive powers of the lung (cough,



Fig. 644.—A, A broncholith from bronchiectatic purulent accumulation removed by peroral bronchoscopy under local anesthesia, in case of a man aged twenty years, with long-standing bronchiectasis. B, Inorganic material from the same pus. The nucleus of the broncholith, A, was of similar character to that of the inorganic material, B.

bechic compression, ciliary action) recuperate so that the patient goes on to recovery, in a very satisfactory percentage of the cases. Thomas McCrae, who has, from the broadest viewpoint of internal medicine, seen so much of bronchoscopy, states in effect that "bronchoscopy is of great value in the treatment of bronchiectasis if used early; but *early* should be stressed.<sup>10</sup> This means prompt diagnosis, which is very rarely done."

**Bronchiectasis Due to Foreign Body.**—In the statistics of the Bronchoscopic Clinic,<sup>1, 2, 3</sup> there are quite a number of cases showing that bronchiectasis due to foreign body practically always gets well spontaneously after removal of the foreign body.

**Broncholithiasis.**—Lung stones have been encountered and removed in a few cases of suppurative diseases of the lung at the Bronchoscopic Clinic. In one case the stone was a true calculus, the calcareous material having been deposited around a nucleus of silica (Fig. 644). It seemed probable that a broken down area of silicosis had been the initial factor. In another case suppurative processes had discharged a broken down calcareous lymph-node into a bronchus.

**Chronic Purulent Bronchitis.**—Bronchoscopic aspiration combined with treatment by vaccines prepared from bronchoscopically removed uncontaminated specimens has yielded a high percentage of cures. In addition to the bronchoscopic aspiration general care by the internist or pediatrician is maintained in all of our patients.<sup>4</sup>

**Acute Pulmonary Abscess.**—Observations in a relatively large number of cases at the Bronchoscopic Clinic have convinced me that there is at times a temporary failure in the normal defensive power of the lung, during which infections gain a foothold. Over and over again we have seen in patients with acute suppurative disease of the lung with cough, expectoration, chilliness, fever to 103° F. or over, a sudden arrest of all the symptoms after a few bronchoscopic aspirations. The fact that in a patient getting progressively worse the tide is promptly turned by the bronchoscopic aspiration, often by the very first aspiration, is very striking; and the additional fact that this has occurred hundreds of times, and in the hands of eight or nine different members of our personnel, eliminates the possibility of mere coincidence. The fact that the same results have been obtained in other clinics by Lynah, Yankauer, Kernan, Orton, Zinn, Ballon, Imperatori, and others places the matter on the plane of an established clinical fact.

**Posttonsillectomic Pulmonary Suppuration.**—The frequency with which tonsillar disease, past or present, is associated with bronchiectasis and sinus disease as apparently the primary focus, justifies the inference that in many cases of posttonsillectomic pulmonary suppuration the patient would have had suppurative disease of the lung eventually even if the tonsils had not been removed. Undoubtedly this complication has been frequently erroneously diagnosed as postoperative pneumonia.

*Prophylaxis* during tonsillectomy is elsewhere herein considered. Prophylaxis of abscess formation, after suppuration is started, is a matter of prompt resort to bronchoscopic aspiration.

**Posttonsillectomic Pulmonary Abscess.**—The phases of this subject that concern the tonsillectomist are discussed by George Fetterolf, in connection with complications of tonsillectomy. Only the bronchoscopic phases will be considered here. We have studied many of these patients in the Bronchoscopic Clinic in consultation with the internists and surgeons. The results showed that even when a posttonsillectomic abscess had been allowed to become chronic, in many cases it was more amenable to bronchoscopic treatment than abscesses of other etiology. But it is in the early stages that the most phenomenal results are obtained. There is nothing in the realm of the treatment of suppurative disease of the lung that is more brilliantly successful than bronchoscopic aspiration in the early stages of this very serious disease. But the patients must be seen in the first few weeks to obtain these brilliant results; later, after suppuration is well established, good results are obtained, but time and many treatments are required to obtain them and the percentage of cures is not so high; many cases will eventually require external operation. There seems to be something lacking in the *normal defensive powers of the lung* that is quickly restored by the bronchoscopic aspiration in the early stages (Fig. 645). When a patient comes in, in the early stages of posttonsillectomic suppuration, showing a daily rise of temperature to 101° or 102° F. we confidently expect to see the temperature drop and remain at a lower range until a cure follows; we are rarely disappointed. The results have been obtained by so many different bronchoscopists as to establish this clinical fact beyond question. There are, of course, certain fulminating cases, probably embolic in origin, in which the patient is so seriously ill that he is doomed from the onset. It is useless to attempt bronchoscopy in such patients. The technic

of bronchoscopic treatment in the curable cases is quite simple. No endobronchial irrigation is used; we consider it contraindicated in all kinds of acute cases. After the bronchoscope has located the diseased area the

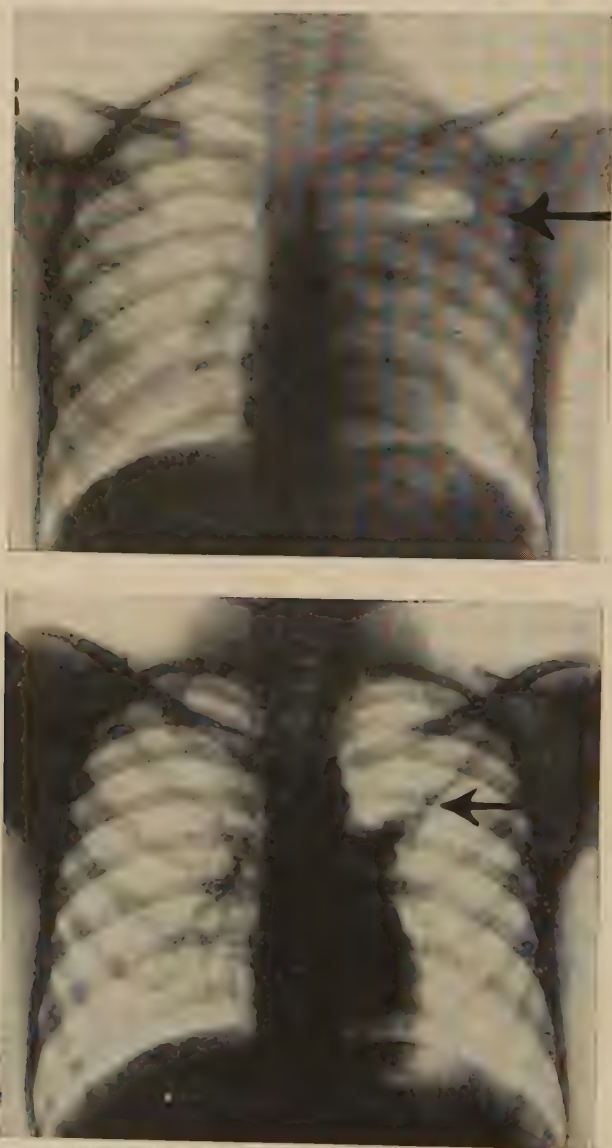


Fig. 645.—Upper illustration shows fluid level in a pulmonary abscess developed three weeks after tonsillectomy, in a boy aged nine years. Cough, expectoration, and all other symptoms disappeared after ten bronchoscopic aspirations by Dr. Gabriel Tucker. Lower illustration shows film taken after disappearance of symptoms. Six months later patient was still entirely well. (Films by Dr. H. K. Pancoast.)

"velvet-eyed" aspirating tube is inserted and the pus withdrawn by the negative pressure pump. Local anesthesia is used in adults; no anesthesia, general or local, in children. The aspirations are repeated once or twice a week.

**Asthma.**—Bronchoscopy for diagnosis and treatment should be considered in every case of asthma, not only because of the complicating secondary infections and the stagnation so efficiently treated with the bronchoscope (Moore<sup>9</sup>), but also because of the fact that objective endobronchial examination by means of the bronchoscope has shown that many of the symptoms of asthma may be present in patients who have not and never did have asthma. "*All is not asthma that wheezes*" (Chevalier Jackson) is an aphorism that should be inculcated into the mind of every medical student. Wheezing, in some cases coming on suddenly at night, and in most cases more or less paroxysmal, has been bronchoscopically discovered to occur in endobronchial stenosis of the following kinds: edematous, exudative, cicatricial, neoplastic (benign and malignant), compressive, aneurysmal, goitrous, paralytic (laryngeal), thymic, congenital. In the experience of the Bronchoscopic Clinic, "stenosis of the trachea and bronchi has so often been mistaken for asthma that it would seem inadvisable to make a diagnosis of asthma without exclusion of organic stenosis" (Chevalier Jackson<sup>10</sup>). Wheezing, coming on or becoming worse during the night, and more or less paroxysmal in character, is quite a common symptom in foreign body cases. Forty-eight children whose symptoms were entirely due to a foreign body and its secondary suppuration have come to the Clinic after the diagnosis of bronchial asthma had been made by practitioners elsewhere. In some cases as many as four or five practitioners had concurred in the erroneous diagnosis. Moreover, the foreign bodies were, in 7 of the 48 cases, in the esophagus and not in the lower air passages. Hence, esophagoscopy is also indicated in some cases tentatively diagnosticated asthma. It is the opinion of pediatricians and internists with whom we have worked that the diagnosis of asthma should not be made in a child without excluding foreign body as a diagnostic possibility, by every means at command. Furthermore, it is now recognized that the asthmatoïd wheeze heard at the open mouth is often a very important diagnostic sign of foreign body. Stricture of a bronchus is not infrequently mistaken for asthma. In one patient supposed to have asthma, Dr. Edmond Aucoin found masses of granulomata, the removal of which stopped the wheeze and cured the patient. In addition to the foregoing, we should bear in mind the bronchoscopic discoveries of Moore,<sup>9</sup> that in certain cases of asthma the most obvious pathologic condition is the presence of thick, tenacious secretion, the bronchoscopic removal of which is often curative.

Willis F. Manges, who has studied the asthmatic patients at the Bronchoscopic Clinic from the roentgenologist's viewpoint, has given us his conclusions as follows:

*Roentgen-ray Studies in Asthma* (By Willis F. Manges).—We believe that a diagnosis of asthma is not complete without a Roentgen-ray examination of the chest, sinuses, and teeth, as well as a very careful clinical search for other foci of infection about the upper air passages and mouth. It is certain that infection plays an important role in a very large percentage of cases of bronchial asthma.

In a Roentgen-ray study of the chests of 157 cases, only 25, or approximately 16 per cent, were without evident tissue changes that could be demonstrated roentgenographically. The changes found were classified as follows and found in the percentages given in the table:

		per cent.
1. General peribronchial thickening.....	42	27
2. Chronic lower lobe infection.....	36	23
3. Tuberculosis.....	28	18
4. Thickened root structures.....	19	12
5. Enlarged heart.....	5	3
6. Emphysema.....	2	1
7. No changes.....	25	16

By general peribronchial thickening we mean that the shadows of the bronchi are increased in thickness, density and length throughout the chest. The parenchymal portion of the lung is clear. It has the appearance of a chronic bronchitis.

Chronic lower lobe infection ranges from localized peribronchial thickening or small quantities of exudate to extensive tissue changes, such as bronchiectasis, and large amounts of exudate; also, adhesions to the diaphragm. Some of these cases resemble the proved foreign body infections and I believe that they are primarily due to the aspiration of some foreign substance into the bronchi.

Tuberculosis, though present in 18 per cent., was not considered to have a causal relation with asthma, but in some of the cases there was extensive tuberculosis and in more than one instance even advanced tuberculous lesions had not been recognized clinically.

By thickened root structures we mean that the structures around the roots of the lungs cast more or less massive and dense shadows, at times sharply defined as if due to glandular enlargement; in other instances, a more or less radiating irregular density evidently due to infection.

Enlargement of the heart was noted only in those cases in which there was an increase in size of at least 25 per cent.

Emphysema to a certain extent was present in all of the cases or, at least, in a large portion of them, but in only 2 was the emphysema strikingly in excess of all other signs.

We have not included in this list the many cases of foreign bodies in the air passages that have been treated for asthma because of a wheeze. Foreign bodies should always be suspected when a diagnosis of asthma is made, especially in children.

Chronic infection of the accessory sinuses is present in a very large percentage of the chronic asthmatics, and we believe that in many cases the sinus infection is primary, that lung infection follows, and the patient then becomes either susceptible to asthma or presents physical signs that are mistaken for asthma.

William F. Moore, who has had large experience in the bronchoscopic study of asthma, has given us the following résumé:

*Bronchoscopic Studies of Asthma* (By William F. Moore).—Asthmatic cases can be classified in two major groups, namely, (1) those with a bronchoscopically evident active suppurative tracheo-bronchitis and (2) those with a bronchoscopically evident chronic passive congestion. In the first group are placed all those showing an inflammatory mucosa with a great deal of secretion. In the latter are placed those cases in which the mucosa has assumed a tawny appearance with little or no secretion or those with inspiratory or expiratory collapse of the bronchi, also with little secretion.

Those cases included in the first group will respond best to bronchopneumonic treatment. In the second group factors outside the tracheo-

bronchial tree are responsible, and the infection, if any, is secondary and plays a minor rôle in causing attacks.

*Bacteriology and Vaccines.*—Organisms most often found are the streptococcus viridans, streptococcus hemolyticus or a hemolytic strain of the streptococcus aureus. The viridans is by far the most constant in pure culture, or associated. They may be (a) causative factors or (b) secondary invaders responsible for a primary bronchitis upon which the asthma develops. The organisms recovered show a marked degree of attenuation.

*Roentgen-ray Findings.*—Willis F. Manges has shown in a series of typical cases of asthma a constancy in the evidence of peribronchial thickening and increased density of the root shadows.

*Bronchoscopic Treatment.*—Two means are employed to rid the mucous membranes of the trachea or bronchi of their overload of secretion, namely, sponging where the secretion is thick and tenacious or aspiration, either through a special aspirating bronchoscope or with a separate aspirating tube inserted through and beyond the bronchoscope. Specimens uncontaminated by oral secretions are obtained at the first bronchoscopy for laboratory examination and the making of vaccines. A solution containing 10 drops of a 10 per cent. solution of cocaine and 20 drops of adrenaline solution (1 : 1000) in 1 fluid ounce of normal saline, as a vehicle, is instilled using 5 c.c. in each main stem bronchus. A 3 per cent. solution of ephedrine sulphate in the amount of 10 drops may be used in place of the adrenaline solution (1 : 1000) with equally good results. A 10 per cent. solution of silver nitrate may be applied to localized inflammatory areas.

*Vaccine Therapy.*—Vaccines prepared from sputum often have little value because of contamination of the specimen. Uncontaminated specimens can be obtained directly from the lesion by bronchoscopy, in a few minutes, without any anesthetic, general or local. The patient may thus get all the benefit possible from vaccine methods of treatment.

*Pneumonia.*—Bronchoscopy as an aid to the internist in the treatment of this disease has been urged by J. E. Sweet,<sup>18</sup> and he has presented new and ample clinical and experimental evidence of the value of the method. We have seen the pneumonic crisis precipitated and convalescence started by bronchoscopic aspiration. Cocaine solution was sprayed in preliminary to the introduction of the bronchoscope; whether this contributed to the result or not we do not know.

*Congenital Bronchial Stenosis.*—This condition is probably not so rare as a cause of chronic pulmonary suppuration as the paucity of literature on the subject would seem to indicate.<sup>10</sup> Congenital narrowing of a bronchus does not afford distinctive physical signs or Roentgen-ray evidence of its existence when pathological sequelæ of obstructed drainage have supervened. Sooner or later congenital narrowings of lumen will give trouble when acute infective bronchial disease has swollen the mucosa sufficiently to obturate the narrow lumen. There are only two means of diagnosis, pneumonography and bronchoscopy. There is only one method of treatment worthy of a moment's consideration, namely, bronchoscopic dilatation. This is easily and safely done by a skilful, careful bronchoscopist.

*Congenital Web of the Bronchus.*—While this condition cannot be a common cause of chronic pulmonary suppuration, it is perhaps more common than the literature of the subject would indicate. The only method by which a positive diagnosis of web can be made is with the broncho-

scope. In the 3 cases we have seen at the Bronchoscopic Clinic the patients came because of a suspicion of foreign body. The case illustrated in Fig. 646 is typical.<sup>10</sup>

**Spirochetosis, Vincent's disease, and blastomycosis** are infections to be excluded by the laboratory examination of bronchoscopically removed specimens in every case of suppurative disease of the lung. When these organisms are found in sputum there is no certainty as to their source; but when found in bronchoscopically removed specimens there is no question about it.<sup>24</sup> Vincent's disease is thoroughly discussed elsewhere herein by Professor Moure who is the highest authority on this subject. Blastomycosis of the bronchi is considered in the article on laryngeal blastomycosis.

Many of our cases started after gassing of the patient in the World War. Our experience indicates that bronchospirochetosis and endo-

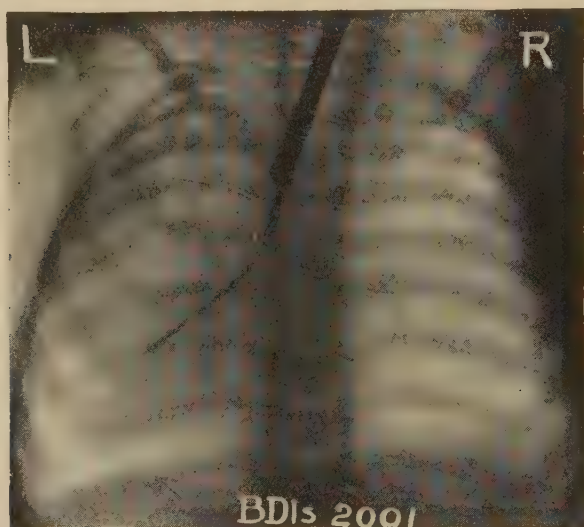


Fig. 646.—Bronchoscopic dilatation of a congenital web-stenosis of the left main bronchial orifice that was the cause of suppurative disease of the left lung. A number of bronchoscopic dilations resulted in complete cure. (Film made by Dr. Willis F. Manges.)

bronchial Vincent's disease are secondary to spirochetal or Vincent's disease of the mouth and pharynx, but the bronchial infection occurs in such a small percentage of cases that to prove the bronchial condition spirochetal requires that the organisms be found in a specimen removed bronchoscopically from the bronchi. The chief symptoms are hemoptysis, cough, expectoration, weight-loss, clubbed fingers, etc. The clinical picture is often identical with that of asthma, in other cases with that of tuberculosis. Dark-field microscopic examination is necessary to find the organisms readily. For prophylaxis, secretion from all mouths should be examined for spirochetes, and the disease when found should be treated locally and systemically. Bronchospirochetosis requires bronchoscopic aspiration once or twice weekly and the local application of a diluted solution of neo-arsphenamine. Salt solution should be used as a diluent; glycerine is irritating. Intravenous injections of neo-arsphenamine are important.

**Benign Tumors of the Bronchi.**—If in the lumen of a bronchus benign growths cause interference with ventilation and drainage; this condition gives rise to suppuration. If obstruction is partial, a valve-like action may result in obstructive emphysema (*q. v.*). If the obstruction is complete, or when it becomes so, there is first *atelectasis*, then suppuration, then *abscess*, sometimes *bronchiectatic abscess*. At the Bronchoscopic Clinic we have found granulomata, infective granulomata (syphilitic, tuberculous, and mycotic), edematous polypi, adenomata, fibromata, lipomata, papillomata, thyroid and amyloid growths. As so clearly stated by Willy Meyer,<sup>11</sup> the bronchoscope is the only means of diagnosis in these cases; fortunately bronchoscopy is quickly and safely accomplished, and its results are certain

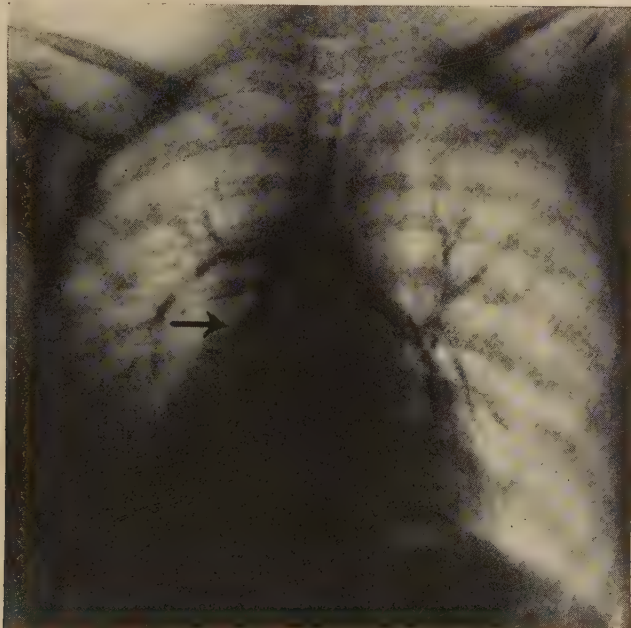


Fig. 647.—Hemostasis of pulmonary hemorrhage by bronchoscopic insufflation of bismuth subnitrate. Exsanguinating hemorrhages due to cancer of the lung ceased immediately and did not recur. After transfusions by Dr. H. H. Jones the patient rallied and the progress of the cancer was arrested for five years by deep Roentgen-ray therapy by Dr. L. P. Larkin. (Film by Dr. Willis F. Manges who suggested the use of bismuth as a hemostatic.)

and conclusive. It should always be preceded by a Roentgen-ray examination. Bronchoscopic removal is curative; recurrences may call for a second removal, but except in the case of papillomata they are rare.

**Hemoptysis.**—The source of blood can be located accurately by bronchoscopy, if the blood is still oozing or has recently ceased, leaving a sign at the spot of the ruptured vessel. If the source of hemorrhage is a growth, a granulating surface, or other lesion it can be found in the larger bronchi, or its location determined if in the peripheral bronchi. Insufflation of bismuth or packing a bronchus<sup>12</sup> may be indicated to arrest hemorrhage (Fig. 647).<sup>4, 13, 14</sup>

In every case of hemoptysis *bronchospirochetosis* and *bronchomycosis* should be considered as a diagnostic possibility. The bronchoscopic aspiration of

secretions uncontaminated by oral organisms is the only certain way of making a diagnosis of Vincent's or other spirochetal infection of the bronchi.

**Scleroma of the Trachea and Bronchi.**—This disease is not uncommon in Poland and the Slavonic countries, and occasionally occurs in this country (see article by S. Shelton Watkins, on Scleroma in Surg., Gynec., and Obstet., p. 47, July, 1921). When it occurs in the bronchi it is as an extension from the trachea and larynx (*q. v.*).

**Acute Infective Laryngotracheitis.**—*Definition and Synonyms.*—Influenza and "*la grippe*" are terms often applied, though rather loosely, to this as well as other conditions that are epidemic and contagious.<sup>15</sup> These terms are relatively new; but the keen observers of a century ago described the condition accurately as "non-membranous croup"; though they did not seem to realize that both it and diphtheria were contagious. The tracheal participation in the respiratory forms of influenza is not accorded the importance it merits. Most of the cough is tracheal in origin in most cases, and very often the lungs will be found uninvolved. The tracheal disease in adults is often treated by the laryngologist in connection with the acute laryngitis as a "common cold." Therefore, perhaps this may be said to be the most common synonym. Perhaps "infectious laryngotracheitis" would be the most appropriate term, since the "influenza" and "*la grippe*" are terms more often applied to the first cases in a pandemic. After partial immunity is established less severe cases, sporadic and epidemic, are seen by those who recognize them every winter for many years after the pandemic.

It is especially the disease as occurring in children that requires consideration here, because direct laryngoscopy is so often necessary for the diagnosis in children.

*Symptomatology.*—The simulation of diphtheria, the croupy cough, the accompanying systemic disturbances, prostration, chilliness, fever, vomiting, sweating, convulsions, etc., seen in severe cases place this disease as properly in the province of the pediatrician; but the intense laryngitis, and especially the subglottic swelling, which often calls for tracheotomy, render a knowledge of this disease of life-saving importance to laryngologists. The ease with which the larynx of any child can be inspected by the direct method without any anesthetic, general or local, has rendered inferential diagnosis as to the lesion in a child with croupy cough unnecessary and in view of its often fatal errors unjustified. The outstanding local symptoms are cough, hoarseness, and pain or discomfort in the larynx or back of the sternum. The cough is usually severe and paroxysmal, with strangling attacks. *Croupy cough* supervenes with the increase in laryngitis, and especially the increase in the subglottic laryngitis. In a few cases cough may be entirely absent.<sup>16</sup>

*Laryngoscopic, Tracheoscopic, and Bronchoscopic Appearances.*—The laryngeal mirror can be used only in older children. Direct laryngoscopic and tracheoscopic appearances have been accurately described and illustrated in colors.<sup>17</sup> The chief characteristics are intense redness becoming somewhat paler in some cases as the swelling supervenes. The mucosa is velvety, bloody spots appear, and the rings are invisible. The subglottic tissues swell out from each side like turbinal bodies in the nose. As they approach, the dyspnea increases. The chink between the swollen bodies becomes smaller and ceases to open on deep inspiration. The secretions are

at first scanty, then more copious, thick, and tenacious; repeated severe coughing and strangling paroxysms are necessary to bring them up. The quantity actually expelled is disappointingly small in proportion to the violence and duration of the paroxysm. Direct examination after such an expectoration will show very little secretion remaining in the trachea; but the intensely inflamed mucosa revealed will explain the etiology of the cough.

*Diagnosis.*—Probably most of the cases are mistaken for diphtheria. This mistake can be prevented only by questioning the diagnosis in every case of diphtheria in which no membrane is visible in the fauces; though antitoxin should not meanwhile be withheld. The most reliable differential diagnostic method is the bacteriological. If the faucial secretions are reported negatively a specimen should be taken directly from the larynx by direct laryngoscopy. An accompanying coryza with secretions negative bacteriologically for diphtheria is an important point. The pandemic character of the disease usually affords evidence of one or more of the protean forms of influenza in other members of the patient's family. The laryngoscopic appearances (*q. v.*) are characteristic. Next to diphtheria, pertussis is the disease most often confused with influenzal laryngotracheitis. Not only symptomatically, but also pathologically, the diseases are much alike. The bacteriology is different, however, and the influenzal disease confers only very temporary immunity. If the child has had whooping-cough, it can be excluded. The presence of influenzal organisms is of diagnostic value, but their absence has no significance, because they are often superseded by mixed infections and probably the influenza may be caused by other organisms. The absence of a pandemic does not exclude influenzal laryngotracheobronchitis, and during a pandemic a child may have a foreign body or diphtheria or whooping-cough; but a consultation with the pediatrician or family physician, one or both of whom should be in charge of such patients anyway, will readily clear up the diagnosis after the laryngologist has contributed a report of the local conditions revealed by his direct laryngoscopic examination. Foreign body, especially one of the vegetal group with the characteristic *vegetal laryngotracheobronchitis*, may very closely simulate an influenzal lesion in the same location. The differentiation will be found in the chapter on "Foreign Bodies in the Air and Food Passages."

*Prognosis.*—If recognized and treated the tracheal and laryngeal disease will recover, if the general disease is not fatal. If the subglottic edema or the absence of the cough reflex is not recognized, the patient may die for want of a tracheotomy. The chronic laryngotracheitis that sometimes follows is quite curable. Unrecognized bronchial obstruction is usually fatal.

*Complications* are bronchopneumonia, pneumonia (usually lobular, very rarely lobar), pulmonary abscess, pericarditis, gastro-intestinal disorders and other well known forms of the protean disease, influenza. Drowning of the patient in his own secretions is an often unrecognized complication.<sup>18</sup> Even more often is fatality due to unrecognized obstruction of both main bronchi by crusts in the early stages, before pus has formed.

*Sequelæ.*—The most common sequel is chronic tracheitis with annoying cough, which persists for weeks or months if not treated. Chronic pulmonary suppuration is a quite common result of the deeper acute invasions; but not of the tracheal disease.

*Treatment.*—The child should be isolated because of the contagious nature of the disease. The air in the patient's room should be fresh and good ventilation should be maintained, but absolute outdoor conditions are not indicated in winter in northern climates. The cough is excited by inhalation of cold air. An abundance of sunshine in the room is very desirable. Alkalinization of the tracheal secretions by the internal administration of sodium bicarbonate at regular intervals is often very efficient. As the alkalinization from one dose wears off the cough will be seen to recur; it will disappear as soon as another dose becomes absorbed. Administration of moderate doses, every three hours, will control the cough. Knowledge of this harmless remedy will obviate the necessity for the always objectionable use of opiates and other sedatives, and antitussives. No attempt should ever be made to apply the sodium bicarbonate in solution directly to the trachea of children. The vapor of compound tincture of benzoin from any open vessel of constantly boiling water in the room is all that is needed in the way of local medication. Dyspnea is due partly to the secretions that cannot get through the glottis, which is not only diminished in area of cross-section, but is unable to co-operate in bronchial expulsion by reason of the impaired motility of the arytenoids. Dyspnea should be watched for and *tracheotomy* done when the signs of *obstructive laryngeal dyspnea* supervene.<sup>4</sup> They are: (1) indrawing at the suprasternal notch; (2) indrawing at the epigastrium. A child with this form of dyspnea soon wears himself out from loss of sleep, because every time he falls asleep he is awakened by the impending asphyxia from loss of the aid of the voluntary muscles of respiration. After a time he will sleep anyhow, and die without a struggle for air. Intubation has been successfully done in these cases by Dr. Howard C. Carpenter, the pediatrician; but unless an expert such as he is in the house constantly, the patient is safer with a tracheotomy. Patients with very tough secretion may require direct laryngoscopic aspiration; and in cases with absence of the cough reflex, the *patient may drown in his own secretions*, unless bronchoscopic or direct laryngoscopic aspiration is carried out as often as necessary. Tracheotomy, if needed for the subglottic swelling that is in itself a hindrance to expulsion of secretion, will permit of the aspirations being carried out by an interne, a competent nurse or even in some cases by a capable mother. The best instrument for this is a soft rubber catheter (child's size) attached to the aspirating pump. For physicians' use the copper tube may be used. If the breathing gets shallow and the breath sounds disappear at the bases and the child seems to be sinking it is certain that there is obstruction of both main bronchi. Quick work with bronchoscope and forceps will be necessary to save the child's life. Hundreds of deaths occur every year from failure to realize the purely mechanical nature of the impending death. The impaired percussion note at the bases and the increased respiratory rate lead to an erroneous diagnosis of pneumonia.

The *sequelar chronic tracheitis* is quickly cured by residence at the seashore, in a warm, moist, sunny region. The ozone, chlorine, and vapor of salt water in the sea air, warmed by sunshine, are so beneficial as to justify almost any sacrifice necessary to obtain them. Patients who do not get well promptly under these conditions are usually those who need alkalies such as sodium bicarbonate in small doses, by mouth, frequently repeated. This alkaline internal treatment is especially necessary in those who cannot

be sent to the seashore. Children do not need local intratracheal treatment, but adults are greatly benefited by intratracheal injection of a solution of monochlorophenol (Eastman) in a strength of 3 grains to the ounce of liquid petrolatum. Minute quantities of camphor and menthol may be added.

**Hemoptysis, Non-pulmonary.**—Blood-spitting is usually of pulmonary origin; sometimes the blood comes from the trachea; less often from the



Fig. 648.—Roentgenogram of a woman aged thirty-two years demonstrating an esophago-tracheal fistula. The rubber cord was passed down the trachea under guidance of the eye with the bronchoscope; after passing the end of the rubber cord through the fistula, the bronchoscope was withdrawn and the esophagoscope was used to find the end of the cord in the esophagus and withdraw it, completing the loop as shown. The fistula was probably due to suppuration, origin uncertain.

hypopharynx or esophagus; still less often it comes from the larynx. Before making endoscopic examinations, however, it is necessary to make careful examinations of the gums, nose, nasopharynx, fauces, palate, lingual tonsil, and in fact the entire mucosa of the air and food passages open to direct inspection. The gums and the large vessels at the base of the tongue should be very closely examined. It is a good plan to have the patient report

at once when blood is noticed, so that if the source is simply a ruptured small superficial vessel there may be some hope of discovering a clot at the point at which bleeding occurred. *Hysteria* and *malingering* should be borne in mind. In over 20 cases at the Bronchoscopic Clinic attempts at simulation of pulmonary hemorrhage were discovered by close watching by the nurses to be produced by slight trauma to the gums between the teeth

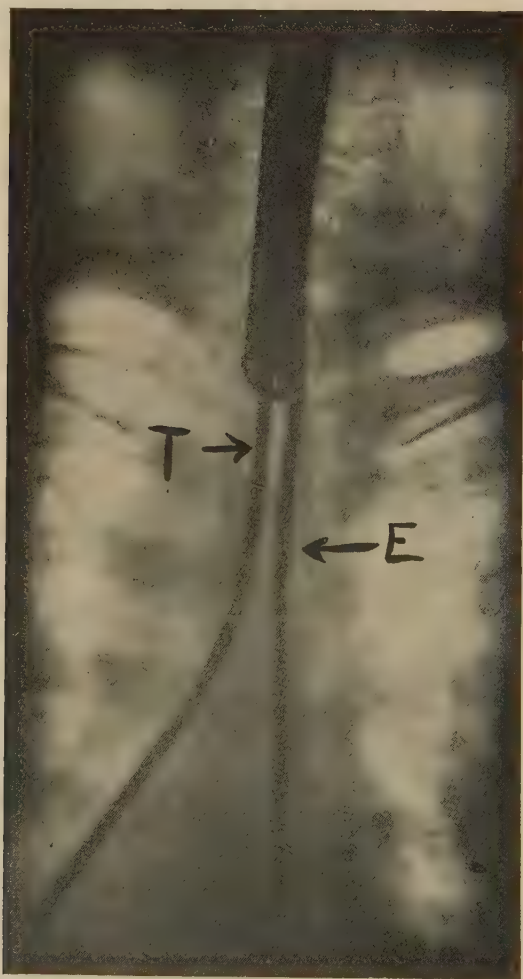


Fig. 649.—Roentgenogram of a woman aged fifty-four years showing an esophago-tracheal fistula. The rubber tube, *T*, has been passed through the esophagoscope and through the fistula, into the trachea and right bronchus. The tube, *E*, had been previously passed through the esophagoscope into the esophagus.

with toothpicks, match sticks, lump sugar, pins, needles, and other implements. Some patients drew the blood from their gums by sucking, that is, creating a negative pressure localized to a certain area of gums or teeth with the tongue and cheek. Serious local diseases are, of course, to be excluded. Cancer, benign growths, varicosities, ulcerative lesions, mycoses, syphilis, lupus, tuberculosis, etc., are to be ruled out. General conditions

other than pulmonary conditions to be considered are gout, purpura, scurvy, pernicious anemia, leukemia, hemophilia, varicella, vicarious menstruation, mercurial and phosphorous poisoning, typhoid fever, yellow fever, and cardiovascular disease.

*Treatment.*—Obviously the etiology and the source of the bleeding furnish the basis for determining the treatment. Bleeding in a plethoric middle-aged individual may be encouraged rather than arrested. Severe hemorrhage from a malignant growth may call for ligation of the external carotid

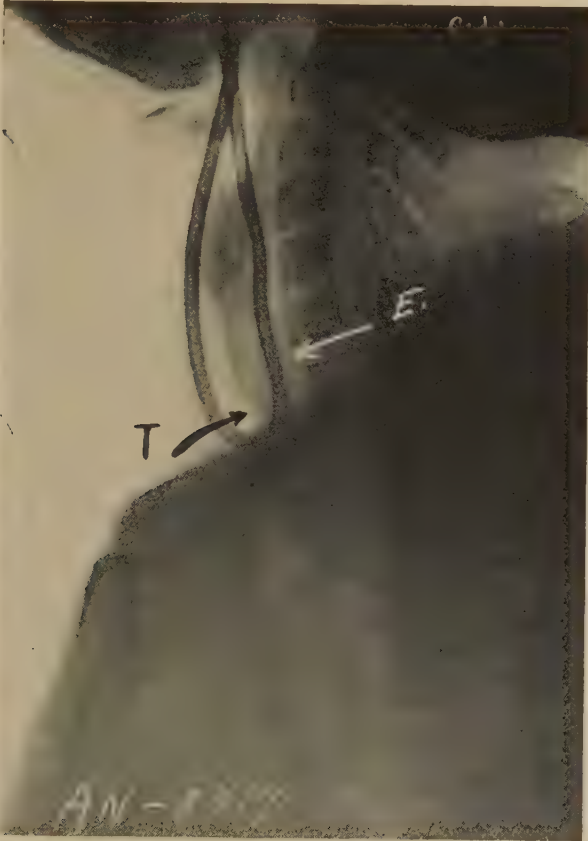


Fig. 650.—Roentgenogram of the same patient as shown in Fig. 648. The rubber tube, *E*, has been pulled into the trachea through the fistula, *T*, and out through the mouth, leaving the bight of the rubber tube in the fistula at *T*.

artery of the corresponding side or both sides. Good local remedies are ice dissolved in the mouth, a touch of strong tannin solution or tincture of perchloride of iron on a swab, adrenaline, and galvanocautery. General remedies are morphine and atropine in full doses hypodermically, calcium lactate, and thromboplastin.

**Tracheocele** is an air-sac communicating with the trachea. It usually inflates and deflates with the plus and minus pressures of the respiratory cycle. In some instances it is congenital; in others pathologic. It is usually symptomless and does not call for treatment. In one of our patients

the left recurrent nerve had been involved and there was a corresponding laryngeal paralysis. We have seen a number of cases of retrosternal goiter movable at will by the patient, and requiring careful palpation to avoid the condition being mistaken for tracheocele.

**Tracheoesophageal Fistula (Esophagotracheal Fistula).**—A fistulous communication between the esophagus and the trachea usually occurs through the party wall between the trachea and esophagus. It may be congenital, ulcerative, or traumatic (Figs. 648–650). The ulcerative form may be due to syphilis, tuberculosis, cancer, blastomycosis, trauma, or prolonged sojourn of a foreign body. Congenital fistulæ are usually fatal; the patients who survive are those with only a small opening, covered by a fold or a thin film of membrane. In one such patient who came to the Bronchoscopic Clinic the film had evidently given way two years before; fatal pulmonary complications had been prevented by feeding with the stomach-tube. When due to foreign body the fistulæ generally heal after removal of the foreign body. Fatal pulmonary complications are certain to follow if the foreign body is not discovered soon enough and removed. Suppuration of a lymph-node is a not uncommon cause of tracheoesophageal fistula.

**Endobronchial Medication.**—The introduction of medication through the bronchoscope is considered of value in the treatment of cases of chronic bronchitis, lung abscess, bronchiectasis, and bronchial asthma. This should always be preceded by removal of the purulent secretion by aspiration with the aspirating bronchoscope and independent aspirating tube. The medications best tolerated are oily preparations of a bland, non-irritating, mildly antiseptic character. These can be used as a topical application or instilled in quantities of 1 to 10 c.c. through the Jackson aspirating tube, using a glass syringe with an intermediate rubber tube. Extensive topical applications to an inflamed mucosa which exhibits a tendency to bleed readily are often associated with mucosal injury; in these cases instillations are preferable. The following have been found useful: monochlorphenol,  $\frac{1}{2}$  to 1 per cent. in liquid petrolatum; a form of oil of cajaput (gomenol), 20 per cent. in liquid petrolatum; argyrol or silvol, 1 per cent. in aqueous solution; a combination of trinitrophenol and Lugol's solution in normal salt solution; mercurochrome in aqueous solution; neo-arsphenamine in normal salt solution; and silver nitrate in aqueous solution. Oil of cajaput (gomenol) is useful, preferably by instillation, in cases of pulmonary suppuration associated with fetor. Neo-arsphenamine, 0.3 gm. in normal saline solution, is of value in bronchial spirochetosis, and is best used as a spray introduced through a bronchoscopic atomizer. Silver nitrate, in 5 or 10 per cent. solution, can be used for exuberant granulations and should be applied on a carefully moistened swab. Granulations are best removed by a dry swab, the lip of the bronchoscope and side-curved forceps. Irrigation should be limited to a portion of the lung. Flooding of a large area of lung either by irrigation or by instillation of a considerable quantity of solution is contraindicated.

**Edematous Tracheobronchitis.**—This is chiefly observed in children. The most frequently encountered form is the epidemic disease to which the name "influenza" has been given (*q. v. supra*). The only noticeable difference between the epidemic and the sporadic cases is in the more general susceptibility to the infective agent, which gives the influenzal form an

appearance of being more virulently infective. Possibly the sporadic form is simply the attack of children not immunized by a previous attack during an epidemic. Aspiration, not irrigation, is indicated.

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#### BRONCHOSCOPIC PNEUMONOGRAPHY

**Definition.**—Pneumonography, or lung-mapping, has for its object the introduction, into the air-passages and pathological cavities and channels, of an inert, radio-opaque substance which will increase the radiographic visibility of these structures. *Bronchoscopic pneumonography* is the term

applied to this procedure as carried out bronchoscopically. The first pneumonogram even made was done by Chevalier Jackson using bismuth subcarbonate by bronchoscopic insufflation.<sup>1</sup>

The substances used for this purpose are either powdered bismuth subcarbonate or iodized oil, 40 per cent.\* Various substances, as barium salts, bismuth in oil, colloidal silver, and aqueous solutions of some of the salts of iodine and bromine, have been tried, but were found impractical.

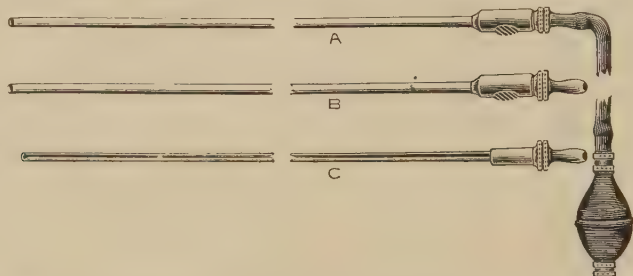


Fig. 651.—The Clerf bronchoscopic powder insufflator used for bronchoscopic introduction of opaque powders in pneumonography. The insufflator (A) consists of a hollow outer tube or cylinder (C) fitted with a removable gutter-shaped carrier (B) which extends throughout the full length of the cylinder (C). The powder carrier, fitted with a cone-shaped end, fits accurately into the sleeve of the outer tube. To fill, the powder is loosely scattered through its entire length. It is then inserted into the cylinder and rotated to dump the powder in a long, loose ridge.

**Instrumentarium.**—In addition to the instruments ordinarily required for diagnostic bronchoscopy, certain special apparatus is necessary for the introduction of the opaque material. If bismuth subcarbonate is to be insufflated, a bronchoscopic powder insufflator is to be used (Fig. 651). If iodized oil is the material to be used, a tube-set for its bronchoscopic instillation is required (Fig. 652).

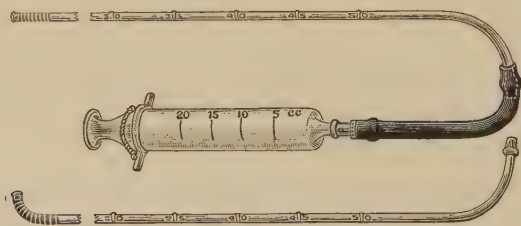


Fig. 652.—Tube-set for instillation of iodized oil, devised by Dr. Gabriel Tucker. The set consists of a straight flexible tipped (Lynah) tube and a curved flexible tipped (Lynah) tube to get around the corner; tubes are graduated at 5-cm. intervals to measure the depth of insertion. A rubber tube is interposed between a Luer syringe connection and the tube so the flexibility is obtained between the tube held by the bronchoscopist and the 20-c.c. oil-containing syringe, operated by the nurse.

When bismuth is to be insufflated in the case of a child, a 5 mm. full lumen bronchoscope should be used in preference to the 5 or 6 mm. standard bronchoscope, as these will not admit the ordinary insufflator tubes; occasionally, a 5 mm. x 45 cm. bronchoscope will be required in the exploration of subdivisions of the lower lobe bronchi of adults.

\* Iodized oil for this purpose was developed experimentally by Forestier and his collaborators (Paris Médicale, May 13, 1922, p. 403; Jour. Med. Française, Vol. XIII, No. 1, June, 1924. It was given the name "Lipiodol" and it remains the most stable, safe, and reliable preparation of iodized oil for pneumonography.—EDITOR.

The bismuth subcarbonate should be chemically pure, dry, and in powder form. Iodized oil should be clear, yellow, and free from precipitate. It should be warmed to slightly above body temperature. If overheated there is danger of liberating iodine.

**Technic.**—Irrespective of the material to be employed in the mapping, it is, first of all, necessary to remove all secretions with a bronchoscope and aspirator to obtain as clear a field as possible. If bismuth subcarbonate is to be used, the bronchoscope is then carried into the bronchus or opposite the orifice of the bronchus to be mapped out, the filled insufflator is introduced through the bronchoscope, and its contents blown



Fig. 653.—A case of marked pulmonary fibrosis involving the left lung, occurring in a girl aged fourteen years. On a pneumonogram made after the bronchoscopic insufflation of bismuth subcarbonate there was observed deviation of the trachea to the left with marked stenosis of the left main bronchus beyond the carina. The cicatricial stenosis of the left bronchus was discovered by diagnostic bronchoscopy. (Film by Dr. W. F. Manges.)

out by compression of the hand bulb. Insufflation is preferably done during inspiration. Slight cough will not interfere with the results. If iodized oil, 40 per cent., is employed, the instillation tube is carried through the bronchoscope into the bronchus or bronchial subdivision corresponding to that portion of the lung to be examined and the oil slowly instilled. There is a distinct advantage in carrying out these procedures with the patient placed on the fluoroscopic table. In the case of iodized oil, it is desirable to utilize the influence of gravity by turning the patient on the side to be examined or by either elevating or lowering the head of the table.

The quantity of material to be used depends upon the results desired.

An ounce of bismuth subcarbonate can be safely used in an adult; usually 2 to 3 drams are sufficient. In adults, as much as 40 c.c. of iodized oil, 40 per cent., have been instilled without untoward effects; 20 c.c. can be considered a safe and adequate quantity for ordinary usage. In children, proper dosage can be computed on the basis of the patient's age.

Cocainization of the tracheal or bronchial mucosa preliminary to the pneumonography, in the case of adults, is desirable. Applications of cocaine in 10 or 20 per cent. solution can be made directly to the mucosa through the bronchoscope, or a 1 or 2 per cent. solution of cocaine can be sprayed

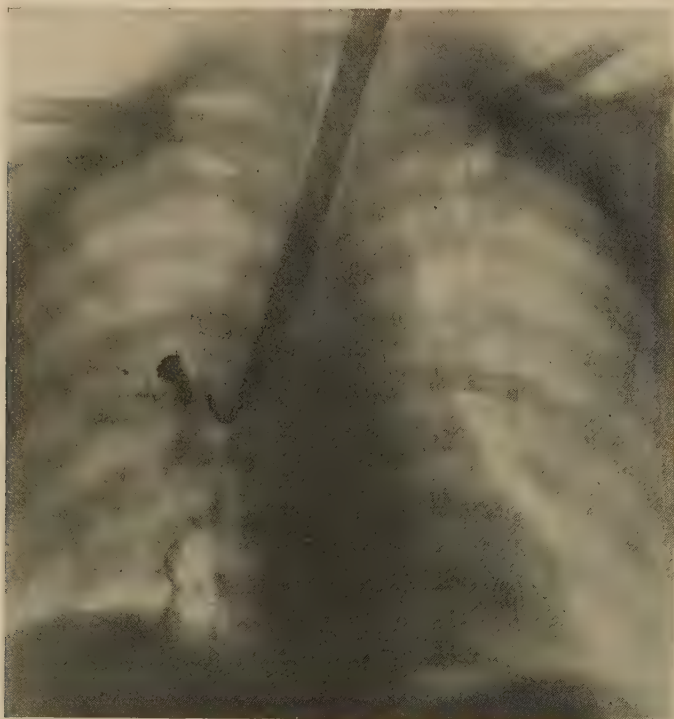


Fig. 654.—Case of abscess of the lung following pneumonia in the case of a man aged twenty-eight years. Definite localization of the process by physical signs could not be made. Under bronchoscopic guidance a Lynah tube was introduced into the right upper lobe bronchial orifice and by fluoroscopic direction iodized oil was slowly instilled until the cavity was filled to overflowing. From the stereoscopic pneumonograms made with the tube and bronchoscope *in situ* the abscess was localized in the lower portion of the upper lobe of the right lung. (Films by W. F. Manges.)

into the bronchus with a specially devised bronchoscopic atomizer. *Cocaine is never used in children* at any of the Chevalier Jackson Bronchoscopic Clinics.

**Indications for, and Advantages of, Bronchoscopic Pneumonography.**—For the bronchoscopic method to be advisable it requires, first of all, the services of a bronchoscopist who has acquired proper technical skill and has the assistance of a trained organization.

Although bismuth subcarbonate and iodized oil can be used interchangeably, each possesses individual advantages in certain conditions. Bismuth powder<sup>1</sup> is practically always expelled from the air passages within two or three days and is, therefore, to be preferred in foreign body studies. It is

especially valuable as an aid in the localization of bronchial foreign bodies "around the corner,"<sup>1</sup> to establish the relation between a peripherally located foreign body and the nearest accessible bronchus, to determine the relative position and size of the nearest bronchus in a case of penetrating



Fig. 655.—A pneumonogram, taken in the lateral plane, following the bronchoscopic instillation of iodized oil into the subdivisions of the right lower lobe bronchus, in the case of a man aged forty-three years. There was a history of lung abscess of two years' duration which had been treated surgically. In all, five surgical operations had been performed. There was marked improvement, however, cough and expectoration of pus persisted. Bronchoscopic investigation was recommended by the surgeon. From the pneumonograms Dr. W. F. Manges reported that there was present a definite area of bronchiectasis involving and limited to the upper posterior subdivision of the right lower lobe bronchus. (Collections of iodized oil indicated by arrows.)

foreign body<sup>7</sup> and to ascertain whether a suspected shadow is a foreign body in a bronchus or a calcareous deposit in the parenchymal tissue. It is useful to outline a stenosis<sup>4</sup> of the trachea or bronchus (Fig. 653), bronchiectasis of the larger bronchi, and to determine the extent of involvement

of a primary bronchial neoplasm.<sup>6</sup> It has the advantage in hemorrhagic cases of being a hemostatic.<sup>6</sup>

Iodized oil, 40 per cent., is preferable in all conditions that are associated with increased secretions, as in suppurative bronchitis, bronchiectasis, and pulmonary abscess (Fig. 654); also, if the pathological process is situated in the periphery of the lung.



Fig. 656.—Pneumonogram made in the case of a man aged fifty-three years, who was referred to the Chevalier Jackson Bronchoscopic Clinic by Dr. E. F. Butler for bronchoscopic aid in the localization of an abscess involving the lower lobe of the left lung. Under bronchoscopic guidance a curved Tucker tube was passed into the posterior subdivision of the left lower lobe (indicated by arrow). Following the aspiration of pus, iodized oil was instilled. With the bronchoscope and tube in position stereoroentgenograms were made and a lateral view was obtained. Dr. W. F. Manges was able to accurately localize the abscess in relation to the bony anatomical landmarks.

In addition to possessing the advantages gained by direct inspection of the interior of the tracheobronchial tree, bronchoscopic pneumonography permits of aspiration of secretions and removal of obstructions. A clear field can be obtained by cleaning out all channels, natural or pathological, a definite localization of the diseased area can be obtained, and the radio-opaque substance can be accurately placed even to carrying it beyond points of constriction.

Direct laryngoscopic pneumonography is feasible in that the material can be delivered into the tracheobronchial tree; however, it lacks the



Fig. 657.—Roentgenogram made in the case of a man aged twenty-eight years, who complained of cough, expectoration of pus, and hemoptysis. The physical signs and Roentgen-ray findings were inadequate to explain all the symptoms. The internist requested a bronchoscopic consultation. At bronchoscopy there was found a suppurative process limited to the right lower lobe. Iodized oil was instilled and pneumonograms made. These clearly indicated that the process was a well-localized bronchiectasis of marked degree. (Film by Dr. J. T. Farrell.)

advantages gained by accurate placing of the opaque substance, and a clear field is not obtainable.

LOUIS H. CLERF.

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